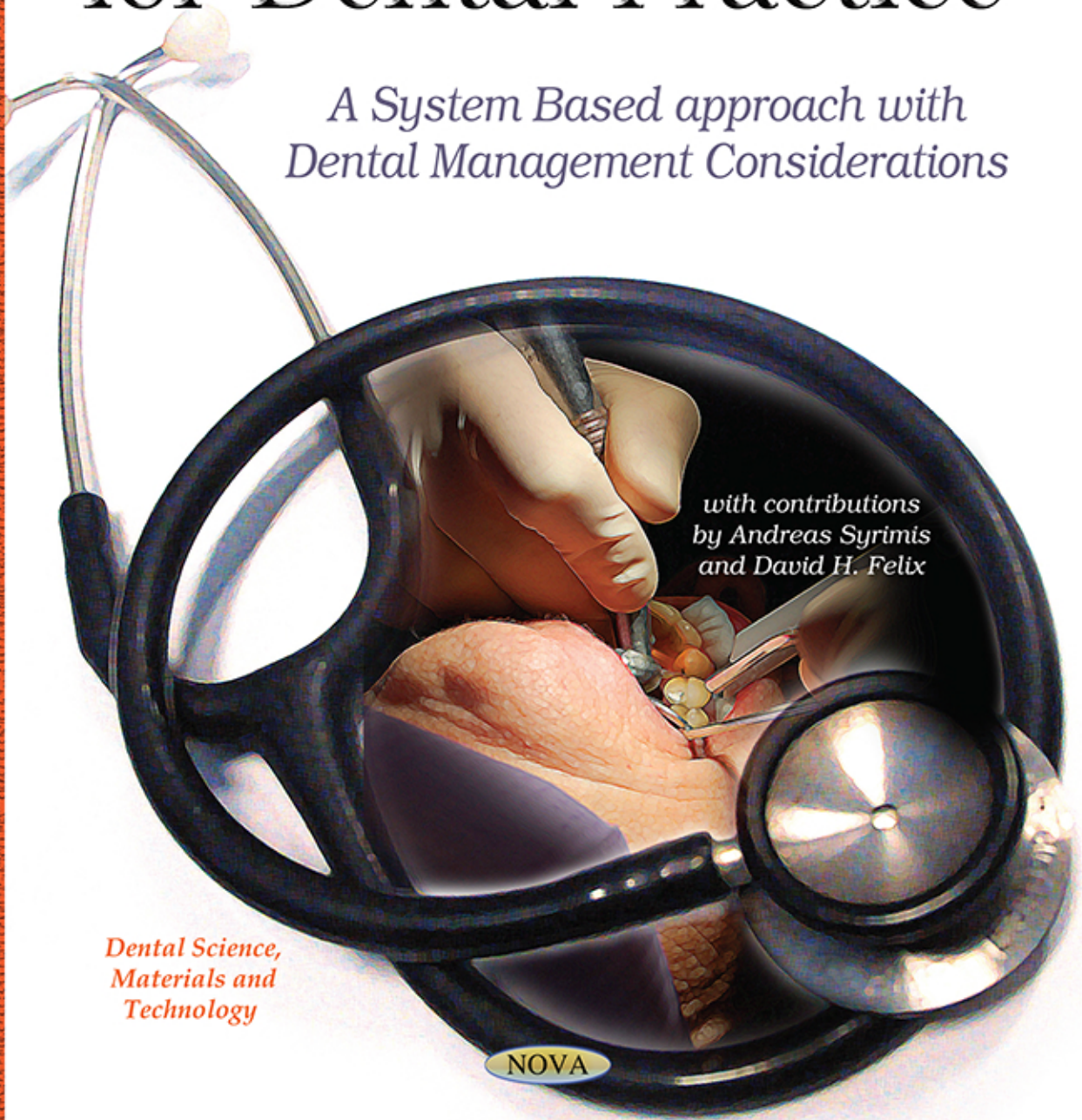
S. R. PRABHU

Lecture Notes on General Medicine for Dental Practice

A System Based approach with Dental Management Considerations



with contributions by Andreas Syrimis and David H. Felix

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LECTURE NOTES ON GENERAL MEDICINE FOR DENTAL PRACTICE

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S. R. PRABHU
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Preface

Although dental practitioners are not expected to treat dental patients for medical conditions, they need an understanding of common systemic diseases and their oral presentations. This knowledge enables them to assess risks prior to offering dental treatment, equips them to manage medical emergencies in dental practice, and to refer patients to general medical practitioners or specialists where appropriate. Comprehensive books on the subject are available in the marketplace, but they are too exhaustive for use in the day-to-day practice of dentistry.

The aim of '*Lecture Notes on General Medicine for Dental Practice'* therefore is to provide dental students and practitioners with a concise book that can be used as a quick reference resource in clinics or hospital wards, and for revision prior to examinations. In keeping with this aim, the book briefly discusses system-based diseases and disorders along with their oral manifestations and relevance to dental management.

Target readership of this book includes undergraduate and postgraduate dental students, practicing dentists, and those preparing for licensing, membership and fellowship, and board examinations. This book would also be useful to oral health therapists and dental hygienists.

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Chapter 1

History Taking and the Patient Interview

Andreas Syrimis

Abstract

This chapter deals with principles involved in taking a patient's history, and with interview techniques that are used in assessing a patient's overall health status. Starting with patient registration formalities, the chapter briefly deals with aspects of a patient's presenting complaints, medical history, dental history, drug history, family history, and social history. As a screening routine, systems inquiry relating to different systems of the body is also discussed. A brief review includes the cardiovascular, gastrointestinal, respiratory, urinary, reproductive, nervous, musculoskeletal, endocrinological and immunological systems. Inquiry also addresses the status of the skin, lymph nodes, blood and mental health. This chapter forms the basis on which a systematic search for the cause of a presenting complaint can be carried out.

Introduction

It is a well-established fact that in medicine, more than 80% of diagnoses are reached from well-conducted case histories. The remaining diagnoses are resolved from physical examination and diagnostic investigations. However, regardless of whether you are dealing with the patient's own story, your observations or laboratory procedures, in order to reach a diagnosis you will still need to process this information in a systematic way. This is called the formulation of differential diagnosis.

The patient registration form will provide you with the personal and administrative details of the patient, and these may also contain some clinically relevant information.

Principles of History Taking and Differential Diagnosis

Key prerequisites for successful case history taking are: good listening, interpersonal skills and analytical ability. Are you and your clinical setting ready to receive the patient? From the moment the patient enters your surgery you must eliminate any distractions, whether in your mind or in your environment. It is particularly important with a new patient to form a good rapport and this, to a large extent, is formulated during case history taking.

Key points/reminders:

- Is your consultation room ready to receive the patient?
- Is the patient registration form properly filled in?
- Have you introduced yourself and explained your role and that of any personal assistants?

What Is Differential Diagnosis?

This is a systematic process of generating hypotheses by evaluating all the data from a case history, physical examination and diagnostic tests in order to arrive at a point where you can identify the most probable diagnosis. "We use the specific findings from the history and examination of the patient to differentiate the relative probabilities of the potential conditions to create a short list of the most likely conditions, the differential diagnosis." [1]

Understanding and applying this process becomes crucial when you are faced with competing possibilities that share similar symptoms or when you are dealing with a patient suffering from multiple or co-existing conditions. For instance, is it an isolated disease, referred symptoms or a combination of both? In order to undertake an effective differential diagnosis you need to process reliable and relevant information from the case history, physical examination, and diagnostics such as imaging. If you do not follow this logical progression and you rush to take an x-ray of the patient's jaw bone, for example, you may arrive at an imprecise diagnosis, or even make an incorrect diagnosis. First, listen for the indicators, then observe, then examine closely, and then diagnose.

Medical case histories, especially at the first consultation, go far beyond the anatomical geography of the presenting complaint. If you have not seen a patient before, you need to gain an understanding of their general state of health for two important reasons: other pathologies may influence their dental presentation, and to identify 'red flags'. This need not be a long, drawn-out interrogation, but a competent and well directed series of questions that aim to reveal the their general state of health. To achieve this you can devise a screen with a series of questions using a structured form. Pause this procedure and focus on areas of relevance. This structured approach covers the following sections:

- Operations
- Accidents
- Hospitalisations
- Drug history
- Dental history

- Family history
- Personal and social history
- Systems inquiry

Depending on the structure of your registration form, if not already covered under the full case history, you should also include:

- Allergies and sensitivities (internal medications or topical).
- Medicines currently being taken.
- Chronic illness (e.g., diabetes, asthma, heart disease, blood disorders, infections).
- Might they be pregnant?

Patient Registration

The personal details recorded on the patient registration form may vary between clinics and countries, or be dictated by professional bodies. In most instances, you will be expected to record the following patient details:

- Date of the consultation
- Patient's name
- Contact details: address and telephone number(s)
- Date of birth
- Their General Practitioner's name and address
- Emergency contact
- Occupation
- Weight and height
- Marital status and any children
- Who referred them to you
- Confidentiality statement
- Signature

Some practitioners or institutions prefer to ask the patient to complete a check-list of common conditions, especially those which have a bearing on dental and oral interventions such as rheumatic fever.

Meeting the Patient

If you have not already done so, ensure that you introduce yourself – and any other assistants or colleagues present – and shake hands. This is the first physical contact and it helps to build trust and reassure the patient. Effective case history taking requires clinicians to exercise good interpersonal and analytical skills. This is the ability to *really* listen to what the patient is actually trying to tell you, what this might mean, what follow-up questions to ask, and to accurately record the patient's account. Ensure that the patient's sitting position and

yours are conducive to case history taking. This is best done with both of you sitting at eye level, either at an oblique angle or facing each other.

After the introduction and other formalities, begin the case history with an open invitation to talk. Give the patient a few moments to compose their thoughts and allow them to speak uninterrupted. This is particularly important at the start of the case history. Your tendency will be to interrupt and ask them clarifying questions or to elaborate further on areas you might consider vital in your evolving hypothesis.

Resist this temptation, as in many instances the patient may soon reveal such information. If necessary, ask the patient to wait while you record key facts and figures, and then allow them to continue. In many cases, after two or three minutes of uninterrupted talk, the patient will normally pause. At this stage, you may wish to reflect on what has been said and refine the line of inquiry by asking them to provide you with specific answers. Employ techniques such as:

- Facilitation, reflection, empathic responses and summarising.
- Open questions
- Use simple gestures such as nodding or a simple expression such as “Please tell me more about...”

The Presenting Complaint and Its History

After the invitation to talk freely, you should be in a position to identify the presenting complaint. There may be instances when the patient is seeking help for several seemingly related, or even unrelated, issues. In such instances, try to ascertain from an early stage if you are dealing with a single issue or multiple problems. Negotiate with the patient the priorities within the parameters of the day’s consultation. There may be a difference of opinion about the priorities as your medical knowledge enables you to see beyond the patient’s perception of their condition. For instance, is the patient eager to improve the aesthetics of their dentition, or is your priority to address their virulent gingivitis? Sometimes, such dilemmas may not become apparent until after the physical examination. If at this early stage of the case history there appear to be several independent complaints, in order to make productive progress, mutual agreement should be reached on which area to focus, and investigate this fully. This is the investigation of the presenting complaint and it is where you will focus your line of inquiry; use a series of questions intended to provide you with key information for your evolving hypothesis and subsequent differential diagnosis. Remember these prompts using the acronym **SOCRATES:**

- **S**ite
- **O**nset
- **C**haracter (of pain or symptoms)
- **R**adiations (of pain)
- **A**ssociated (manifestations or symptoms)
- **T**iming
- **E**xacerbating and Relieving factors
- **S**everity

The Presenting complaint section, in most instances, is the area where you would focus most of your time resources, to ensure that you fully extrapolate the nature of the anatomical or physiological disturbance.

Site ➔ Please point to the area of pain (or symptoms). Is it always there?

Onset ➔ How did it start? Was it acute with a short duration, or insidiously over a long period? When did you first experience this?

Character (of pain or symptoms) ➔ Can you describe the sensation? Is it always like this?

Radiations ➔ Does the pain spread to any other locations? Does is start from another location? What does this other pain feel like?

Associated (manifestations or symptoms) ➔ What else is happening to you at the same time or just before or after? E.g., Locking of the jaw, headache, neck pain, sinusitis, stress?

Timing ➔ How long do the symptoms last? When do you get them? Is there a pattern?

Exacerbating and relieving factors ➔ What brings them on? What makes them worse? What makes them go away? What makes them better? For example, heat, cold, pressure or all of them? Anti-inflammatories or opiates?

Severity ➔ How bad is this for you? If pain is not the issue, then clarify. Is this getting better or worse?

General Medical History

- History of present illnesses
- Operations
- Accidents and injuries
- Hospitalisations

This and subsequent sections of the medical inquiry will vary in depth and detail depending mostly on two elements: the nature of the presenting complaint, and the age and state of the patient's health.

Let us illustrate this with some examples: a) a healthy 8 year old with a shaky milk tooth that needs extraction, or b) an adult needing to have his wisdom tooth extracted and is also suffering from diabetes, heart disease, asthma and other ailments, and is taking a cocktail of medications. In any case, if you have not seen that patient before, whatever their presenting complaint and state of health, you must ensure you gain a sufficiently inclusive picture of their health. The order of these sections might vary but this should not influence the outcome. Make the necessary allowances for the extreme of ages with a paediatric and geriatric medical history.

- **History of Present Illness (HPI)**

The chronological narrative is the patient's account of any conditions they may suffer from at present, and whether they are under a formal treatment regime or are self-managed. Some patients may also consider this as an opportunity to offload all their ailments on to you, or to turn the consultation into a form of 'therapy'. Try to manage such instances by guiding

the patient back to pertinent details for this type of consultation. Record all key and relevant facts. Superfluous information should be avoided.

Clarify any unclear statements the patient makes, but refrain from using your own terminology and interpretations at this stage. It is important when talking to your patient that you convert any medical or pharmacological terms into common, understandable expressions. If appropriate, link any illness revealed with more specific details in the subsequent systems review section. Even indirectly related conditions may be of relevance, for example, knowing that a patient is suffering from rheumatoid arthritis will influence the care you need to take when positioning their neck.

- What conditions are they suffering from at present?
- How are these currently treated or managed?
- Which conditions are currently in remission or relapse?

Ensure That You Ask About

- Rheumatic fever and infectious endocarditis.
- Diabetes.
- Infections: HIV, tuberculosis, hepatitis, syphilis, herpes, influenza, mumps.
- Recent travel to the tropics or an endemic area.
- Epilepsy
- Operations

Briefly inquire about any major operation the patient may have had, and the reasoning behind them, any complications arising, and the outcomes. Recording minor operations such as the removal of a bunion on their big toe may not seem to be of significance to the dental practitioner, but even this instance, they may identify a possible complication with a diabetic patient.

- **Accidents and injuries**

Inquire if the patient has suffered any *major* accidents or injuries, such as road traffic accidents (RTAs), work injuries, sports injuries or falls. Judge the importance of these within the context of the consultation and expand on them further if necessary. For example, assess the significance of a head injury in a road traffic accident, an accidental stabbing with a syringe by a health worker, or TMJ injuries in an ex-boxer.

- **Hospitalisations**

The significance in this question lies not in whether they have ever been admitted to hospital, or for how long they stayed, but in revealing information that the patient may not have volunteered or considered significant. Consider here the implications of a patient who has had TB or rheumatic fever.

- **Dental history**

Find out how well the patient cares for their teeth, any procedures they have had, which practitioner the patient normally sees, how frequently they have check-ups, and how long ago was their last one. Depending on the presentation, you may need to gather information on past and present oral care, any trauma to the oro-facial structures, any reactions to dental materials, and any local anaesthetics or medicines used.

Drug History

The significance of inquiring about and recording the patient's drug history cannot be underestimated. As we get older, we often find ourselves on an ever increasing cocktail of drugs, some of which are prescribed, while others are purchased 'over the counter' (OTC). Some detailed patient forms may have a list of medications which need to be recorded due to their influence on your procedures, and alerting you to possible interactions and side-effects. These are some of the medicines about which you need to inquire:

- Anticoagulants: warfarin, heparin, aspirin
- Anticonvulsants
- Corticosteroids
- Immunosuppressants
- Heart medications: anti-arrhythmics, digoxin

Has the patient had any adverse drug reactions or allergies, such as an allergy to penicillin or non-steroidal anti-inflammatory drugs (NSAIDs)?

Does the patient suffer from pathology for which certain drugs may be contraindicated? Consider the following conditions where several drugs may be contraindicated:

- Liver disease
- Renal failure
- Gastrointestinal erosions
- Serious pathologies affecting the heart, lungs or brain
- Pregnancy or breast-feeding

Ensure that you inquire about OTC medications, their strengths and frequency. Since an increasing number of patients are now seeking alternative or complementary treatments, also inquire about any herbs or supplements they might be taking. One example is potentiated or standardised extracts of Ginkgo biloba, which exhibit anti-coagulant effects, and anther is St John's Wort, which interferes with liver enzymes. Some may also interact with a patient's prescribed medications.

Family History

One way of approaching this topic is to inquire about the state of health (or cause of death) of the patient's parents and siblings. Alternatively, ask them if they know of any conditions that run in their family such as:

- Diabetes
- Heart disease
- Cancer (esp. Malignant melanoma, breast cancer)
- Blood disorders
- Seizures

Personal and Social History

In many instances, your patient's personal habits may have a significant bearing on their dental health. Consider the local and systemic impact of smoking and alcohol consumption on their dental health. Smoking and drinking (especially spirits) are associated with oral, oesophageal and stomach cancers. Inquire about:

- Smoking habit
- Alcohol consumption
- Recreational drugs, including the chewing of tobacco, betel nuts and others
- Diet
- Occupation and possible environmental hazards
- Sleep
- Exercise and recreational activities

Systems Enquiry

- **Rationale for systems enquiry**

This section is intended to ensure that all major systems of the body are considered as part of your **screening routine**. Earlier, when you asked the patient about their general state of health, it is possible that important facts may have been missed. Some clinicians cover this aspect of medical history by basing their questions on the physiology of the systems and their organs. Others prefer to take a more topographical or regional approach. For instance, you could ask questions by referring to the cardiovascular system, or to symptoms emanating or stemming from within the thorax; the choice is yours provided you adopt a logical and systematic approach.

Record all positive answers, and any abnormal findings or facts. Some clinicians prefer to work from a check list to ensure all important aspects are covered, especially red flags- to avoid making this section seem like an interrogation, make it as brief and succinct as possible.

Systems approach	**Equivalent topographic approach**
Cardiovascular system	The thorax
Respiratory system	The thorax
Systems approach	**Equivalent topographic approach**
Gastrointestinal system	The abdomen
Genitourinary system	The pelvic region
Nervous system	The head, special senses and spinal cord
Reproductive system	The pelvic region
Musculoskeletal system	The limbs and joints
Integumentary system	The skin
Endocrine system	The various glands

The extent and depth of this stage in the case history is often dictated by the type of patient and their presenting problem: are we dealing with a healthy 16-year-old or a 75-year-old with multiple chronic pathologies affecting several systems? Although we take an all-inclusive approach to systems review in the training of clinicians, in the real clinical environment you will need to exercise your judgement as to what is relevant. It is good practice to begin with some non-system specific questions such as:

- How is your appetite?
- Are you gaining or losing weight?
- Any night sweats or night pain?

Cardiovascular System Review

Inquire about the functioning of the heart, arteries, and veins. Some of the following prompts are described with medical terminology. Clarify broad or loosely used terms such as ‘palpitations’: does it mean irregular, fast, intense, or just awareness of, the heart beat?

- Blood pressure?
- Chest pains?
- Cough, frothy sputum?
- Palpitations, irregular or racing heart rhythm?
- Dyspnoea, especially on exertion?
- Orthopnoea?
- Calf claudication with walking?
- Cyanosis or pallor?
- Abdominal swelling (ascites)?
- Ankle oedema?
- History of rheumatic fever, infectious endocarditis, MI?
- Medications: digoxin, warfarin, quinidine, nitroglycerine?

Gastrointestinal System Review

This section covers structures from the mouth to the anus. In addition to the digestive tube, also consider associated organs and the various glands, the liver, gall bladder and pancreas.

- Abdominal pain, indigestion, colic?
- Mouth ulcers, sore tongue?
- Reflux?
- Difficulty swallowing (dysphagia)?
- Bloating?
- Vomiting blood (haematemesis)?
- Changes in bowel habit, retention, incontinence?
- Anal blood; fresh or black stools (melaena), pale stools (steatorrhoea)?
- Urinary problems, retention, incontinence, dark urine?
- History of hepatitis, jaundice, ulcers?
- Medications: antacids, laxatives, corticosteroids?

Respiratory System Review

Consider the whole of the respiratory tree, beginning from the nose and mouth and finishing at the alveoli. Inquire about pathologies that might affect the nasal passages, paranasal sinuses, trachea, bronchi, lung tissues, the pleural membranes and respiratory mechanism.

- Shortness of breath?
- Chest pain?
- Cough?
- Character of sputum, blood (haemoptysis)?
- Wheezing?
- Problems with upper respiratory tract, and air sinuses?
- Chest or spinal deformities?
- History of asthma, pneumonia, COPD, TB?
- Ever had a chest x-ray?
- Medications: bronchodilators, corticosteroids, antibiotics, antihistamines?

Urinary System Review

Although the urinary and reproductive systems share some common anatomy in the more distal parts, it is simpler to consider them separately. Inquire about symptoms which may stem from the kidneys, the ureters, the bladder, the urethra and associated sphincters.

- Pain or burning with urination (dysuria)?
- Discharge, pus, mucus, blood?
- Frequency, retention, incontinence?
- Excessive night-time urination (nocturia)?
- Changes in urine colour or smell?
- Genital lesions, rashes or lumps?
- History of high blood pressure, kidney problems, stones, infections? Medications: antibiotics, alpha blockers?

Reproductive System Review

Avoid repeating questions that have already been covered under the urinary system. Dividing this section into male and female reproductive structures helps to focus your line of inquiry. For female patients, include the ovaries, uterine tubes, uterus, vagina, external genitalia and associated glands. For male patients, include the prostate, testes, penis, associated glands and tubes.

- Pregnancies, children, abortions and miscarriages?
- Menstrual problems?
- Currently pregnant?
- Breastfeeding?
- Contraceptive method?
- Breast examinations, lumps, discharge?
- Menopausal, on HRT, post-menopausal bleeds?
- History of STDs, infections and malignancies?
- Testicular lumps?
- Problems with libido, erection?
- Medications: HRT, contraceptive

Nervous system

There are various ways of organising the inquiry on the broad and complex nervous system. You may need to consider structures of the central and peripheral nervous system. For the central nervous system, include the cerebrum, cerebellum and brain stem, the cranial nerves and their pathways, their effector structures (e.g., organs of special senses), ganglia, and the spinal cord. For the peripheral nervous system, consider the efferent and afferent nerve roots, ganglia, peripheral nerves and effector structures. Remember to include structures associated with the sympathetic and parasympathetic autonomic systems.

- Headaches and migraines
- Facial pains, jaw problems, neuralgias?
- Problems with vision -, blurred or double (diplopia)?
- Problems with hearing, tinnitus?
- Dizziness, fainting, fits, blackouts

- Problems with gait (ataxia), dizziness and vomiting?
- Loss of smell (anosmia), rhinitis?
- Difficulty swallowing (dysphagia)?
- Changes in voice or word articulation (dysarthria)?
- Changes in voice (dysphonia)
- Any areas of altered sensation (paraesthesiae), or numbness
- Muscle spasms, weakness, wasting or twitches?
- Problems with memory, sleep or stress?
- Head injuries?
- History of high blood pressure, cholesterol or strokes?
- Medications: analgesics, opiates, anti-inflammatories, antiepileptic, sedatives?

Musculoskeletal System Review

In this section, inquire about pathologies or symptoms that might affect connective tissues and locomotor structures, including the joints, bones, muscles, ligaments, tendons and bursae.

- Joint pain or swelling?
- Morning stiffness?
- Muscle weakness, wasting, atrophy?
- Cramps when walking (vascular claudication)?
- Joint pains with recent skin rashes, mouth ulcers, urogenital or eye symptoms?
- History of gout or inflammatory connective tissue disease, e.g., rheumatoid arthritis?
- Medications: NSAIDs, steroids, immunosuppressants.

Endocrine and Immune Systems Review

Like the nervous system, this area covers a broad and diverse domain. Consider here the endocrine and exocrine glands, common pathologies and their symptoms. Include the pituitary, thyroid, parathyroid, thymus, pancreas, adrenals, ovaries and testes.

- Thirst with frequent urination (polydipsia, polyuria)?
- Changes in sensation in fingers and toes?
- Throat discomfort or neck swelling?
- Shaking of the hands (tremor)?
- Weight or appetite changes?
- Restlessness and intolerance to heat?
- Palpitations
- Lethargy and sensitivity to cold?
- Changes in skin (purple striae, pigmentation, spots), hair, voice?
- Headaches and visual problems?
- Changes in shape or size of feet and hands?
- Changes in libido or sexual characteristics?

- Menstrual irregularities?
- Frequent infections or slow resolution?
- Medications: thyroxin, insulin, steroids, immunosupressants, antibiotics
- Miscellanea (skin, lymph nodes, blood, psychiatric)

The previous inquiries addressed the major body systems. Some areas of our anatomy and physiology do not fit neatly into these somewhat artificial system boundaries. Consider pathologies and symptoms relating to the skin and epithelial tissues, palpable lymph nodes and the structures they drain, and hereditary and acquired blood disorders. If appropriate, also inquire about psychoemotional disorders.

- New skin moles, or changes to old moles?
- Persistent skin sores?
- Skin itchiness and exudates?
- Changes to skin colour, hair and nails?
- Skin lumps or frequent infections?
- History of use of corticosteroids?
- Enlarged or tender lymph nodes (under mandible, neck, near clavicles, axillae, groin)?
- Bleeding or bruising predisposition (including the gums)?
- Familial blood disorders (haemophilia, thalassemia)?
- History of anaemia, blood transfusions?
- Predisposition to blood clots?
- Emotional lability (rapid, often exaggerated, changes in mood)?
- Sleep problems?
- History of depression, panic attacks, hallucinations?
- Use of psychotropic drugs, sedatives, recreational drugs?

Chapter 2

General Physical Examination

Andreas Syrimis

Abstract

Having completed the history taking exercise, a general physical examination is the next important step in the diagnostic process. Starting with principles of physical examination and preparation of the clinical setting, this chapter briefly discusses the steps involved in measurement of the vital signs which include the pulse, respiratory rate, blood pressure and body temperature. In addition, visual inspection of nail, hands, face, eyes, mouth, neck, thorax, abdomen and lower limbs is also briefly discussed in this chapter.

Introduction

- **Principles of physical examination**

During your encounter with the patient and taking their case history, in your mind you will be processing the information gathered. By doing so, you will be re-considering, changing or discarding your hypotheses of the patient's condition; however, proper differential diagnosis must be based on objective information. During the case history discussion, the patient discloses a description of their symptoms and details of health-related events. Although this account may be valid, it is considered as subjective information embedded within the patient's feelings and perceptions, and may be affected by their ability to describe them. Essentially this is an account of their symptoms. Your evolving differential diagnosis should, in most cases, be validated by seeking out corresponding *signs*. Clinical signs are findings revealed by the clinician during the process of physical examination. Signs can be gathered by simple techniques such as observation, or by more specialised techniques like auscultation, palpation and percussion. Information gathered from clinical signs is considered as *objective* and more reliable in nature. For example, a patient may complain of feeling abdominally bloated, or from a tooth sensitive to bite or pressure. Subsequent

observation and palpation of the abdomen may reveal overt bloating, and similarly, putting pressure on, or tapping, a suspect tooth may not reveal any sensitivity. Although we stated earlier that most diagnoses are reached from case history alone, for safety reasons, it is important that you look for or elicit signs to help confirm the patient's account.

Clinical examination of the body systems should be carried out in a logical, progressive manner. The accepted protocol is that you start with simple techniques such as observation and then progress to more specialised procedures.

Listen to their complaint ➔ look ➔ feel ➔ move ➔ investigate

Before any physical examination, you must explain to your patient what you intend to do and, if appropriate, tell them why. Certain procedures may require a verbal or written consent whilst others may require the presence of a chaperone. Minors and those with mental disabilities will require a legal guardian to be present, but as these regulations can vary you must ensure you comply with your professional body's guidelines.

Although not frequently done, if you need to examine areas other than those directly associated with dentistry, you may need to ask your patient to remove some of their clothing. Explain to them what you need to do and why. Provide a suitable place for them to undress, such as a screen. It is also important that the examination environment is appropriately heated, especially if the patient is elderly or infirm.

Preparation of the Clinical Setting

Before receiving a patient in the consultation room or the surgery, ensure that all preparatory activities have been carried out and all evidence of the previous clinical encounter are removed. Remove all used or disposable items.

Clean or sterilise all previously used or contaminated areas and surfaces. Don't forget the light handles, chairs with arm rests, door handles and computer mouse.

Ensure that all examination equipment is prepared and functional. For most systems examination procedures – excluding dental-specific instruments – you might need:

- Stethoscope (for heart, lungs, abdomen, vessels)
- Sphygmomanometer (blood pressure measurement)
- Ophthalmoscope and auroscope (eyes, nose, ears)
- Tongue depressors (inspecting the oropharynx)
- Patella hammer (reflex taking)
- Snellen chart (visual acuity)
- Thermometer
- Peak flow meter (lung capacity, e.g., asthmatics, COPD)
- Sterile pins/neuropins and cotton wool (sensation testing)
- Tuning fork (256Hz) (testing vibration, e.g., peripheral neuropathies)
- Tape measure
- Weighing scales

Vital Signs

In a comprehensive examination of a patient, the recording of vital signs should precede all other specific or regional examinations. This is particularly important if the patient has co-existing morbidities, may not be feeling constitutionally well, or looks ill at the time of attending a dental surgery. Examples include a patient with signs of pallor, cyanosis, jaundice or evidence of febrile illness.

The measurement of vital signs involves the recording of general and important physiological indicators of the function of vital systems of the body.

Essential:

- Pulse rate and rhythm
- Respiratory rate
- Blood pressure
- Temperature measurement

Additional procedures:

- Seek evidence of pain
- Oximetry
- State of pupils
- General observation of the patient.

Pulse Rate and Rhythm

The pulse is normally taken at the radial artery just above the thumb. Other possible sites are the brachial artery in the anterior and medial aspect of the elbow, the carotids on the side of the thyroid cartilage, or the temporal, just anterior to the tragus, of the ear. Larger arteries are preferred when evaluating the character of the pulse. Measure the beats for 30 seconds and then multiply by two.

Pulse rates:

- Infants: 80 - 140 bpm
- Children: 70 - 110 bpm
- Adults: 60 - 100 bpm

Respiratory rate:

The patient must not be aware that you are evaluating their breathing rate as this will influence the measurement. There are various inconspicuous techniques:

- Listen for the sounds in loud breathers
- Pretend you are taking the pulse whilst observing the lower parts of their thorax or abdomen.
- Pretend you are taking the pulse whilst resting the hand over the diaphragm.

Blood pressure:

Blood pressure may be taken using:

- Aneroid sphygmomanometer
- Electronic sphygmomanometer
- Mercury sphygmomanometer (may not be permitted in certain environments or need to be fixed onto a wall)

If using a manual type, you will need to be trained to auscultate the brachial artery during the inflation and deflation phases of the procedure whilst listening for the systolic and diastolic sounds.

A professional, reliable electronic device will provide you with accurate measurements of blood pressure and pulse rates simultaneously. Please observe the following important points when taking a patient's blood pressure reading:

- The arm should be positioned at the same level as the patient's heart
- The arm should be passive and relaxed
- The blood pressure may be influenced by:
    - Stress and possible '*white coat effect'*
    - Medications
    - Stimulants such as coffee
    - Recent exercise/exertion

Normal ranges of blood pressure

	Systolic	**Diastolic**
Infants	75-100 mmHg	50-70 mmHg
Children	80-110 mmHg	50-80 mmHg
Adults	90-140 mmHg	60-90 mmHg

Temperature:

Temperature can be taken using various devices. In the mouth, it can be taken sublingually using a mercury or coloured alcohol-based device, or with an electronic type. In the ear, the electronic type has become more popular with the use of quick disposable sleeves. The normal body temperature is 37 °C or 98.6 °F.

Blood oximetry:

The saturation of oxygen in arterial blood can easily be measured using a digital oximeter. The device measures the percentage of saturation of arterial oxyhaemoglobin.

Normal range of saturation should be between 95-99%. If below 94% (or 88-94%), the patient is in a state of hypoxia.

General Inspection of the Patient

After recording the vital signs, perform a general examination of the patient. For this procedure, the patient must be sufficiently undressed in order to be able to inspect areas such as the thorax, abdomen and limbs.

Some clinicians start with the hands whilst others with the face. The advantage in starting with the hands is that it is considered the least intrusive contact and may help to put the patient at ease.

Nail and Hand Features

Nails:

- Clubbing (also known as drumstick fingers and watch-glass nails): chronic and serious diseases of lung, heart, bowel, cancer.
- Leukonychia (white discolouration on the nails): hypoalbuminaemia (liver, kidney, bowel).
- Koilonychia (spoon shaped nails): iron deficiency anaemia.
- Splinter haemorrhages (tiny blood clots that tend to run vertically under the nails): infectious endocarditis, vasculitis.
- Pitting and onycholysis (detachment of the nail from the nail bed): psoriasis.
- Beau's lines (deep grooved lines that run from the side of the finger nail):
- Various protracted systemic illnesses, fever, chemotherapy.

Then check the hands for:

- Palmar erythema: red warm palms (liver palms). Causes: liver diseases, oestrogens, pregnancy.
- Heberden's nodes: bony deformities in distal interphalangeal joints. Causes: osteoarthritis.
- Symmetrical soft swelling in proximal interphalangeal joints and deformity of the wrists. Causes: rheumatoid arthritis.

Examination of the Face, Eyes and Mouth

Look out for evidence of endocrine pathologies.

- Staring protruding eyes, restlessness ➔ hyperthyroidism.
- Cold, pale, puffy looking skin, loss of eyebrows ➔ hypothyroidism.
- 'Moon face', thinning of skin, acne ➔ Cushing's disease (excess corticosteroids)

- Increased pigmentation, especially buccal membranes, creases and old scars ➔ Addison's disease (adrenal insufficiency).
- Gaps between teeth, broad mandible, large tongue, tunnel vision ➔ Acromegaly (pituitary adenoma, excess growth hormone).
- Yellow sclera ➔ jaundice (liver disease).
- Pale conjunctivae ➔ anaemia.
- Corneal arcus (a greyish-white ring occurring in the periphery of the cornea) or xanthelasmas (soft, yellow-orange plaques on the eyelids) ➔ hypercholesterolemia.
- Blue conjunctivae and mucus membranes ➔ central cyanosis.
- Lack of saliva, creases on tongue, slow skin recoil when pinched ➔ dehydration, diabetes, Sjögren's syndrome.
- Angular stomatitis, cheilitis ➔ badly-fitting dentures, oral candidosis.
- Red 'beefy' tongue, soreness ➔ Deficiencies of iron, B_{12}, Niacin, folic acid.
- White coating on tongue ➔ enlarged filliform papillae, candida.
- White patches or plaques in buccal mucous membranes ➔ leukoplakia, lichen planus, hyperkeratosis.
- Ulcers in oral mucous membranes ➔ recurrent aphthous ulcers, Crohn's disease.
- Gingival disease associated with infections i.e., viruses, fungus, bacteria and hyperplastic gingiva due to medications (calcium channel blockers) and leukaemia.

Inspection of the Neck

Observe the neck from the front and in profile. Look out for:

- Neck swelling. Localised: lymph nodes, cysts, tumours. Generalised/diffuse: goitre, tonsils.
- The cervical spine: shape, deformities, restrictions.
- Muscle bulk, distribution and symmetry.
- Skin lesions: pigmentation, erythematous, suppuration, excoriation
- Scars: past trauma or surgery.

Inspection of the Thorax

The thorax is best observed sitting or standing so that anterior, lateral and posterior aspects can be examined. Look out for:

- Deformities of the thorax: barrel chest, pigeon chest, funnel chest
- Deformities of the thoracic spine: kyphosis, scoliosis, evidence of ankylosing spondylitis
- Scars: past trauma or surgery.
- The breathing pattern.

Inspection of the Abdomen

Observation of the abdomen should be performed with the patient lying supine on an examination couch. Look out for:

Overall Shape of the Abdomen and Pelvic Region

- Abdominal swelling, and whether uniform or localised.
- Skin lesions, scars and discolourations.
- Prominent abdominal veins.
- Evidence of hernia.
- Abnormal pulsations.

Inspection of Upper and Lower Limbs

Observe the arms and legs paying particular attention at the distal areas. Look out for:

- The colour and perfusion.
- Their shape and proportion in relation to the torso.
- The muscle bulk and symmetry.
- The distribution of the hair and condition of the nails.
- Evidence of skin infections, lesions, and swelling such as oedema.
- If indicated, also palpate the peripheral pulses.

The above procedure should take the form of a quick scan of the entire body in a systemic way looking out for any overt signs of pathology. If you observe any suspect signs, examine that area more thoroughly. At this juncture of the general examination procedure, it may appear to overlap with the system-specific examination (Chapter 3).

Naturally, depending on the clinical scenario, only consider a comprehensive examination of one of the body's systems if you suspect a pathology affecting that system or its organs. Your decision to carry out a system-specific examination should be based on information provided during case history taking, your observations, or from signs observed during the general scan of the body which usually follows the vital signs stage.

Chapter 3

System-Specific Examination

Andreas Syrimis

Abstract

History taking and physical examination form the basis for the third important step in the patient assessment protocol. This involves system-specific examination. When patients complain of symptoms that may relate to one or more systems of the body, clinicians should be able to demonstrate the ability to examine the systems. This will enable them to determine the system-based origin of the symptom. This chapter briefly describes the steps involved in system-based physical examination. Steps listed in the boxes include the anatomical regions and systems/structures of the body, focused case history questions and the systems examination procedure. Examination of the major systems of interest to dental practitioners is included in this chapter.

Introduction

The need to carry out a more detailed system-oriented examination may arise as a result of information revealed during case history taking, or clinical signs discovered during the general examination procedure. For example, you might wish to perform a cardiovascular examination in a patient who presents with blue lips suggestive of central cyanosis. Similarly, you might perform a respiratory examination if there is dyspnoea, wheezing or cough.

In general practice, a patient may present with a suspect pathology relating to a region such as the thorax, volunteer symptoms implicating an organ such as the heart, or affecting a whole body system such as the cardiovascular system. In such instances, the physician must focus on this region and investigate it further with specific questions relating to the function and structure of the suspect area. These focused and well-directed case history questions must precede any subsequent system-specific examination. The following regional, system-specific examinations provide a brief summary of: (A) Anatomic Structures (B) Focused Case History Questions and (C) Systems Examination Procedure.

The gastrointestinal system

Structures associated with the gastrointestinal system
Mouth
Oesophagus
Stomach
Duodenum
Liver and Gall bladder
Pancreas
Small intestine
Large intestine

Case history questions
Changes in appetite
Nausea
Vomiting
Vomiting blood (haematemesis)
Reflux
Weight changes
Dysphagia (swallowing difficulties)
Mouth ulcers, sore tongue
Abdominal pain
Changes in bowel habit
Bloating
Blood in stools (melena), mucus
Steatorrhoea (oily, poorly formed stools)
Jaundice
Incontinence (faecal, urinary)
Food intolerances
History of acute abdomen
Operations
Medications

Clinical examination
Inspect the hands: colour, temperature, clubbing, leukonychia, liver palms, Dupuytren's contracture, flapping tremor
Inspect the face & eyes: endocrine facies, anaemia, jaundice, xanthelasmas
Mouth and mucus membranes: cyanosis, anaemia, tongue coating, ulcers, oral health, oral smell
Inspect the abdominal area : general skin features, scars, spider naevi, caput medusae, pink striae, surface vessels **Uniform swelling:** (the "five F's") flatus, faeces, fat, foetus, fluid **Non-symmetrical visible swellings**: masses, fibroids, cysts
Auscultation: bowel sounds (borborygmi),: presence, excessive, or absent The aorta or renal arteries for bruit
Percussion: changes in sound, masses, organ delineation (liver-spleen-bladder), kidney punch Fluid wave and shifting dullness for ascites
Palpation: general / superficial: feel for tenderness General deeper palpation (bimanual): masses, tissue quality Rebound tenderness or using cough technique Specific organ palpation: liver, gall bladder (Murphy's sign), kidney balloting, spleen (Traub's space), aorta, appendix (McBurney's point) Lymphatic palpation: especially inguinal and left supraclavicular (Virchow's node)
Other procedures: examination of urine: colour, blood, protein, glucose, bilirubin, urobilinogen (use of urinalysis dipsticks)

The cardiovascular system

Structures associated with the cardiovascularSystem
Heart
Arteries
Veins
Lymphatic vessels
(Lungs)

Case history questions
Chest pain
Dyspnoea
Dizziness
Cyanosis or pallor
Ankle swelling / oedema
Orthopnoea, paroxysmal nocturnal dyspnoea
Fatigue
Nocturia
Varicose veins
Fits, faints, blackouts
Hypertension
High cholesterol
Diabetes
Rheumatic fever
Claudication, cramps
Palpitations
Family history of heart disease
Operations
Medications

Clinical examination
Measurement of blood pressure and pulse rate
General observation: pale, flushed, cyanosed, deformities of the thorax, scars
Inspect the hands and nails: clubbing, cyanosis, splinter, haemorrhages, Osler's nodes, arterial perfusion, Raynaud' s phenomena
Inspect the eyes: anaemia, central cyanosis, corneal arcus, xanthelasmas
Inspect the mouth: anaemia, cyanosis, oral health, ulcers
Inspect the neck for elevated jugular venous pressure
Inspect the thorax and abdomen: chest deformities, parasternal heaves, ascites, spider naevi
Inspect the lower limbs: colour, perfusion, oedema
Palpation and comparison of distal pulses: temporal, dorsalis pedis, posterior tibial
Palpation of the precordium and apex beat
Auscultation: physiological 1st and 2nd heart sounds and pathological 3rd and 4th sounds, murmurs, added sounds

The respiratory system

Structures associated with the respiratory System
Nose and paranasal air sinuses
Nasopharynx
Larynx
Trachea, bronchi, bronchioles
Alveoli
Pleurae
Diaphragm
Ribs

Case history questions
Cough (with/without sputum)
Shortness of breath
Asthma, wheezing
Recurring chest infections, bronchitis
Chest pain
Coughing up blood (haemoptysis)
Smoker
Recurring fevers
Exposure to environmental hazards
Previous investigations, X-rays
History of TB, pneumonia, COPD
Medications

Clinical examination
Observe the face: general appearance, pale, flushed, cyanosed, clubbing, 'blue bloater, pink puffer', colour of lips and conjunctivae
Observe the thorax: kyphosis, scoliosis, barrel, funnel chest, pigeon chest
Observe the breathing movements: rate/min, depth, chest expansion, rhythm, dyspnoea
Observe the hands: finger clubbing, cyanosis, temperature, tremor, wasting of intrinsic muscles
Palpation of the trachea, central or deviating
Chest expansion
Tactile vocal fremitus
Percussion: over all lobes, anterior, lateral and posterior. Resonance, dullness, tympani
Auscultation: over all lobes, anterior, lateral and posterior. Crackles, wheezes, pleural rubs
Other procedures: peak flow measurements, spirometry

The nervous system

Structures associated with the nervous system
Brain (cerebrum, cerebellum, brainstem)
Spinal cord
Peripheral nerves
Autonomic nervous system
Special senses

Case history questions
Changes in sensation, numbness
Muscle weakness, fasciculations, tremors
Pain
Visual disturbances
Hearing problems, tinnitus
Changes affecting smell
Changes affecting taste
Difficulty swallowing (dysphagia)
Changes in voice (dysphonia)
Difficulty articulating words (dysarthria)
Balance problems, ataxia, unco-ordination
Fainting, loss of consciousness
Headaches
Injuries to head, neck, spinal column
Affective disorders, depression, mania, panic attacks
Medications

Clinical examination
Evaluate mental status, alertness, memory
Observe posture, gait, balance
Palpate major muscles: spasticity, flaccidity
Sensory examinations: Pain: pin prick Light touch: Cotton wool Vibration: 129 Hz tuning fork Temperature: hot, cold discrimination Other modalities: Joint position sense, graphaesthesia, stereognosis
Tendon reflexes: biceps, triceps, brachioradialis, knee, ankle Other reflexes: plantar, cremasteric, abdominal, jaw jerk
Muscle power Test for clonus
Cranial nerves:
I. Olfactory: smell
II. Optic: vision
III. Occulomotor: eye movement, pupil constriction, eyelid
IV. Trochlear: eye movement
V. Trigeminal: sensation to face, muscles of mastication, teeth, corneal reflex, jaw reflex
VI. Abducent: eye movement
VII. Facial: muscles of facial expression, taste, saliva, lacrimation, (calibration of eardrum)
VIII. Vestibulocochlear: balance, hearing
IX. Glossopharyngeal: buccal membranes, saliva production, taste
X. Vagus: gag reflex, voice production (major parasympathetic component)
XI. Accessory: shoulder elevation, head rotation
XII. Hypoglossal. tongue movement

The musculoskeletal system

Structures associated with the musculoskeletal system
Joints
Muscles
Tendons
Ligaments
Synovia
Bursae

Case history questions
Muscle or joint pain
Stiffness
Swelling
Dislocation
Muscle weakness
Muscle twitches, spasms
Injuries and operations
Autoimmune, connective tissue disease
Medications

Clinical examination
Observe: muscle symmetry, joint deformity, evidence of inflammation
Palpation: muscle tone, wasting, fasciculation
Active movements: compare range of movement, pain, crepitation, locking
Passive movement: compare range of movement, pain, crepitation, locking - compare with active movements
Active-resistance: power, control, pain, instability
Reflexes: present, absent, hyperreflexia, hyporeflexia
Joint-specific assessment: series of evaluative, orthopaedic tests

Miscellanea (skin, thyroid, lymph nodes)

Structures
Skin
Lymph nodes
Lymph vessels
Endocrine glands: pituitary, thyroid, parathyroids, pancreas, adrenals, ovaries, testes.

Case history questions
Skin changes: pigmentation, itching, redness, fragility.
Lymph node swelling, pain, discomfort, fevers, recurring infections.
Pituitary: growing problems, visual changes, headaches, changes in libido.
Thyroid: changes in weight, appetite, tolerance to cold, restlessness, changes to menses, irritability, depression.
Parathyroids: tingling, twitches, cramps, fatigue, constipation, bone problems.
Pancreas: evidence of diabetes
Adrenals: evidence of Cushing's or Addison's disease.
Ovaries: Changes in libido, sexual characteristics, breasts, menstrual cycle.
Testes: changes in libido, sexual characteristics, breasts.
Medications

Clinical examination
Skin: observation, close inspection, woods light, biopsy.
Lymph nodes palpation: pre- and post-auricular, occipital, submental, submandibular, cervical, supraclavicular and infraclavicular, pectoral, axillary, epitrochlear, inguinal, and popliteal.
Inspection and palpation of thyroid gland
Inspection of the pelvic region and genitalia
Palpation of the breasts
Palpation of the abdomen and pelvic regions
Palpation of the testes

Chapter 4

Common Health-Related Complaints

S. R. Prabhu

Abstract

Patients visit medical or dental practitioners seeking diagnosis and treatment for their symptoms and signs of ill health. Based on a patient's presenting complaint, the clinician should be able to form an opinion on the underlying cause of the health problem. In this chapter, some commonly reported complaints are briefly discussed. These include headache, nasal discharge and stuffiness, chest pain, palpitations, shortness of breath and breathlessness (dyspnoea), cough, difficulty in swallowing (dysphagia), indigestion, nausea and vomiting, bloody stools (melaena), constipation, diarrhoea, jaundice, burning sensation on urination, blood in the urine (haematuria), fever, fatigue or tiredness, dizziness/vertigo, hearing loss, significant increase in the volume of urine (polyuria), seizures and tremors, blood stained sputum (haemoptysis) and vomiting blood (haemetemesis).

Introduction

Patients seek medical and dental consultations for a variety of reasons, but the majority seek diagnosis and treatment of health-related complaints, including those involving the head and neck region. Some common complaints are briefly dealt with in this chapter.

Headache

Headache is an extremely common complaint. Based on the cause, there are several types of headaches:

1. Tension headache
2. Migraine
3. Cluster headaches

4. Headaches associated with disorders of the eye, ear, paranasal sinuses, neuralgias, temporomandibular joint problems, intracranial tumours, infections, increased intracranial pressure and those following head injury.
5. Drug-induced, such as those associated with glyceryl trinitrate, nifedipine and substance withdrawal.

It is important to include the following aspects in the case history when assessing headaches:

Onset:

- Sudden onset is due to vascular causes.
- Cluster headaches and migraines intensify over a period of minutes and last for hours.
- Headache due to meningitis lasts for hours and days
- Headache due to increased intracranial pressure (due to tumour or subdural haemorrhage) is progressive and develops over a period of days or weeks.

Site:

- Retro-orbital pain is due to a cluster headache; sinus pain is unilateral; pain in the temporal region accompanied by jaw claudication is due to temporal arteritis; and ocular pain is due to glaucoma.

Character of pain:

- In tension headaches, the pain is described as a tight band around the head.
- In migrainous headaches, the pain is throbbing or dull aching.

Severity of pain:

- Headache due to subarachnoid haemorrhage is very severe and associated with a stiff neck.
- Slow, progressive headache is a feature of slowly evolving intracranial pressure due to tumours.
- In neuralgias, pain is sudden, sharp and severe.

Precipitating factors:

- Posture, coughing and sneezing aggravates pain in the headache due to raised intracranial pressure
- In migraines, photophobia is usually present. Certain foods such as cheese may precipitate migraine
- Common cold can precipitate headache
- Drugs such as glyceryl trinitrate used for angina can precipitate headaches.
- Associated symptoms:

- Flashing lights in migraine and unilateral visual loss occur in temporal arteritis. In hydrocephalus with headaches, dementia, ataxia and drowsiness may occur.

Nasal Discharge and Stuffiness (rhinorrhoea)

Rhinorrhoea manifests as watery eyes, nasal discharge, discomfort in the throat, and itching in the nose, throat and eyes. Viral infections or hay fever can cause rhinorrhoea.

Chest Pain

The following conditions can give rise to chest pain:

- Angina pectoris and myocardial infarction (retrosternal pain is of a crushing type lasting 1-3 minutes in angina, and up to 20 minutes in myocardial infarction)
- Dissecting aneurism of aorta (pain is of a tearing quality)
- Pericarditis (sharp knife-like pain in the precordial region)
- Oesophageal reflux disease (pain is of a burning and squeezing type restrosternally, and mimics the pain of angina)
- Anxiety (dull ache in the precordial region)
- Tracheobronchitis (a burning quality around the sternum associated with a cough)
- Shingles (sharp pain along the dermatome associated with blisters)

Palpitations

Palpitations are an unpleasant awareness of the heartbeat, reported as skipping, racing or fluttering. Palpitations can be felt in cardiac and non-cardiac disorders. Cardiac disorders which may cause palpitations include aortic regurgitations, aortic stenosis, and patent ductus arteriosus.

Transient skips may be due to premature contractions and atrial fibrillations.

Non-cardiac causes include anxiety, thyrotoxicosis, and drugs (epinephrine, for example).

Shortness of breath, or breathlessness (dyspnoea)

Causes of dyspnoea include left-sided heart failure, chronic bronchitis, pulmonary emphysema, bronchial asthma, pneumothorax and lung disease.

Cough

A cough is a reflex response to stimuli that irritate receptors in the larynx, trachea or large bronchi. Coughs can be dry or productive: sputum with or without traces of blood, for example.

Coughing up blood is called haemoptysis. Causes of haemoptysis include bacterial pneumonia, chronic bronchitis, pulmonary TB, lung abscess, lung cancer, left ventricular failure, vasculitis and pulmonary emboli.

Difficulty in swallowing (dysphagia)

Causes of dysphagia include: Oesophageal strictures, oesophageal reflux disease, oesophageal spasm and oesophageal cancer. Odynophagia refers to pain during swallowing.

Indigestion (dyspepsia)

Indigestion is a collective term used for heart burn, excessive gas, an unpleasant fullness after heavy meals, abdominal pain, and nausea and vomiting.

Nausea and vomiting

Nausea is the unpleasant feeling of wanting to vomit. Vomiting is the forceful expulsion of the gastric contents through the mouth produced by involuntary contraction of the abdominal muscles.

Causes of nausea and vomiting include upper gastrointestinal tract disorders, motion sickness, pregnancy, psychological disorders, neurological and metabolic factors.

Bloody stools (melaena)

The production of bloody stools may be caused by disorders of the gastrointestinal tract such as peptic ulcers, oesophageal varices, colon cancer, diverticula of the colon, ulcerative colitis, infectious dysentery, haemorrhoids (most common), and anal fissures.

Red blood (fresh blood) in stools indicates a haemorrhage of the colon, anus or large intestine, whereas black stools (deoxygenated blood in stools) indicate haemorrhage from the upper gastrointestinal tract (as occurs with peptic ulcers, for example), and iron therapy.

Constipation

Constipation refers to the decreased frequency of abnormally firm motions (stools).

Causes include a diet deficient in fibre, irritable bowel syndrome, cancer of the sigmoid colon or rectum, faecal impaction, drugs such as aluminium-containing antacids, iron therapy, pregnancy, hypothyroidism and spinal injuries.

Tenesmus is the feeling of incomplete rectal evacuation with a persistent desire to defecate.

Diarrhoea

Diarrhoea refers to the production of frequent and loose (fluid) stools with a persistent desire to defecate, or faecal incontinence.

Large diarrhoeal stools indicate small bowel or proximal colon disorders. Small and frequent stools with urgency indicate disorders of the left colon or rectum. The condition where large, grey or yellowish, foul smelling fatty stools (which float) are produced is called steatorrhoea. This is suggestive of malabsorption.

Causes of diarrhoea include infections of the gastrointestinal tract, drugs (such as magnesium-containing antacids), irritable bowel syndrome, lactose intolerance, ulcerative colitis, malabsorption syndromes, Crohn's disease, and cancer of the sigmoid colon.

Jaundice

Jaundice refers to the yellowish discolouration of the skin and sclera due to increased amounts of bilirubin (a bile pigment derived from the breakdown of haemoglobin).

Causes of jaundice include viral hepatitis, liver cirrhosis, liver damage due to medications (oral contraceptives, for example), gall bladder disease, and pancreatic cancer.

Urine stained by bilirubin looks dark in colour. Jaundice also may be associated with skin itchiness. This is due to cholestatic or obstructive jaundice.

Burning sensation or pain on urination (micturition)

Inflammation or irritation of the bladder or urethra causes pain on micturition. Men feel it in, or proximal to, the glans penis, whereas females feel the burning sensation in the external labia during micturition. Causes include cystitis (inflammation of the bladder), urethritis (inflammation of the urethra), urinary stones, acute prostatitis, vulvovaginitis, or as a result of foreign bodies or tumours in the bladder.

Blood in the urine (haematuria)

Haematuria refers to the passage of blood in the urine. It is identified by patients as pinkish, brownish or red coloured urine.

Causes include cystitis, malignancy of the prostate, bladder or kidney, urinary stones, trauma, renal tuberculosis and acute glomerulonephritis.

Drugs, such as iron preparations or laxatives, and beetroot meal, can colour urine. Colour can often be due to menstrual bleeding; this should not be mistaken for haematuria. Haematuria can be diagnosed by dipstick testing, and this should then be confirmed by microscopy.

Fever (pyrexia)

Fever or pyrexia refers to the abnormal elevation of body temperature.

Causes include infections, trauma (surgery or a crushing injury), malignancies, infarction, blood disorders, and immune disorders. Often other associated symptoms such as joint pains, nasal congestion, and cough may be present.

Fatigue or tiredness

Loss of energy can be regarded as fatigue.

Causes include anxiety, depression, infectious diseases, diabetes, hypothyroidism, Addison's disease, anaemia, leukaemia, connective tissue diseases, neoplastic diseases, alcoholism, liver and kidney diseases.

Weakness, however, should not be confused with tiredness. Weakness refers to loss of muscular power.

Dizziness and vertigo

An unsteady state, with a feeling of impending fall, is dizziness. Vertigo, on the other hand, is generally a perception that either the self (the patient), or the environment, is rotating or spinning.

Causes of dizziness include anxiety, hyperventilation, cerebrovascular diseases, cardiovascular diseases, anaemia, neurologic disorders, visual disturbances and medications.

Causes of vertigo include trauma or infection of the aural labyrinth, acoustic nerve lesions, brain stem lesions, and cerebral cortex lesions.

Hearing loss

Causes of hearing loss include inner ear disorders, cochlear nerve disorders, obstruction of the ear canal, otitis media, perforation of the ear drum, drugs such as NSAIDs, infections of the inner ear, and aging.

Significant increase in the volume of urine (polyuria)

Causes of polyuria include disorders of the hypothalamus and posterior pituitary gland, kidney diseases, uncontrolled diabetes mellitus, and excessive fluid intake.

Joint pains (arthralgia)

Joint pains can be due to bursitis, tendonitis, monoarticular arthritis or trauma.

In rheumatoid arthritis, pain and swellings occur in interphalengeal joints of the hands and metacarpo-phalengeal joints of the feet. In osteoarthritis, wrists, knees, hips, cervical and lumbar joints are involved. In gouty arthritis, ankles, knees and the base of the big toe are involved.

Seizures and tremors

Seizure is a paroxysmal (a sudden outburst of violent action) disorder that may or may not involve loss of consciousness, but may involve abnormal sensations, movements and thought processes. Tremors are rhythmic, oscillatory movements.

Seizures are common in epilepsy and where there are cerebral cortex lesions present. Tremors are common in Parkinsonism, multiple sclerosis and hyperthyroidism.

Blood stained sputum (haemoptysis)

Expectoration of blood causes blood stained sputum.

Major causes of haemoptysis include pneumonia, bronchial carcinoma, tuberculosis, chronic bronchitis, bronchiectasis, pulmonary oedema, Wagener's granulomatosis, pulmonary embolism, mitral stenosis, hereditary haemorrhagic telengiectasia and coagulation defects.

Vomiting of blood (haematemesis)

Haemetemesis is the vomiting of blood, usually caused by lesions proximal to the duodenojejunal junction.

Causes of haemetemesis include oesophageal varices, epistaxis, reflux oesophagitis, oesophageal carcinoma, peptic ulcer disease, thrombocytopenia, haemophilia, uraemia, connective tissue diseases, and long term use of aspirin, steroids and anticoagulants.

Chapter 5

Systemic Infections

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Abstract

In medical and dental practice, a sizeable proportion of patients seeking consultations include those with systemic infections. The majority of systemic infections in the developed world fall into three categories: bacterial, viral and fungal. In the developing world, in addition to these infections, protozoal and helminthic infections also pose serious threats to health. In this chapter, common symptoms of infectious disease and investigations carried out for their diagnosis are briefly discussed. This is followed by a brief discussion on systemic infections of dental interest. These include scarlet fever, methicillin resistant *Staphylococcus aureus (*MRSA) infections, diphtheria, tetanus, pertussis (whooping cough), legionnaires' disease, rheumatic fever, typhoid fever (enteric fever), infective endocarditis, pneumonia, tuberculosis, syphilis, gonorrhoea, actinomycosis, acute necrotising ulcerative gingivitis and noma, influenza, dengue fever, infectious mononucleosis, HIV disease, measles, mumps, rubella, herpes simplex virus infections, human papillomavirus infections, Coxsackie virus infections, chickenpox, shingles (herpes zoster), candidiasis (candidosis), aspergillosis, histoplasmosis, rhinosporidiosis, malaria, amoebiasis, and helminthic diseases. Antibiotic resistance is also briefly discussed in this chapter.

Introduction

Infection is the invasion of a host's organs and tissues by pathogenic organisms. The host's response to the microbial invasion is manifested in several ways causing disease. Dental practitioners need to possess adequate knowledge of the common symptoms, signs, management protocols, oral manifestations and dental management considerations of systemic infections.

- **Common symptoms of systemic infections**

Symptoms of systemic Infection may include any of the following: Aches and pains, chills, fever, nausea, vomiting, weakness, loss of appetite and tiredness. These may be considered as general constitutional symptoms.

- **Investigations in systemic infections**

Investigations into systemic infections include microscopic, cultural, serological and molecular methods. A full blood count (FBC) to establish a white cell count is also an important measure in investigating infectious diseases.

- **Systemic infections of dental interest**

The following discussion deals with the clinical and management aspects of some of the more common infections and their relevance to dental practice.

Bacterial Infections

- **Scarlet fever**

Definition: Scarlet fever is a streptococcal infection involving the throat, tonsils and skin.
Cause: *Streptococcus pyogenes.*
Symptoms and signs: Fever of sudden onset, sore throat, headache, cervical lymphadenopathy, vomiting, erythematous skin rash on the limbs, and tonsillar exudates. Rarely, scarlet fever may cause nephritis or rheumatic fever.
Oral manifestations: An altered taste sensation and the presence of furred or red tongue (strawberry tongue) are oral features of scarlet fever.
Investigations: Throat swab for microbial identification, and culture and serology for antistreptolysin O (ASO) titre.
Management: Antibiotics, antipyretics and bed rest.

- **Methicillin resistant *Staphylococcus aureus (*MRSA) infections**

Antibiotic resistance is a form of drug resistance whereby microbes, particularly bacteria, are able to survive after exposure to antibiotics. Pathogens resistant to multiple antibiotics are considered multidrug resistant (MDR).
Resistant pathogens: These include *Staphylococcus aureus* (Methicillin resistant *Staphylococcus aureus* (MRSA), Streptococcus and Enterococcus, *Pseudomonas aeruginosa*, *Clostridium difficile*, Salmonella and *Escherichia coli* (E. coli), and *Mycobacterium tuberculosis*. The mechanism of antibiotic resistance includes spontaneous or induced genetic mutation, or acquisition of resistant genes from other bacterial species by horizontal gene transfer. Misuse or overuse of antibiotics may also contribute to the resistant strains. MRSA

infections have become a major concern in recent years. Dental practitioners should be aware of the range of infections caused by MRSA.

Methicillin resistant *Staphylococcus aureus (*MRSA) is an opportunistic pathogen often carried asymptomatically by humans. It is quite common in hospitals and is becoming common in the community as well (Community acquired MRSA). MRSA strains are resistant to methicillin and other beta-lactum antibiotics (penicillins and cephalosporins, for example). MRSA strains, particularly those acquired in hospitals, are often resistant to other antibiotics as well.

Methicillin sensitive and methicillin resistant strains can be found on the normal skin of axillae, the perineum and on the mucosal surfaces of anterior nares and nasopharynx. The incubation period for MRSA can vary from four to ten days.

Clinical signs of MRSA infections: These include impetigo, folliculitis, furunculosis, cellulitis, abscesses and wound infections. When infection is invasive, MRSA can cause pneumonia, endocarditis, septic arthritis, osteomyelitis, meningitis and septicaemia. An infected person can transmit, by direct contact, infection to other people, and also to a variety of animal species.

Diagnosis/investigations: Diagnosis can be confirmed by culturing the infected site. Staphylococcal food poisoning can be detected by examination of the food for organisms and/or toxins. If *S. aureus* is isolated from an infection, genetic testing or antibiotic susceptibility testing should be done to identify MRSA. Fluoroquinolone-resistant *S. aureus* strains should be suspected of being MRSA.

Management: Skin infections such as abscesses should be drained. Invasive infections need antibiotics which include vancomycin and more recent drugs such as linezolid, tigecycline, quinupristine/dalfopristin and daptomycin.

Prevention measures include good hygiene (hand washing), environmental cleaning and disinfection, and isolation precautions for MRSA-infected hospitalised patients. Skin lesions and wounds must be covered with clean, dry bandages.

- **Meningitis**

Definition/Description: Meningitis is inflammation of the meninges of the brain or spinal cord. Common forms are acute bacterial meningitis and aseptic meningitis. Based on the onset and duration of the disease, meningitis can be classified as acute, subacute and chronic.

Causes: The most common cause of meningitis is infection. Common organisms involved are meningococci (*Neisseria meningitidis*), pneumococci *(Streptococcus pneumoniae),* and group B streptococci in infants. These are responsible for acute bacterial meningitis. Aseptic meningitis is usually caused by viruses, although fungi and bacteria may also be involved.

Symptoms and signs: The classic triad of meningitis is fever, headache and nuchal rigidity. These symptoms can develop within hours or days. Passive flexion of the neck is restricted and painful, whereas extension and rotation is not. In severe cases, attempts to flex the neck may induce flexion of the hip or knee. This symptom is called Brudzinski's sign. Other symptoms include lethargy, confusion and seizures. In acute bacterial meningitis, CSF may be purulent, and the condition is fatal if not treated promptly. Aseptic meningitis is milder and self-limiting.

Oral Manifestations and Dental Management Considerations

There are no specific oral manifestations apparent in acute meningitis. In the chronic form of meningitis, spinal or cranial nerve root deficits are common. Due to the stiffness of the neck, any dental treatment should be undertaken with care. A physician's consultation is required for these cases.

Diagnosis and investigations: Blood cultures are obtained, and a lumbar puncture is done to obtain CSF for culturing, gram stain, cell count, differential and estimation of glucose and protein content. CSF tests should also include TB and HIV investigations.

Management: Active meningitis is a medical emergency where patients need to be hospitalised. Antibiotics (cephalosporins, vancomycin and ampicillin, for example) are started as soon as blood culture samples are drawn.

- **Diphtheria**

Definition: Diphtheria is an acute pharyngeal or cutaneous infection caused by *Corynebacterium diphtheriae.*

Cause: *Corynebacterium diphtheriae* through droplet spread.

Symptoms and signs: Sore throat, swollen tonsils with a yellow/red membrane, tachycardia, high fever, husky voice and nasal discharge. A swollen neck may be seen in advanced cases.

Oral manifestations: A yellow or red membrane may extend from the tonsillar area on to the soft palate, causing considerable discomfort to the patient.

Investigations: Culture of a throat swab.

Management: An intramuscular injection of diphtheria anti-toxin, to be immediately followed by parenteral administration of antibiotics for two weeks.

Prevention: Prevention of diphtheria is by active immunisation (DPT) of 3- to 12-month-old children. In the event of an outbreak, carriers should be identified, isolated and treated with antibiotics for a week.

- **Tetanus**

Definition: Tetanus is a non-transmissible bacterial disease, caused by *Clostridium tetani,* which can be fatal if not treated promptly.

Cause: A gram positive bacillus, called *Clostridium tetani,* a commensal organism of the intestines or found in the soil, is the causative agent of tetanus. This organism may contaminate the wound where it remains localised and produces exotoxin. Exotoxin then spreads to central nervous system causing serious disease. The incubation period ranges from 1-15 days.

Symptoms and signs: Muscle spasms resulting from unexpected noise or touch can cause asphyxia. Muscle spasms of the trunk cause opisthotonus (arching backwards). Fever is a common symptom. Cardiac complications also occur, such as arrhythmias and swings in blood pressure.

Oro-facial manifestations: Masseter muscle spasm results in trismus (lock jaw). Myalgia, and rigidity of the face and neck, can result in a characteristic facial appearance called 'risus sardonicus' (raised eye brows and an open grin).

Investigations: Clinical features are suggestive of the disease. In patients who have had no history of tetanus toxoid immunisations and clinical symptoms, a history of trauma resulting in a wound during the past two weeks offers important clues to the diagnosis. Laboratory diagnosis includes the isolation of *Cl. tetani* through culture studies.

Management: Intravenous or intramuscular administration of human tetanus antitoxin, wound toilet to prevent further absorption of toxin, administration of antibiotics, and nursing in a quiet room are all important aspects of this disease's management. Intubation and artificial ventilation may be necessary.

- **Pertussis (whooping cough)**

Also known as whooping cough, Pertussis is a highly infectious disease caused by the bacteria *Bordetella pertussis.*

Cause: *Bordetella pertussis* is spread by droplet infection. Incubation period is from 7-14 days.

Symptoms and signs: Pertussis is usually an infection of pre-school children. It starts with a runny nose, conjunctivitis and an unproductive cough, then proceeding to coughing bouts.

Oral manifestations: Erosion of the lingual frenum and haemorrhagic spots on the palate are often encountered in children with whooping cough

Investigations: Clinical features are suggestive. A FBC (lymphocytosis in the early stages) and culture from perinasal swabs are diagnostic.

Management: Administration of antibiotics, cough suppressants, hydration, ventilation and nutrition. Prevention includes active immunisation of pre-school children (DPT).

- **Legionnaires' disease**

Definition/description: Legionnaires' disease is a flu-like disease caused by a bacterium called *Legionella pneumophila.*

Cause: Infection is transmitted by inhalation of aerosolised water or soil contaminated with the gram negative, aerobic bacteria. Water tanks, cooling towers and evaporative condensers of large air-conditioning systems can act as sources of infection.

Symptoms and signs: These include flu-like symptoms with acute fever, chills, myalgia, malaise and joint pains. These may proceed to pneumonia characterised by dyspnoea, pleuritic pain and haemoptysis.

Diagnosis/investigations: Diagnosis is by examination of the sputum or bronchoalveolar lavage fluid for direct fluorescent antibody staining. Polymerase chain reaction (PCR) with DNA probing is also useful. Chest x-rays for pleural effusion and lung infiltrates are useful in detecting pneumonic manifestations.

Management: Treatment includes administration of drugs such as doxycycline, macrolides or fluoroquinolones.

- **Rheumatic fever**

Definition: Rheumatic fever is an abnormal immune response to an acute infection caused by *Streptococcus pyogenes.*

Cause: The organism *Streptococcus pyogenes.*

Symptoms and signs: Children between 5 and15-years old are susceptible. Infection is characterised by fever, pains in large joints (acute and migrating), pericarditis, endocarditis and skin rashes.

Investigations: Detection of high titres of streptococcal antibodies (Antistreptolysin O titre), raised ESR, mild leukocytosis, and anaemia are useful diagnostic indicators of rheumatic fever.

Oral manifestations: There are no known specific oral manifestations of rheumatic fever. It must be noted, however, that the risk exists of promoting infective endocarditis during dental treatment in patients with rheumatic heart valve damage.

Management: Antibiotics, analgesics/antipyretics and bed rest.

- **Typhoid fever**

Definition: Typhoid, also known as enteric fever, is caused by a gram negative bacillus called *Salmonella typhi.*

Cause: *Salmonella typhi,* found in contaminated food.

Symptoms and signs: These include lethargy, fever, head ache, dry cough, macular rose spots, dehydration, splenomegaly and gastrointestinal bleeding.

Oral Manifestations: No specific oral manifestations of the disease are known. Occasionally, oral ulcerations may accompany typhoid fever.

Investigations: Blood cultures (in the first and second weeks of the disease) and stool cultures (in the second week of the disease) test positive for the disease.

Management: Appropriate antibiotics, antipyretics and rehydration.

- **Infective endocarditis**

Definition: Infective endocarditis is an infection of the heart valves or endothelium, usually by streptococci or staphylococci, and very occasionally by fungi, mycoplasma or chlamydiae.

Cause: Microorganisms as mentioned above. *Viridans streptococci* remain the single most common group of bacteria responsible for infective endocardiitis.

Symptoms and signs: These include fever, malaise, anorexia and anaemia. Septic emboli on the heart valves, and defective heart valves, may cause heart failure.

Oral Manifestations: Although there are no specific oral manifestations of the disease, sources of infection may include bacteraemia from oral sepsis, or from oral surgical procedures.

Investigations: Blood cultures for identification of the causative organisms, cardiac ultrasound for detection of abnormal heart valves, or vegetations and C-reactive proteins for ongoing infection, are diagnostic.

Management: Antibiotics, antipyretics, heart valve replacement and local treatment of dental sepsis.

- **Pneumonia**

Definition: Pneumonia is the term used to denote inflammation of the lung caused by bacterial and viral agents. This acute condition affects one or more segments of the lung, in which case it is referred to as lobar pneumonia.

Cause: The largest proportion of pneumonias is caused by *Streptococcus pneumonia* (pneumococcus). Infection is usually acquired by inhalation of pneumococcus from a victim of the disease, or from 'carriers' of the organisms.

Symptoms and signs: A sudden rise in body temperature, headache and body aches are initial symptoms. This stage is soon followed by a localised pain of pleural type and a short, painful cough, rapid respiration and rapid pulse. Sputum is either rust-coloured or blood stained. Chest x-rays show homogeneous opacity confined to the affected lobe.

Oral Manifestations: There are no specific oral manifestations reported for pneumonia, however, herpes labialis may occur in these patients due to the activation of the herpes simplex virus.

Investigations: Clinical features and radiographic findings are suggestive of the infection. Identification of the organisms in sputum is an important step in the diagnosis of pneumonia.

Management: Penicillin is the treatment of choice. Other therapeutic agents include clyndamycin, erythromycin and cyclosporine. Pneumococcal vaccine is helpful in prevention of the disease.

- **Tuberculosis**

Definition: Tuberculosis is a chronic infectious disease caused by *Mycobacterium tuberculosis.*

Cause and pathogenesis: An acid-fast bacillus, *Mycobacterium tuberculosis*, can gain entry into the body via the skin, respiratory tract or gastrointestinal tract. Individuals at high risk include those with poor standards of living, are HIV positive, or alcoholic. In the majority of cases, the primary infection is via the lungs as a result of droplet infection.

A primary point of infection is formed in the lung, called a Ghon focus. A caseous involvement of mediastinal lymph nodes occurs. The Ghon focus and the caseous involvement of the lymph nodes are collectively called a 'primary complex'. In a large majority of cases, the primary complex heals and calcifies, and the individual remains asymptomatic. If the healing is not complete, progressive tuberculosis may result. A haematogenous spread of infection, called miliary tuberculosis, can occur, involving several other organs including bone marrow, lungs, kidneys, joints, heart and brain.

Symptoms and signs: These vary depending on the systems involved.

General features include weight loss, tiredness, night sweats, loss of appetite, and fever.

Pulmonary involvement causes a productive cough, haemoptysis, breathlessness, hoarseness and bronchopneumonia. Diarrhoea and intestinal obstruction can occur in intestinal tuberculosis.

Skin involvement of tuberculosis is called lupus vulgaris.

Lymph node involvement is known as scrofula.

Heart involvement includes cardiac arrhythmias and pericarditis. Tuberculoma and meningitis are seen in brain involvement.

Oral manifestations: Primary tuberculosis infection of the oral soft tissues is extremely rare. Secondary involvement of oral tissues can, however, occur in patients with pulmonary tuberculosis. These include tuberculous ulcers on the dorsum or lateral borders of the tongue. Maxillary or mandibular involvement may result in tuberculous osteomyelitis. Salivary gland infection can also occur in rare cases.

Investigations: These include chest x-rays, chest tomography, CT scans, the Tuberculin test (Mantaux test), a bacteriological examination (Ziehl-Neelsen stain), culture studies, laryngeal swabs, gastric aspirations, ESR and FBC.

Management: Anti-tuberculosis chemotherapy includes rifampicin, Pyrazinamide isonizid, and streptomycin. BCG vaccination is given to prevent infection.

- **Leprosy**

Definition: Leprosy is a chronic infectious disease commonly affecting the skin and peripheral nerves, and if not treated in the early stages, can result in considerable disfigurement. The disease is common in the less developed tropics.

Cause: The causative organism of leprosy is an acid-fast bacillus called *Mycobacterium leprae.*

Symptoms and signs: Clinical features of the disease show a wide range of variation.

In the early stages of the disease, skin lesions include hypopigmented macular lesions with impairment of sensation (indeterminate leprosy). This stage is followed by thickened nerve trunks and skin lesions with hypopigmented patches (tuberculoid leprosy). As the disease advances, extensive involvement of the skin and nerves (borderline leprosy), occurs. Bone, mucous membranes, lymph nodes, skeleton, testes and other internal organs are involved in the advanced stages (lepromatous leprosy).

Oro-facial manifestations: In the early stages of the disease, skin lesions on the face may occur, as well as lepromatous infiltration of the helices and lobes of the ears. Involvement of the facial nerves results in a range of clinical features which include paresthesia, facial paralysis, and facial disfiguration due to involvement of facial cartilage and bone. Typical facies, called *facies leprosa,* is characterised by atrophy of the anterior nasal spine and anterior maxillary alveolar process. Collapse of the nose (saddle nose) is a common feature.

Intraoral soft tissue involvement includes ulceration, atrophy, necrosis and fibrous repair of the soft palate and uvula resulting in functional impairment. The tongue shows lepromatous infiltrations giving rise to a ‘cobble stone’ appearance.

Investigations: Skin smears, nasal smears, skin and nerve biopsies and lepromin tests are useful investigations in confirming the clinical diagnosis.

Management: Dapsone, clofazimine, rifampicin and prothionamide have been used in the management of leprosy. These are leprostatic agents and do not cure the disease. Disfigured parts of the body need reconstructive and plastic surgery.

- **Syphilis**

Definition: Syphilis is a disease caused by a spirochaete called *Treponema pallidum*. The disease is divided into two forms: Congenital syphilis and acquired syphilis. The acquired form is a sexually transmitted disease (STD).

Cause: *Treponema pallidum* is the causative agent transmitted through sexual routes. The incubation period ranges from 10 to 90 days.

Symptoms and signs:

Congenital syphilis: may cause spontaneous abortions, or still births. Bone and teeth abnormalities, failure to thrive, and eighth nerve deafness are some other features of congenital syphilis.

Acquired syphilis is further divided into early (primary, secondary and latent forms) and late syphilis (latent, tertiary and quaternary).

- Primary syphilis: In this stage, papules appear at the site of entry of the spirochaetes (genitals, lips, nipples, etc.) and which become painless ulcers. Lesions of primary syphilis are called chancre, and these are highly infectious lesions with a rubbery consistency. Regional lymphadenopathy is present. Chancres resolve within 6-8 weeks, sooner if treatment is instituted.
- Secondary syphilis: This stage is characterised by fever and the occurrence of a generalised macular rash of the skin (palms and soles as well); flat, papular lesions (condyloma lata) on moist areas (the perianal region, for example); painless, generalised lymph node enlargement; mucosal superficial ulcers (snail track ulcers); arthritis; and hepatosplenomegaly. Secondary syphilis begins 1-4 months after the disappearance of the chancre(s). Clinical manifestations may resolve in the absence of treatment and the patient may enter a stage of latency (latent stage) which can last several years.
- Tertiary syphilis: Gumma formation of the bone, skin and subcutaneous tissues is a characteristic feature of this stage of the disease and can occur many years after the primary infection. From here on, the disease can progress to the quaternary stage.
- Quaternary syphilis: Cardiovascular and nervous systems are affected in this stage. Cardiac involvement may result in aortic dilatation. Tabes dorsalis is the feature of nervous system involvement characterised by demyelination of the sensory neurons. The degenerating nerves are in the dorsal columns of the spinal cord. Endarteritis obliterans is another feature of the disease at this stage. This is characterised by the obliteration of the lumen of the arteries.

Oral Manifestations

- In primary syphilis, chancres can develop on oral mucosa. The upper lip is usually the site of occurrence in males, and the lower lip in females. Oral chancres are indurated and often ulcerated lesions.
- In secondary syphilis, moist papules and mucous patches appear on the oral mucosa concurrently with skin lesions. These ulcerate and give rise to a glistening, white

appearance. These are called 'snail track' ulcers. These lesions are highly infectious. Regional lymphadenopathy is present at this stage of the disease.

- In tertiary syphilis, oral manifestation may occur at any time from three to ten years after the primary infection. A typical lesion is a gumma, characterised by an ulcer usually on the palate. Occasionally, this may result in gummatous perforation. On the tongue, syphilitic involvement may be seen as syphilitic glossitis (also known as chronic superficial interstitial glossitis) and characterised by a dull red, bald, smooth and glistening dorsum of the tongue.

Investigations: Common tests include: dark field microscopy of the fluid obtained from ulcers (identification of *T. pallidum*), serology (positive one month after infection), Venereal Diseases Research Laboratory (VDRL) test, rapid plasma reagin (RPR) test, and *Treponema pallidum* haemagglutination test (TPHA) or fluorescent treponemal antibody absorption (FTA-abs) tests.

Management: This involves administration of antibiotics (penicillin, or tetracycline for those allergic to penicillin). Contact tracing is an important aspect of preventive measures.

- **Gonorrhoea**

Definition: Gonorrhoea is a sexually transmitted disease caused by the bacteria *Neisseria gonorrhoeae.*

Cause: A gram negative bacteria called *Neisseria gonorrhoeae*. The incubation period ranges from 2-10 days.

Symptoms and signs: Gonorrhoea is characterised by urethritis, painful urination (dysuria) and genital maculopurulent discharge, inflammation of prostate glands, salpingitis (infection and inflammation in the fallopian tubes), pelvic inflammatory disease, pustular skin rash, arthritis, and pharyngitis in male homosexuals.

Oral manifestations: Often, involvement of the oropharyngeal region in gonorrhoea is asymptomatic. Gonococcal stomatitis and pharyngitis may occur with erythematous and oedematous lesions on the pharynx and uvula. Ulcerative lesions on the tongue may also occur in some cases. Oral involvement is usually found among patients practicing oral sex. Temporomandibular joint involvement is often reported in patients with gonococcal arthritis.

Investigations: Gram stain and culture of discharge or blood.

Management: Antibiotics: ampicillin, erythromycin or ciprofloxacin.

- **Actinomycosis**

Definition: Actinomycosis is a chronic infectious disease caused by the organisms of the order of Actinomycetales. This is not a contagious disease

Cause: *Actinomyces israeli* is the pathogen responsible for the clinical manifestations of the disease.

Clinical features: Four clinical types of actinomycosis occur. They include cervicofacial, abdominal, thoracic and cutaneous types. Cervicofacial actinomycosis is the most common form of the disease.

Cervico-facial manifestations: The first sign of cervicofacial actinomycosis is characterised by the appearance of a palpable mass at the angle of the jaw or in the vicinity of

the parotid gland. Initially this mass is reddish-purple which eventually fluctuates and may drain through sinuses. The material drained is called 'sulphur granules'. Actinomycosis does not spread to the regional lymph nodes. Occasionally jaw bones, salivary glands, periapical tissues and the tongue may be involved in cervicofacial actinomycosis.

Investigations: Clinical presentation, identification of 'sulphur granules' from the drained material, and isolation of anaerobic organisms through culture studies are adequate measures in the diagnosis of actinomycosis.

Management: Large amounts of penicillin for a long period until signs disappear, and for a short period afterwards. Erythromycin, cephalosporin and clindamycin are other agents used in the treatment of actinomycosis. Exploration and drainage of the sinus tracts needs to be carried out to facilitate antibiotic therapy.

- **Necrotising ulcerative gingivostomatitis**

Definition: Necrotising gingivostomatitis (NUG) is characterised by inflammation of the gingival tissues with formation of ulcerations consequent to necrosis of the gingival papillae and their margins.

Causes and predisposing factors: Necrotising ulcerative gingivostomatitis is caused by a mixed flora of organisms including *Treponema* species, *Fusobacterium* species and *Bacteroides melaningenicus*.

Predisposing factors include pre-existing gingivitis, poor oral hygiene, low-socioeconomic status, malnutrition and emotional stress. NUG is more common in immunocompromised patients.

Symptoms and signs: Crater-like ulcerations of the gingival papillae which may extend into the marginal gingiva, pain, halitosis and bleeding from ulcers are hallmarks of this condition. Lymph node enlargement, fever and malaise may occur in advanced stages of the disease.

Investigations: Clinical features are suggestive of the disease. A smear from a lesion may show predominance of the fuso-spirochaetal population. Biopsy of the lesions is not recommended as histopathology is not pathognomonic.

Management: Debridement combined with topical use of oxygenating antiseptics (hydrogen peroxide, for example) are useful. Metronidazole is the antibiotic of choice**.** NUG has a tendency to recur.

- **Gangrenous stomatitis (Noma; cancrum oris)**

Definition: Gangrenous stomatitis is a progressive and mutilating disease of the oro-facial structures occurring in young children in certain parts of the world.

Cause and predisposing factors: Microbial factors as well as malnutrition, stress and exanthematous disease are etiologically associated with gangrenous stomatitis. Microbes found in the disease include *Fusiformis fusiformis, Borelia vincenti and Bacteroides melaninogenicus (now named as B. asacharasolyticus)* and are found to have a causal relationship.

Symptoms and signs: In its early stage, the disease is characterised by the occurrence of popular red or purplish eruptions on the alveolar margin which are generally located in the premolar or molar region. Ulceration follows and spreads rapidly to the vestibular region. The

patient complains of a sore mouth and salivates profusely at this stage of the disease. Halitosis is intense. As the disease enters the advanced stage (in a matter of two or three days), extra oral skin shows blue-black discolouration. This is immediately followed by a gangrenous phase which is characterised by the appearance of slough, a perforating wound and exposure of the teeth and bone.

Investigations: Clinical findings are suggestive of the disease and special investigations are generally not required.

Management: In acute stages of the disease, hospitalisation, administration of intravenous fluids and the use of antibiotics such as penicillins or sulphonamides, local care of the wound, and feeding are essential. Reconstructive surgery is recommended for healed cases.

Viral Infections

- **Influenza**

Definition/description: Influenza (Flu) is a common viral infection of the respiratory system.

Cause: The disease is caused by the influenza viruses Types A and B. The incubation period ranges from 1 to 4 days. Influenza spreads by airborne droplets, person to person contact, or contact with contaminated items.

Symptoms and signs: Typical features include: sudden chills, fever, cough, generalised aches and pains, headache, and photophobia. Initial symptoms include sore throat, acute nasal congestion (coryza), headache, and a feeling of general discomfort or uneasiness (malaise). The elderly, and those with cardiopulmonary diseases and immune deficiencies, are vulnerable to develop pneumonia which can sometimes be fatal.

Diagnosis/investigations: Diagnosis is usually clinical. In the high risk group with clinical signs of respiratory distress, chest x-rays may be necessary to rule out pneumonia.

Management: Most patients recover fully within one or two weeks. Symptomatic treatment such as antipyretics, rest and hydration are adequate for the majority of patients. Aspirin should be avoided in children. For patients with complications such as pneumonia, antibacterial treatment is necessary. Antiviral treatment is also recommended for those in the high risk category. Influenza can be prevented by annual vaccination. Vaccines contain two strains of influenza A and one strain of influenza B virus. Vaccination is indicated for persons over 65 years of age, and those with chronic diseases including diabetes and cardiopulmonary disorders.

Avian influenza (Bird flu): Avian influenza, also called bird flu, is caused by strains of influenza A that normally infect wild birds and sometimes pigs. Infections in humans have been detected in recent years. Avian influenza type H5N1 is the major causative virus for human infections, although H7N7, H7N3 and H9N2 have also been associated with human infections.

Swine influenza (Swine flu): Swine flu is caused by one of several types of the swine viruses that are endemic in pigs. Occasionally human infection can occur, particularly in persons who are regularly in contact with pigs. Swine influenza virus (SIV) strains include subtypes of the influenza A virus (H1N1, H1N2, H2N1, H3N1, H3N2 and H2N3) and

influenza C virus. Symptoms of swine flu in humans are similar to those of influenza (Flu). Antiviral treatment can make the illness milder. Supportive care for fluid balance, fever, headaches and pains is also necessary. A common cause of death is respiratory failure. Most patients recover without any antiviral treatment. Oseltamivir (Tamiflu) or zenamivir (Relenza) are used for both prevention and/or treatment of swine flu.

Severe Acute Respiratory Syndrome (SARS): SARS is caused by a coronavirus and is spread by respiratory droplets. With an incubation period of between two and ten days, SARS produces an influenza–like illness that can progressively lead to respiratory insufficiency. The mortality rate of SARS is 10%. Patients need to be isolated in order to prevent the spread of the infection. Supportive treatment is necessary.

- **Dengue fever**

Definition/description: Dengue fever is an infectious disease of the tropics caused by the dengue virus.

Cause: Dengue virus, which is an RNA virus of the family *Flaviviridiae***,** is transmitted by several species of mosquito of the genus *Aedes* (*A. aegypti* in particular).

Symptoms and signs: Dengue fever can be asymptomatic in the majority of patients. The incubation period is three to 14 days. Children often display symptoms similar to the common cold along with gastroenteritis. Symptomatic patients complain of the sudden onset of fever, headache, muscle and joint pains, and skin rash.

During the febrile phase, petechiae occur in the mouth and nose. This may proceed to severe haemorrhage of the gastrointestinal tract (dengue haemorrhagic fever) and shock. Fluid accumulation in the thorax and abdominal cavity can occur in this phase. In a small number of cases the disease can be life threatening due to bleeding, low blood pressure, low platelet numbers and leakage of plasma, or due to dengue shock syndrome.

Diagnosis/investigations: Dengue is diagnosed on clinical grounds. Tests for dengue virus-specific antibodies, (IgG and IgM antibodies) are useful in confirming the clinical diagnosis.

Management: There are no specific antiviral drugs for dengue. Symptomatic treatment includes paracetamol (Ibuprofen and aspirin are to be avoided because of the increased risk of associated bleeding)**,** intravenous and oral hydration and blood transfusion in patients presenting with unstable vital signs. Prevention is through vector control programs. Infection with one type of the dengue virus offers lifelong immunity to that type, but short term immunity to other types of the virus.

- **Infectious mononucleosis**

Definition: Infectious mononucleosis is also known as glandular fever or kissing disease. This is caused by the *Epstein-Barr virus* (EBV).

Cause: EBV through close contact such as kissing. This disease is commonly encountered in young adults.

Symptoms and signs: Infection is characterised by fever, sore throat, malaise, and cervical lymphadenopathy. Hepatosplenomegaly, anaemia and meningitis may occur in rare cases.

Oral Manifestations: Blood blisters on the palate may be present.

Investigations: WBC count (leukocytosis), identification of a heterophile antibody that can agglutinate sheep RBCs (Paul Bunnell test) and EBV antibody titres (IgM and IgG).

Management: the disease is self-limiting. Alcohol consumption should be avoided until the patient recovers fully. Symptomatic treatment for fever and rest are adequate.

Note: The Epstein Barr Virus (EBV) has also been etiologically associated with the development of Burkitt's lymphoma and nasopharyngeal carcinomas. Discussion on these entities can be found elsewhere in the book.

- **Human immunodeficiency virus/Acquired immunodeficiency disease (HIV/AIDS)**

Definition: HIV is a sexually transmitted disease caused by human immunodeficiency virus (HIV) infection. Bacterial, fungal and viral infections, protozoal infestations and malignancies are common in HIV/AIDS patients due to immunodeficiency.

Cause: An RNA virus, called human immunodeficiency (HIV) virus, is the cause of immunodeficiency. Infection is transmitted through sexual activity. Non-sexual transmission of the disease also occurs. This mode of infection includes mother to child transmission (also called vertical transmission) and intravenous use of recreational drugs. Blood transfusion of infected blood can also cause disease but this is extremely rare.

Symptoms and signs: Depending on the stage of the HIV disease, the following symptoms may be encountered: malaise, fatigue, fever, weight loss and diarrhoea. **Signs** include lymphadenopathy, wasting and splenomegaly. The timeframe between the exposure of a person to HIV, and the time the test becomes positive for HIV antibodies, can be between three and six months. This period of time is called the "window period" for HIV testing.

Oral Manifestations: There are no specific oral manifestations caused solely by the human immunodeficiency virus. However, a wide range of oral diseases occur in HIV/AIDS patients, mainly due to immunodeficiency caused by the virus. These include opportunistic infections such as fungal, viral and bacterial infections, neoplasms and a few other diseases associated with the HIV disease. Oral diseases seen in HIV/AIDS patients are listed below:

Fungal infections:

- Pseudomembranous candidiasis
- Erythematous candidiasis
- Angular cheilitis
- Hyperplastic candidiasis
- Histoplasmosis
- Cryptococcosis

Viral infections:

- Herpes simplex virus infections
- Herpes zoster infection (Shingles)
- Human papillomavirus infections

- Cytomegalovirus infections
- Hairy leukoplakia

Bacterial infections:

- Acute necrotising ulcerative gingivitis
- Mycobacterium avium intracellulare
- Neoplastic lesions:
- Kaposi's sarcoma
- Lymphoma
- Other lesions associated with HIV disease:
- Oral ulcerations (recurrent aphthous ulcers)
- Linear gingival erythema
- Salivary gland disease and xerostomia
- Thrombocytopenic purpura
- Mucosal pigmentations due to HIV medications

Detailed descriptions of the above listed lesions are beyond the scope of this section and the reader is referred to standard oral medical textbooks for further information.

Dental management considerations: There is no evidence to support alterations in oral health care based solely on HIV status, however, practicing dentist should be aware of the patient's susceptibility to infections, impaired haemostasis, drug actions and interactions, and their ability to withstand the stress.

Priorities of a dental treatment plan include relief of pain, prevention of further oral disease, restoration of function and aesthetics, and improvement in quality of life.

The following factors should be considered in the management of dental disease:

- Avoid acetaminophen in HIV/AIDS patients with severe liver disease.
- Recognise the side-effects of, and drug interactions with, antiretroviral medications.
- Be aware of the patient's risk of infection and bleeding tendencies.

Haematology: Obtain complete blood counts (CBC), platelet counts, differential blood cell counts, liver enzymes and coagulation tests. Many HIV+ patients are neutropenic, thrombocytopenic, and anaemic. Values indicate susceptibility to infection and bleeding. Tests should be repeated at three to six month intervals.

Antibiotic prophylaxis is indicated with neutrophils <500 cells/mm^3. Many clinicians use the American Heart Association regimen, however, others feel that antibiotic therapy should continue for as long as open wounds are present in the oral cavity. The need for antibiotic prophylaxis is not based on CD4 count.

Non-specific transaminases (liver enzymes: ALT, AST values) are often elevated with acute liver disease present in HIV disease. Marked elevation may indicate decreased liver function and patients may be prone to haemorrhage. Drug metabolism may be impaired in these patients. Significantly elevated coagulation test results may require the modification of dental treatment.

CD4 Count indicates HIV progression and the degree of immune suppression. A normal CD4 count is 800-1000 cells/mm^3, while a CD4 cell count <200 cells/mm^3 is an AIDS defining diagnosis. Major opportunistic infections are also frequently seen with CD4 cell counts of <200 cells/mm^3. In the absence of opportunistic diseases**,** the CD4 count alone is not an indicator to withhold dental treatment

Plasma Viral Load indicates the degree of viral replication and suggests the level of immune suppression, which reflects the destruction of CD4 lymphocytes, and indicates the measure of therapeutic (HAART) success or failure. This also offers prognostic information. The higher the viral load, the faster the progression of HIV disease and the poorer the long term prognosis. However, in the absence of opportunistic diseases, the magnitude of the viral count alone is not an indicator to withhold dental treatment for the patient.

Local anaesthesia. Local anaesthetics can be used but deep block injections should be avoided.

Caries treatment. Most principles of conservative (operative) dentistry for HIV positive patients are similar to those of the general population. Poor candidates for extensive restoration include those with rampant caries, reduced salivary flow, oral acidity, alteration in taste, compromised motor skills, upper airway obstruction and poorly controlled oral infections and other diseases. If recurrent caries cannot be controlled, extensive crown and bridge work should be avoided.

Endodontic treatment appears to offer many benefits for HIV patients. These include reduced infection risk, reduced need for extraction, improved ability to chew and improved self-esteem. Consider using one-step endodontic therapy when appropriate.

Periodontal treatment: Periodontal disease is not more common in HIV disease, But it is often more severe and difficult to control. A shorter recall period is recommended. Debridement, antimicrobial rinses and the use of antibiotics may be necessary.

Oral surgery: Collaborate with other members of the primary care team. Routine antibiotic use is contraindicated**.** Haemostatic function assessment is indicated before extensive surgery**.** Aseptic technique reduces post-procedural complications**.** The incidence of complications is no higher in the HIV+ population. If haemoglobin is > 7g/dl, no increased complications with routine treatment are required. If haemoglobin is < 7g/dl, conservative treatment is recommended. If extensive surgical treatment is required, consult the patient's physician. Avoid NSAIDs, including aspirin, in HIV/AIDS patients with impaired haemostasis.

Orthodontic and prosthodontic considerations: Most principles are similar to those of the general population.

Preventive treatment is highly emphasised early in the HIV disease process. Prophylaxis, fluoride treatment and sealants are highly recommended. The recall interval should be appropriate to maintain good oral health.

Xerostomia: An increased caries rate is found in HIV patients due to xerostomia. Diet modification, fluoride treatment, frequent recall interval and the use of sialogogues are recommended.

Confidentiality: At all times, confidentiality must be maintained for all patients, regardless of HIV serostatus. Proper consent should be obtained before any confidential medical or dental information is released to other medical or dental providers.

Infection control: In treating HIV positive patients, as for other patients, strict infection control protocols need to be followed.

Needle stick injuries: When they occur, appropriate reporting and post exposure prophylactic measures must be carried out without delay.

Investigations: Detection of HIV antibodies in serum by an enzyme-linked immunosorbent assay (ELISA), followed by a Western Blot test **(**confirmatory). HIV antigen levels can be measured in blood using a polymerase chain reaction (PCR) test. An ESR, FBC (anaemia, thrombocytopenia), CD4 count (low), and viral load are usually performed in suspected HIV patients. Two symptoms, two signs and two positive tests (shown above) are required to make a diagnosis of an AIDS-related complex.

Management: Three groups of antiretroviral drugs are available:

1. Nucleoside reverse transcriptase inhibitors (NRTIs) - Zidovudin,
2. Non-nucleoside reverse transcriptase inhibitors (NNRTIs) - Nevirapine and
3. Protease inhibitors (PIs) - Indinavir.

Combinations of NRTI and PI are more effective; this therapy is called highly active antiretroviral therapy (HAART).

Symptomatic and specific treatment modalities for infections and neoplasms are necessary. Avoidance of risk factors of HIV transmission is important.

- **Measles**

Definition: A highly contagious viral disease caused by paramyxovirus infection.

Cause: Paramyxovirus. The incubation period is 7-14 days. Infection spreads by droplets and is common in childhood.

Symptoms and signs: Measles is characterised by fever, cough, runny nose, conjunctivitis and irritability. Rash behind the ears may spread to the face and other parts of the body.

Oral manifestations: Clustered, white spots on the buccal mucosa near each Stensen's duct (Koplik’s spots) are seen on the cheek mucosa.

Investigations: Clinical diagnosis is suggestive of the disease. IgM and IgG antibodies can be measured.

Management: Symptomatic treatment includes paracetamol for fever. Isolation from school and active immunisation of children is recommended. Once attacked by measles, immunity lasts lifelong.

- **Mumps**

Definition: Mumps (parotitis) is an acute contagious disease characterised by a non-suppurative enlargement of one or both parotid glands. The disease occurs frequently in children.

Cause: The mumps virus is an RNA virus which belongs to the family of paramyxoviridiae. The virus is transmitted by droplet spread or direct contact with infected saliva. It has an incubation period of 16 to 18 days.

Symptoms and signs: Often the first sign of the infection is the painful enlargement of one or both salivary glands accompanied by fever. In adults, complications such as orchitis, meningitis and oophoritis may occur.

Oral manifestations: No specific intraoral manifestations are evident in mumps. Since this is a contagious disease, appropriate precautions should be taken while examining the patient.

Investigations: Diagnosis of mumps is made on clinical grounds. Isolation of the virus from saliva or serological tests is confirmatory.

Management: Symptomatic treatment is adequate. MMP vaccine is used for immunisation.

- **Rubella**

Definition: Rubella, also known as German measles is a mild exanthematous infectious disease caused by rubella virus

Cause: An RNA virus of the family *Togaviridae*. Incubation period is about 18 days. Transmission of the virus is by the respiratory route.

Symptoms and signs: During the prodromal period, suboccipital and posterior auricular lymph nodes are enlarged followed by rash. Infection during pregnancy may involve foetal infection which may result in congenital defects.

Oral Manifestations: Occasionally petechial lesions on the soft palate and uvula appear during the course of infection. In the congenital form of the disease, developmental defects of the face and teeth may occur.

Investigations: Presence of lymphadenopathy in the post-auricular and suboccipital region is a useful indicator of the infection. Viral isolation and identification of specific IgM antibodies in the serum are confirmatory.

- **Herpes simplex virus (HSV) infections**

Definition: Herpes simplex virus type 1(HSV-1) and herpes simplex virus type 2 (HSV-2) belong to the *Herpesviridae* family which cause a range of infections in humans.

Symptoms and signs: HSV-1 causes primary herpetic gingivostomatitis, herpetic eczema, keratoconjunctivitis, meningoencephalitis and herpes labialis, whereas HSV-2 is responsible for genital herpes and neonatal herpes. HSV-2 has also been associated with uterocervical cancer.

Oral manifestations:

Primary herpetic gingivostomatitis occurs during childhood between the ages of 2 and 5. It is characterised by fever, malaise, inflammation of the gingivae, painful vesicular eruptions/erosions in clusters on the oral mucosa, oral malodour and bilateral cervical lymphadenopathy. The keratinised oral mucosa is most frequently involved in HSV infections.

Recurrent herpes labialis is the most common form of HSV type 1 infection. It is characterised by vesicular lesions appearing on the mucocutaneous junctions of the lips after a prodromal period of burning sensation. In most cases constitutional symptoms are absent in this form of infection.

Recurrent intraoral herpes is found less frequently. Hard palate, tongue or the gingivae are the preferred intraoral sites in this form of the infection.

Investigations: Clinical findings are suggestive of the infection. Confirmatory investigations include cytological identification of intranuclear inclusion bodies of the

infected cells of the vesicular floor by Giemsa's stain. Serological investigation such as enzyme-linked immunosorbent assay (ELISA) is also useful in the diagnosis. Two to three weeks after the onset of the infection, antiherpes antibody titre is increased fourfold in this test.

Primary herpes simplex virus type 1 infection can also cause keratoconjuctivitis. When recurrent infection occurs it may lead to corneal scarring. Meningoencephalitis caused by HSV type 1 sometimes has a fatal outcome. Herpetic whitlow is a painful vesiculo-ulcerative lesion on a finger or thumb caused by the herpes simplex virus, predominantly seen in health care workers exposed to infected oral secretions, or in children by autoinoculation of the virus due to thumb sucking.

HSV type 2 causes genital herpes which is often seen in male or female genitals. This is characterised by vesicles, fever, malaise and inguinal lymphadenopathy. Infection can be transmitted from active lesions of the mother's genital tract to a neonate. This is called neonatal herpes**.** HSV type 2 has also been implicated in squamous cell carcinoma of the cervix (uterocervical cancer).

- **Chickenpox (varicella)**

Definition: Chickenpox is a highly contagious childhood viral infection characterised by papular, vesicular and crusted skin lesions accompanied by fever.

Cause: Primary infection by varicella-zoster (a herpes virus) causes chickenpox. Infection is usually transmitted by droplet spread. The incubation period is between 12 and 16 days.

Symptoms and signs: Initial symptoms include malaise and fever followed by papular rash on the trunk which soon spreads to face and limbs. Within three to four days, crops of vesicles and crusted lesions appear with pruritus. Symptoms are more severe in adults.

Oral Manifestations: Intraoral vesicular lesions appear in most patients. Sometimes these precede skin lesions. These mimic vesicles of herpes simplex virus lesions, but the occurrence of accompanying skin involvement in chickenpox makes the diagnosis easy.

Investigations: Clinical findings are sufficient to make a diagnosis. If necessary, cytological examination may be carried out to confirm the diagnosis. Cytological changes similar to those of the herpes simplex virus infection are seen on the smear biopsy of the vesicular contents. Serology is also occasionally used to confirm the diagnosis.

Management: No specific treatment is required. Symptomatic treatment with bed rest is adequate.

- **Shingles (zoster)**

Definition: A vesicular infection due to activation of the latent varicella-zoster (V-Z) virus which is usually unilateral and localised to sensory dermatomes.

Cause: Reactivation of the varicella-zoster virus, which is latent (after the primary attack) in dorsal root ganglion cells, may give rise to a new generation of V-Z virions and cause shingles.

Symptoms and signs: Persons in the fifth to seventh decades of life and those with defective cell-mediated immunity are prone to develop shingles. A prodrome of pain and burning along the course of affected nerves followed by the appearance of vesicular

eruptions, also along the course of affected nerves, accompanied by mild fever and regional lymphadenopathy, are hallmarks of the condition. In some patients, postherpectic neuralgia is a consequence of the infection due to fibrosis in the neural ganglion.

Oral manifestations: Intraoral zoster may occur with vesicular eruptions along any division of the trigeminal nerve. Involvement of the first division of the trigeminal nerve may cause herpetic keratitis and Argyll Robertson pupils (AR pupils are bilateral small pupils that constrict when the patient focuses on a nearby object).

Ramsay Hunt syndrome in shingles may occur in some individuals. This is characterised by the appearance of vesicles on the uvula, palate, anterior tongue and auricle. This may be accompanied by transient facial palsy.

Investigations: Clinical findings are suggestive of the diagnosis. Laboratory investigations such as cytological or serological examinations are confirmatory.

Management: Uncomplicated cases do not need any specific treatment. Symptomatic treatment for pain is required. Antiviral medications such as acyclovir, famciclovir or valacyclovir are effective in improving the condition if instituted within the first three days of the appearance of eruptions. In postherpectic neuralgia, antidepressants such as amytriptyline are often used.

- **Human papillomavirus infections**

Definition: Human papillomavirus (HPV) infections generally produce benign wart-like lesions of the skin and mucosa. Oncogenic types have been etiologically associated with cancers of the cervix and anogenital and oropharyngeal anatomical regions.

Cause: Over 120 serotypes of human papillomavirus have been identified. More than 30 types of HPV are typically transmitted through sexual contact and infect the anogenital region. Persistent infection with "high-risk" HPV types (HPV: 16, 18, 31 and 45, for example) —different from the ones that cause skin warts — may progress to precancerous lesions and invasive cancer. It must be realized that most HPV infections are cleared rapidly by the immune system and do not progress to cancers. Cancer occurs in people who have been infected with HPV for a long time, usually over a decade or more (persistent infection).

Symptoms and Signs:

Benign skin lesions: These include common skin warts, plantar warts, subungual or periungual warts, flat warts, genital warts and condyloma accuminatum.

Common skin warts (verruca vulgaris) occur on hands, feet, elbows and knees with a cauliflower-like appearance. These do not carry malignant potential. Plantar warts are found on the soles of the feet and grow inward causing pain. Subungual or periungual warts form under the fingernail (subungual), around the fingernail or on the cuticle (periungual). Flat warts are most commonly found on the arms, face or forehead.

Genital warts include venereal warts and condyloma accuminata. HPV types 6 and 11 account for about 90% of all cases. These do not carry any malignant potential. Condyloma accuminatum is generally related to sexual activity. Lesions are papillomatous growths found around anal and genital openings where moisture is pronounced. Their occurrence in the mouth is on the increase due to the popularity of orogenital sex. They present as papillomatous growths.

Potentially malignant and malignant lesions caused by HPV include cervical intraepithelial neoplasia (CIN), invasive cervical carcinoma, vaginal intraepithelial neoplasia,

vaginal carcinoma, vulvar intraepithelial neoplasia, (Bowenoid papulosis and erythroplasia of Queyrat), vulvar carcinoma, penile carcinoma and anal and perianal carcinoma.

Oral manifestations: HPV can cause a range of benign oral papillomatous lesions. These include squamous papillomas, oral verruca vulgaris, oral condyloma accuminatum and focal epithelial hyperplasia.

Oral squamous papilloma is usually a single, wart-like, pedunculated lesion with finger-like projections. The tongue, lips and palatal mucosa are the preferred sites of their occurrence squamous papilloma is caused by HPV 6 and 11. It carries no malignant potential.

Oral condyloma accuminatum (venereal wart) are characterised by multiple, broad, wart-like, sessile pink or white lesions found on the tongue, lips, soft palate or gingiva. Predominantly these are found in persons practicing orogenital sex. HPV 6, 11 and 16 are found to be causative agents of oral condyloma acuminatum.

Focal epithelial hyperplasia is uncommon and predominantly seen in children. Lesions are multiple, flat, round and sessile. These lesions are of normal mucosal colour. These heal spontaneously. HPV 13 and 32 have been identified as causative agents. Spread of the infection occurs through sharing of food and personal objects.

Oral verruca vulgaris lesions are sessile, papillomatous, painless and hyperkeratotic. Lesions are usually found on the labial mucosa of the lower lip in children. Autoinoculation of the virus (HPV 2 and 4) from lesions on the fingers is often the reason for oral lesions.

In a small percentage of patients, HPV has also been found to be associated with oral potentially malignant disorders and malignant lesions (**OPMDs**). These include oral leukoplakia, proliferative verrucous leukoplakia, oral squamous cell carcinoma and oral verrucous carcinoma.

Investigations: Immunological localisation of HPV antigens and DNA hybridisation methods, and amplification of DNA with polymerase chain reaction (PCR) are useful diagnostic tools.

Management: Benign lesions generally regress spontaneously. Surgical excision or chemical destruction of the lesions may be carried out where clinicians and patients feel that treatment is necessary. Potentially malignant and malignant lesions need appropriate surgical, chemotherapeutic or radiation therapy.

- **Coxsackie virus infections**

Hand, Foot and Mouth Disease and herpangina.

Definition/Description: Coxsackie groups (A and B) of viruses cause a variety of diseases. The Coxsackie group of viruses are RNA viruses which belong to the family of *Picornoviridae*. Two important infections of Coxsackie viruses include hand, foot and mouth disease and herpangina. These conditions are caused by Coxsackie A group of viruses. Infection is usually by droplet spread, ingestion or direct contact.

1. **Hand, foot and mouth disease (HFMD):** HFMD usually affects infants and children, and is quite common. It is moderately contagious and is spread through direct contact with the mucus, saliva, or faeces of an infected person. It typically occurs in small epidemics in nursery schools or kindergartens, usually during the summer and autumn months. The usual incubation period is 3–7 days.

Symptoms and signs: Fever, malaise sore throat are the initial symptoms. Soon painful vesicles appear on the palms and soles, especially near the base of the fingers and toes. Intraoral sites include cheek mucosa and tongue.

2. **Herpangina: Oral lesions:** In herpangina, small vesicles appear which soon rupture to form crops of ulcers on the anterior tonsillar pillars, soft palate uvula, tonsils and pharyngeal walls.
   Investigations: Clinical findings are suggestive. Confirmatory tests include virus isolation in early stages of the disease by serology.
   Management: Symptomatic treatment for pain and fever. Both diseases are self - limiting.

Fungal Infections

- **Candidiasis (Candidosis)**

Definition: Candidiasis, also known as candidosis, is the term used to denote infections caused by Candida species. The spectrum of infections includes those of the skin, nails, mucous membranes and internal organs.

Causes: Over 100 Candida species exist, but not all are pathogenic. Important pathogenic species are *Candida albicans, Candida tropicalis, Candida glabrata, Candida parapsillosis, Candida guilliermondi, Candida cruisei, Candida dubliniensis and Candida stellatoidea.*

Predisposing factors that render the host vulnerable to candidal infections include: infancy or old age, pregnancy, prolonged use of drugs such as wide-spectrum antibiotics, corticosteroids, cytotoxic drugs and immunosuppressive agents. Other factors include radiation, indwelling intravenous tubes, bladder catheters, prosthetic heart valves, poorly maintained dentures, self-administered intravenous recreational drugs, primary and acquired immune deficiency states (HIV/AIDS), acute and chronic leukaemia, lymphoma, diabetes, hypothyroidism, Addison's disease and iron deficiency.

Symptoms and signs: Candidiasis caused by *Candida albicans* is very common. Some entities are discussed below.

- **Candidal onychia and paronychia:** This refers to candidal infection of the nails and soft tissues around the nails. Involved nails show green black discolouration and transverse ridges. Soft tissue surrounding the nail is inflamed and tender.
- **Interdigital candidiasis:** This is characterised by pruritic, inflamed and scaling lesions between the fingers and toes.
- **Intertriginous candidiasis:** This refers to candidal infection of the skin in the inguinal folds, submammery area, axilla, umbilicus and scrotum. Tender pruritic vesicular lesions occur in these areas.
- **Vaginal infection** with candida is influenced by local factors such as glycogen concentrations, local pH, and epithelial cellular conditions.
- **Candidiasis of the gastrointestinal mucosa**: Infants, diabetics, and those on long term antibiotic or corticosteroid therapy are vulnerable to develop gastrointestinal candidiasis.

Candidiasis of the genitourinary mucosa: Vaginal infection with candida can occur in pregnancy or in those with diabetes and long term antibiotic use. Vaginal pruritus and discharge are common in these women. In men, candidal balanitis with erythaematous plaques on the glans and around the prepuse is common.

Oral Manifestations: Oral Candidiasis: One of the most common manifestations of human candida infection is oral candidiasis. Symptomatic oral manifestations of oral candidiasis generally include white and red lesions. White lesions include acute pseudomembranous candidiasis (thrush), candidal leukoplakia (chronic hyperplastic candidiasis) and mucocutaneous candidiasis. Red lesions of candidiasis include erythematous (or atrophic) candidiasis comprising denture-related stomatitis, median rhomboid glossitis and antibiotic- or steroid-related candidiasis.

Oral symptoms and signs: Pseudomembranous candidiasis is characterised by white curd–like mucosal plaques. These can be easily rubbed off with gauze to leave a raw, erythematous and bleeding base.

In chronic hyperplastic candidiasis, the patch is a persistent lesion with white or speckled red/white appearance. This lesion cannot be rubbed off due to the keratotic nature of its development. Chronic mucocutaneous candidiasis is often seen in patients with endocrinopathies and immune disorders. In these patients, oral white patches are irremovable. Skin, nails and other mucosal regions are also involved in this condition.

Erythematous candidiasis is seen in long term antibiotic or steroid users. Wide spread erythema and soreness of the oral mucosa are common in these patients. Median rhomboid glossitis or glossal central papillary atrophy shows a rhomboid, central depapillated area anterior to the circumvallate papillae on the tongue. Occasionally lesions may be lobulated. Denture related stomatitis (also known as denture sore mouth or chronic atrophic candidiasis) presents mild erythema of the mucosa beneath the dental appliance. In most cases this occurs in upper, full denture wearers. Papillary hyperplasia on the vault of the palate may be seen in this condition and which requires surgical intervention before the denture is replaced. Angular cheilitis may be associated with this condition.

Investigations: Clinical findings are suggestive of candidal involvement for cutaneous and mucosal lesions. Confirmatory tests include microscopic identification of the organisms in large numbers in smears or tissues. Culture studies or serology and skin tests are also useful.

Management: Antifungal treatment (Nystatin or Amphotericin B topical application) and identification and elimination of predisposing factors are essential therapeutic measures in the treatment of oral candidiasis. In addition, systemic antifungal treatment may be required for candidiasis of the gastrointestinal tract and other mucosal sites.

- **Aspergillosis**

Definition: Aspergillosis is an infection by the species of the genus *Aspergillus.*

Cause: Pathogenic species such as *Aspergillus fumigatus, Aspergillus niger, and Aspergillus flavus* are causative organisms.

Symptoms and signs: The respiratory tract, external auditory canal, nasopharynx, cornea, gastrointestinal tract, and occasionally the skin, may be the primary sites of infection. Oral infection is generally seen in the disseminated form of the disease. Paranasal sinuses are

frequently involved in aspergillosis. Those with impaired immune responses are prone to develop aspergillosis.

Oral manifestations: Though rare, oral involvement of the soft palate can occur as an ulcerative lesion surrounded by a ring of blackened necrotic tissue.

Investigations: Identification of the organisms and culture studies are confirmatory tests. Specimens are obtained from sputum and biopsy material.

Management: Amphotericin B is the treatment of choice. Intravenous antifungal treatment and surgical debridement of the lesions may be necessary in those with an immune compromised status.

- **Histoplasmosis**

Definition: Histoplasmosis is a fungal disease caused by *Histoplasma capsulatum and Histoplasma duboisii.*

Cause: *Histoplasma capsulatum,* a dimorphous fungus, causes histoplasmosis. This exists in a hyphal form in the soil, and a yeast form in the tissues.

Symptoms and signs: Disease presents in three forms: Acute primary histoplasmosis, progressive disseminated histoplasmosis and chronic cavitory histoplasmosis. In the acute primary form of the disease, pulmonary involvement with cough and pleuritic pain may be present. Chest x-rays may show infiltration and calcifications. In the progressive disseminated form, pulmonary, bone, kidney and gastrointestinal involvement may occur. In the cavitory form the condition is characterised by fever, dyspnoea, cough and weight loss mimicking tuberculosis.

Oral Manifestations: In the progressive disseminated form of the disease, oral manifestations are common. Lesions may be papular, ulcerative, nodular or verrucous in appearance. Any mucosal area may be involved. Lesions may extend on to the pharynx. Sore throat is a common complaint.

Investigations: Identification of the fungus from infected material, and culture studies are confirmatory.

Management: Systemic Amphotericin B and supportive treatment are required for the management of histoplasmosis.

- **Rhinosporidiosis**

Definition: Rhinosporidiosis is a chronic fungal granulomatous disease of the nasal mucosa

Cause: The causative agent is a fungus called *Rhinosporidium seeberi.*

Symptoms and signs: Nasal irritation and mucoid discharges are initial symptoms. This phase is followed by sessile or papillomatous lesions of the nasal mucosa. Conjunctiva, larynx, mouth and bronchus are other sites of occurrence.

Oral manifestations: Pink granulomatous growths may be seen on the soft palate and uvula. Often, these are extensions of pharyngeal polyps caused by the fungus.

Investigations: Smear biopsy and culture studies are confirmatory tests.

Management: Application of cautery or surgical removal of the growths is recommended.

Diseases Caused by Protozoa

- **Malaria**

Definition: Malaria is a disease caused by infection with protozoa of the genus *Plasmodium,* and transmitted by the bite of female *Anopheles* mosquitoes.

Cause: There are four species of plasmodia responsible for malaria: *Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale* and *Plasmodium malariae.*

Symptoms and signs: Malaria is common in Africa, Asia, South America and the Caribbean. Symptoms occur 10-16 days after infection by mosquito bites. Chills, fever, headache and myalgia are common symptoms of the disease. The pattern of paroxysmal illness interspersed with periods of relative well-being is the hallmark of malaria. Anaemia, thrombocytopenia, and splenomegaly are other features present in malaria.

P. falciparum malaria may present fatal complications such as cerebral malaria.

Oral manifestations: There are no specific oral manifestations of malaria. The oral cavity may become vulnerable to infections (herpes labialis, for example) and a tendency to bleed may occur due to thrombocytopenia. In Africa, where malaria is endemic, Epstein-Barr virus (EBV) infection has been found to be associated with Burkitt's lymphomas of the jaw bones in children.

Investigations: Blood smear examination with Giemsa stain during the febrile stage of the illness for identification of plasmodia is confirmatory.

Management: The antimalarial agent Chloroquine is the treatment of choice. Supportive therapy for headaches, myalgia and fever are required during the illness. Immunity to malaria develops over an extended period of repeated infections.

- **Amoebiasis**

Definition: Amoebiasis was defined by the World Health Organization in 1969 as "the condition of harbouring *Entamoeba hystolytica* with or without clinical manifestations". [2]

Cause: There are a number of amoebae resident in the intestine. *Entamoeba hystolytica* is the only species known to be pathogenic to humans.

Symptoms and signs: The majority of individuals with amoebic infections are asymptomatic. When symptomatic, diarrhoea and abdominal pain are common. The faeces are musty or watery, foul smelling, and may contain blood. In chronic cases, low grade fever, anorexia and anaemia may be present. Liver abscess and amoebic colitis occur as complications of amoebiasis. Rarely, amoebic abscesses may develop in the brain.

Oral manifestations: There are no specific oral manifestations of amoebiasis. Oral symptoms secondary to anaemia may occur.

Investigations: Identification of the causative agents in freshly passed stools. Sigmoidoscopy is a valuable diagnostic tool. Serology is also useful.

Management: Metronidazole (Flagyl) is the drug of choice. Anaemia should be treated. Improved personal hygiene is important in preventing the spread of the disease.

Helminthic Diseases

Definition: Helminths are multicellular parasites commonly referred to as worms. Helminthic diseases are common in the tropics and in communities with low socioeconomic status.

Cause: There are three types of helminths which cause disease: cestodes (tapeworms)**,** trematodes (flukes) and nematodes (round worms).

Symptoms and signs: Clinical features of helminthic diseases are wide ranging. The majority of infections are primarily related to the gastrointestinal system. Occasionally neuropsychiatric, cardiovascular, respiratory and cutaneous disorders may occur.

Oral manifestations: There are no specific oral manifestations associated with helminthic disorders. Oral findings secondary to anaemia caused by helminths are common in the tropics.

Investigations: Recovery of larvae or eggs of helminths is diagnostic.

Management: Management involves specific chemotherapeutic agents. Surgery may be required in some instances. Nutritional supplements and preventive strategies are also required.

Chapter 6

Diseases of the Gastrointestinal System

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Abstract

This chapter deals with common diseases and disorders that affect the gastrointestinal system. The chapter begins with symptom based, common gastrointestinal complaints and briefly describes the system specific clinical examination methods and investigations employed in the diagnosis of disorders of the gastrointestinal system. Since the oral cavity is a part of the gastrointestinal system, common mucosal diseases such as stomatitis, oral ulcers and gingival enlargements are discussed. Disorders of the oesophagus and intestines discussed in this chapter include dyspepsia, dysphagia, gastro-oesophageal reflux disease, gastritis, hiatus hernia, gastroenteritis, appendicitis, acute abdomen, peptic ulcer disease, gastric and duodenal ulcer, celiac disease, inflammatory bowel disease (IBD), Crohn's disease and ulcerative colitis, irritable bowel syndrome (IBS), haemorrhoids, dysentery (amoebic and bacillary dysentery), malabsorption and diarrhoeal disease. Each disease entity starts with a definition or description followed by its causes, symptoms, signs, the investigations employed and the principles of management. Oral and dental aspects of the gastrointestinal diseases are also described in this chapter.

Introduction

The primary structures of the gastrointestinal system include the mouth, pharynx, oesophagus, stomach, small intestine (duodenum, jejunum and ileum), large intestines (caecum, ascending colon, transverse colon, descending colon and sigmoid colon) and rectum. Dental students and practitioners need to have an adequate knowledge of the diseases involving these structures because of their possible association with oral disease and dental management issues.

Common Gastrointestinal Symptoms

Some of the common gastrointestinal symptoms include: abdominal pain, change in bowel patterns, weight change, heart burn, nausea and vomiting, difficulty in eating and swallowing, jaundice, chest pain, diarrhoea, abdominal swelling, constipation, loss of appetite, rectal bleeding, bruising tendencies, intestinal bloating, and weakness. It should be noted that some of the above mentioned symptoms can also occur in diseases of other systems.

Some symptoms that are common to gastrointestinal problems are briefly discussed below:

Abdominal pain: Abdominal pain can be classified as visceral, parietal or referred.

- Visceral pain results from intestinal distension or stretching of the solid abdominal organs. Pain is often described as burning, cramping, diffuse and poorly localised.
- Parietal pain results from inflammation of the parietal peritoneum. Pain is severe, localised and aggravated by movement.
- Referred pain is felt at a site away from the site of its origin.

Location of pain can serve as an important indicator of the abdominal disease. Pain in the umbilical area, for example, may be due to an abdominal aortic aneurysm or appendicitis.

Pain in the upper epigastric region left of midline may indicate a gastric ulcer or angina and myocardial infarction.

Pain radiating to the back, neck or jaw may indicate gastro-oesophageal reflux disease (GORD), angina and myocardial infarction.

Substernal chest pain with difficulty breathing after a meal may be due to hiatus hernia.

Change in bowel patterns: Changes in bowel movements occur in a variety of GI disorders. These include malabsorption syndromes, irritable bowel syndrome, colorectal cancer, infections of the GI tract, food intolerance and reactions to medications.

The colour of the stool is also an indicator of GI disease. Upper GI bleeding causes black, tarry stools, lower GI bleeding causes red, bloody stools and clay coloured stools are indicative of increased bile seen in obstructive jaundice.

Constipation: This common condition displays decreased frequency of the evacuation of the bowels along with abnormally firm stools. Constipation is a symptom and not a disease. Ignoring the normal habit of bowel movement, decreased fibre residue and dehydration are common causes of constipation.

Diarrhoea: Diarrhoea is defined by the World Health Organization as having three or more loose or liquid stools per day, or as having more stools than is normal for that person. Severe diarrhoea is a common cause of death in the developing countries and the second most common cause of infant death worldwide. Diarrhoea in children under five years of age is commonly due to viral gastroenteritis with rotavirus. In travellers, bacterial infections are the major causes of diarrhoea. Various toxins and drugs can also cause acute diarrhoea. Common causes of chronic diarrhoea include ulcerative colitis, Crohn's disease, celiac disease, irritable bowel syndrome and bile acid malabsorption

Indigestion/gassiness/burning: Collectively, this is known as dyspepsia. Indigestion is often described by the patient as ‘heart burn’. This is due to acid from the stomach flowing

into the lower oesophagus, causing a burning sensation. This is a major feature of gastroesophageal reflux disease (GORD). Heart burn is also a common complaint in gastric ulcer, duodenal ulcer, and gall bladder disease. Indigestion associated with belching and flatulence suggests cholecystitis.

Nausea and vomiting: Nausea is the unpleasant feeling of needing to vomit, whereas vomiting is the forceful expulsion of gastric contents produced by involuntary contractions of the abdominal musculature. Gastric regurgitation, on the other hand, is the spitting up of gastric contents without associated nausea and abdominal muscular contractions. Common gastrointestinal diseases which cause vomiting include intestinal obstruction, peptic ulcer disease, viral and bacterial infections of the GI tract, and appendicitis.

"Lump in the throat": This is called also globus sensation. Patients describe this as the sensation of a lump in the throat unrelated to swallowing. In this condition, there is no mass or lump in the throat. This is common in patients with gastroesophageal reflux disease (GORD).

Hiccups: Hiccups are repeated involuntary spasms of the diaphragm followed by the sudden closure of the glottis producing the characteristic sound. Causes include gastric distension, alcohol, swallowing irritating or hot substances, GORD, and oesophageal disorders.

Examination of the Gastrointestinal System and Abdominal Contents

Although dental practitioners are generally not required to perform a physical examination of the abdomen and its contents, they should be familiar with the indications and steps involved in the examination of abdominal contents. Prior to the abdominal examination, it is essential to perform a general examination of the patient. General examination includes inspection of the face, eyes, mouth, hands, nails, neck, axilla thorax and abdomen.

Face: Look for signs of pallor (anaemia), jaundice and cyanosis, and dilatation of the superficial facial capillaries resulting in facial erythema.

Eyes: Look for signs of xanthelasma. These are yellowish skin papules around the eyes indicative of hyperlipidaemia. Among other causes, these may be due to prolonged cholestasis as a consequence of primary biliary cirrhosis. Sclera of the eyes also show jaundice in liver disease. In iron deficiency and pernicious anaemia, the conjunctivae are pale.

Fingers, nails and palms: Look for finger clubbing. Among gastrointestinal causes, this occurs in liver cirrhosis, Crohn's disease and celiac disease. Nails are spoon-shaped (koilonychia) in iron deficiency anaemia. White nails (leukonychia) are also seen in chronic liver disease. Contracture of the ring and little fingers (Dupuytren's contracture), red palms (palmar erythema) and flapping tremors are often seen in chronic liver cirrhosis and liver failure.

Thorax and axilla: Look for spider nevi. These are (spider-like) radiating blood vessels (also called spider angioma) on the skin of the anterior chest wall. These blanch on digital pressure. If there are more than five spider nevi seen, it is likely that the individual has a chronic liver disease. These are not to be confused with purpura (which does not blanch on pressure). Look for the enlargement of male breasts (gynaecomastia) and loss

of axillary body hair. Chronic liver disease in men may result in feminisation of the male due to increased circulating oestrogens and decreased testosterone.

Mouth: Look for oral ulcers. These may occur in gastrointestinal disorders such as Crohn's disease, celiac disease and ulcerative colitis. In Peutz-Jegher syndrome, intestinal polyposis and perioral pigmentation are common. In advanced anaemia, the tongue shows a smooth surface (bald tongue), with or without inflammation of the angles of the mouth. Patients with severe liver disease may have foul-smelling breath (fetor hepaticus).

Examination of the Abdomen

For descriptive purposes the abdomen can be divided into nine regions:
From right to left:

1. Right hypochondrium
2. Epigastrium
3. Left hypochondrium
4. Right flank (right lumbar region)
5. Umbilical region
6. Left flank (left lumbar region)
7. Right iliac fossa
8. Hypogastrium (suprapubic region)
9. Left iliac fossa

Examination methods include inspection, palpation, percussion and auscultation.
The patient is supine on the examination table, and the examiner stands to patient's right.

Inspection

- Look for abdominal symmetry, visible pulsations (from abdominal aorta) and dilated veins, abdominal movement with respiration and from gastric peristalsis, abdominal skin lesions such as striae or stretch marks (following stretching by pregnancy, obesity or Cushing's syndrome), surgical scars, swellings, nodules, spider nevi and protruding hernia. These are localised bulges in areas of weak abdominal wall and will increase in size on coughing or sitting up. Uniform abdominal swelling is a sign of ascites. Causes of a swollen abdomen may be due to fluid, flatus, fat, foetus, and faeces (the five F's).

Auscultation

- The diaphragm of the stethoscope is used to auscultate the abdomen. Auscultation is performed before percussion or palpation in order to avoid disturbances in the intestines by the vigorous touching of the abdomen during these procedures.

Auscultation is performed to listen for bowel sounds and vascular bruits. Bruits are "swishing" sounds heard over the major arteries during systole or, less commonly, systole and diastole. The area over the aorta, both renal arteries, and the iliac arteries should be examined carefully for bruits. Normal peristaltic movements can be heard as gurgling sounds.

Percussion

- Place one hand on the area to be examined, and with the index finger or the index and middle fingers of the other hand, strike the hand resting on the patient's abdomen.
  1. Assess whether the abdominal area is solid (dull), hollow (resonant), air filled (tympanic or drum-like) or fluid filled (dull). Percussion of the abdomen provides information on abdominal organ size delineation. Percussion is carried out from resonant to dull.
  2. For deeper structures, (the upper border of the liver, for example) deep percussion is necessary.
  3. When eliciting 'shifting dullness', the patient is asked to lie on his/her side.
  4. Percuss towards the flank until a dull note is obtained.
  5. Without moving the hand, roll the patient onto the opposite side and wait for the fluid to redistribute.
  6. Percuss again, and if the note is resonant, confirm the finding by percussing back towards the midline, where a dull note will be present.

Palpation

Two types of palpations are used: light (superficial) palpation and deep palpation.

- Light palpation in each of the nine regions, beginning away from any painful area.
- Palpate for rigidity, tenderness, and swellings.

Palpation of the groin for inguinal lymph nodes and inguinal or femoral hernias is also carried out during the abdominal examination.

Light palpation. Light palpation is the first step. Use the pulp of fingers (not finger tips). Move/roll your hand over the abdomen lightly, keeping in contact with the abdomen. If the patient complains of pain, palpation should be started in an area away from the site of that pain. Ask the patient to tell you if it is tender when you press. Look at patient's face for evidence of pain. During this process, tender areas for guarding or rebound tenderness are assessed. Instantaneous contraction of abdominal muscles over a painful area is guarding. This is due to an inflamed organ or peritoneum. Rebound tenderness occurs when pain is experienced after quickly lifting your hand off the affected area of the abdomen. This is also a sign of inflammation.

Deep palpation: Palmar aspects of the fingers are used for deep palpation. Deep palpation is organ specific. It is used for internal organs such as the liver, spleen and kidneys, and for abdominal masses. During deep palpation, the patient is asked to breathe deeply through the mouth. If masses are palpated, the clinician should determine their location, size, consistency, shape, mobility and the presence or absence of tenderness and pulsations.

Palpation of the Liver

Steps:

- Patient is positioned in supine position.
- Ask the patient to take deep breaths in and out.
- Start palpating in the right iliac fossa and move towards the right costal margin. Use second and third right fingers to feel for the edge of the liver.
- With each expiration, move your palpating fingers upwards.

In normal individuals, the liver is not usually palpable.

Palpation of the Spleen

Steps:

1. The front of the examining hand is laid flat on patient's abdomen with the fingers at right angles to the left costal margin.
2. Press inwards and upwards.
3. After each inspiration, move palpating hand upwards until the finger tips are under the costal margin.
4. Repeat the process along the rib margin until the spleen is felt (which can vary in its position). If enlarged, the spleen projects towards the umbilicus.

The spleen is located under the left costal margin beneath the 19^{th} rib, anterior to the axillary line. Normally, the spleen is not palpable.

Palpation of the Kidneys

Steps:

1. Bimanual palpation is used to palpate the kidneys.
2. Place the posterior hand in the renal angle and with the fingers pressed forwards. (The renal angle is an area located on either side of the human back between the lateral borders of the erector spinae muscles and the inferior borders of the 12^{th} rib, so called because the kidney can be felt at this location).

3. Position the anterior hand along the horizontal plane with the finger tips over the rectus muscle.
4. Use deep palpation and bring your two hands as close together as possible while the patient breathes deeply.
5. The kidneys may be caught between the two hands.

Usually, kidneys are not palpable in healthy individuals. Palpation of the kidneys is contraindicated in renal transplant patients.

Palpation of the abdominal aorta: In a thin individual, the normal aorta may be felt as a pulsatile structure in the midline of the abdomen.

Investigations in Gastroenterology

A detailed health history and physical examination of the abdomen precedes investigations in assessing the diseases of the gastrointestinal system. Investigations employed in gastroenterology include the following:

Endoscopy:

- This is performed in order to visualissee of parts of the GI tract and also to take biopsies of the involved tissues.

Gastroscopy:

- The oesophagus, stomach and proximal duodenum can be investigated with a gastroscope.

Colonoscopy:

- The large bowel and terminal ileum can be investigated using the colonoscope.

Sigmoidoscopy:

- Diseases of the rectum can be investigated with sigmoidoscope.

Laparoscopy:

- This is a method of directly inspecting the abdominal organs using a fibreoptic system via one or more small incisions.

Radiology:

- The whole of the GI tract can be investigated radiologically using a contrast medium such as barium swallow, barium meal or enema.

Crosby capsule:

- A device used for obtaining biopsies of small bowel mucosa. This is used for jejunal biopsies in celiac disease.

Faecal fat collection:

- Faeces collected for 3-5 days to quantify the fat content (in the diagnosis of malabsorption).

Faecal occult blood:

- Simple bed-side methods are used to detect haemoglobin in the blood in faeces.

Breath tests:

- A urea breath test is used for the detection or absence of *Helicobacter pylori.* A lactose hydrogen breath test is used for the detection of disaccharide deficiency.

Pancreatic function tests:

- Tests are available to assay pancreatic exocrine function.

Intestine motility tests:

- These involve the use of radio-opaque markers along the GI tract.

Oesophageal manometry:

- Oesophageal manometry is a test used for assessing motor function of the upper oesophageal sphincter, oesophageal body, and lower oesophageal sphincter. This test measures intra-oesophageal pressure during swallowing.

pH monitoring:

- pH monitoring is carried out using portable pH probes positioned above the gastro-oesophageal junction and connected to a 24 hour recording system (used in gastro-oesophageal reflux disease).

Gastrointestinal Diseases of Dental Interest

Common oral mucosal diseases

1) Stomatitis

Definition/description: Stomatitis refers to inflammation of the oral mucosa. Sometimes the term mucositis is erroneously used to indicate inflammation of the oral mucosa. The term mucositis should be reserved for inflammation of the mucosal surfaces anywhere in the body. Usually mucositis is a result of radiotherapy or chemotherapy for cancer, and due to infections involving several mucosal surfaces.

Stomatitis may be mild, severe, localised, diffuse, or painful. Some conditions with stomatitis are recurrent.

Causes: Local causes of stomatitis and oral ulcers include:

- Trauma from physical agents (ill-fitting dental appliances, for example) and cheek biting.
- Thermal agents (pizza burn, for example).
- Chemical burns (e.g., aspirin burn), and tobacco use (smokers stomatitis).
- Allergic responses to food items (fish, for example).
- Ingredients of tooth paste, lip stick, and chewing gum (e.g., cinnamon used as flavouring agent).

- Conditions with obscure aetiology which cause stomatitis include lichen planus, burning mouth syndrome and recurrent aphthous stomatitis.
- Local and systemic infections (bacterial, viral and fungal).

Stomatitis predominantly attributable to local infections include acute necrotising ulcerative gingivitis (caused by fuso-spirochetal bacteria), **streptococcal stomatitis**, **staphylococcal mucositis**, primary and secondary herpes simplex virus infections (**herpetic gingivostomatitis),** and Coxsackie virus infection (**herpangina**). Stomatitis caused by *Candida albicans* is particularly common in immunocompromised patients. In denture wearers, candidal infection can cause **denture stomatitis** and in those who are on long term antibiotics, the fungus causes **antibiotic stomatitis**.

Stomatitis predominantly due to systemic causes include Behcet's disease, celiac disease, cyclic neutropenia, erythema multiforme, inflammatory bowel disease (IBD), iron deficiency (anaemia), leukaemia, mucous membrane pemphigoid, pemphigus vulgaris, Stevens-Johnson syndrome, thrombocytopenic purpura, vitamin B deficiency (pellagra), vitamin C deficiency (scurvy), gonorrhoea **(gonococcal stomatitis),** primary and secondary syphilis, muco-cutaneous candidiasis blastomycosis, cryptococcosis, primary and secondary herpes simplex virus infections, primary and secondary varicella zoster virus infections (chicken pox and shingles), Epstein-Barr virus infections (infectious mononucleosis), and Coxsackie virus infection (hand foot and mouth disease). Radiation damage (**radiation mucositis**) and chemotherapy also cause stomatitis. Hormonal changes occurring in puberty and pregnancy are also known to cause stomatitis which is especially confined to the gingival tissues. Clinical manifestations may differ based on the causes involved. Some present inflammatory changes with redness whereas others may present as erosions or ulcers.

Symptoms and signs: Any of the cardinal signs of inflammation of the oral mucosa may be present in patients with stomatitis. Signs and symptoms depend on the causes, including the underlying systemic disorders. Patients may be acutely ill, as in Stevens-Johnson syndrome, or may not exhibit any systemic manifestations if the cause is of local origin. Discussion on symptoms and signs of all conditions listed above is beyond the scope of this chapter.

Diagnosis and investigations: A thorough history and clinical examination are of paramount importance in the diagnosis of stomatitis. A systemic search for the underlying cause should be carried out if a local cause cannot be found. Skin examination is important for muco-cutaneous lesions such as those seen in lichen planus, secondary syphilis, or pemphigus.

Investigations include microscopic examination of the samples obtained from smears, mucosal scrapings or oral rinse, and from biopsy specimens. Conditions of autoimmune background need to be investigated using immunofluorescence or serological methods. When systemic infections are suspected, FBC, differential count, serology and cultures are recommended. In these circumstances, consultation with a physician is essential. Rarely, molecular studies involving polymerase chain reaction (PCR) methods may become necessary to identify the specific pathogen (a virus, for example) causing the disease.

Management: Treatment of stomatitis involves identification of the cause, and eliminating it. If the cause is local (physical, thermal or chemical, for example), elimination of the cause and symptomatic treatment and follow up are adequate. Topical local anaesthetic agents may be necessary for pain. If stomatitis is caused by local infections, these need to be

managed with topical and/or systemic antibiotics for bacterial infections. Topical steroid treatment may be necessary for oral lesions of lichen planus and pemphigus vulagaris. The majority of viral infections causing stomatitis are self–limiting and do not require antiviral medications unless the symptoms are severe. Candidal infections can be treated with antifungal agents. Oral and denture hygiene should be emphasised to these patients. In consultation with the physician, underlying predisposing factors need to be identified, investigated and treated. When systemic causes are suspected (autoimmune disorders or leukaemia, for example) treatment for stomatitis should be carried out in consultation with the physician. In all cases of stomatitis, maintenance of oral hygiene and symptomatic treatment for oral pain and discomfort are most important.

- **Oral ulcers and ulcer-like lesions**

Oral ulcers are common in dental practice. Causes of the majority of oral ulcers can be traced. Some ulcers are preceded by blisters, with or without constitutional symptoms. Oral ulcers can be recurrent or persistent. After elimination of the cause, the majority of oral ulcers heal within a week. A small percentage of oral ulcers may not heal, even after the cause has been removed - these may carry malignant potential at the time of presentation.

Definition/description: The term, ulcer, refers to lesions with a full thickness loss of surface epithelium, thus exposing the underlying connective tissue. Often, ulcer-like lesions present diagnostic confusion. These include erosive and atrophic lesions. Erosive lesions refer to superficial breaches of epithelium without exposing the underlying lamina propria, and atrophic or desquamative lesions refer to a thinning of the epithelium, assuming a red appearance.

Causes: There are several local and systemic causes and predisposing factors. These include trauma (traumatic ulcers, for example), infection (e.g., bacterial, viral and fungal infections), malignancy (e.g., carcinoma, melanoma, adenocarcinomas and Kaposi's sarcomas), autoimmune disorders (pemphigus and Wagener's granulomatosis, for example) and unknown causes. The salient features of some oral ulcers are discussed in this chapter. For detailed descriptions, the reader is referred to oral medicine or pathology texts.

- **Traumatic ulcer**

Traumatic ulcers are common. The cause is usually known to the patient. These are located adjacent to the injurious agent that has caused the ulcer. Usually, traumatic ulcer is single and variable in size, depending upon the cause. The shape of the traumatic ulcer is round or crescentic/crater-like and the base is usually shallow. Margins are red, and may be raised in chronic ulcers. A yellow (acute)/granulating floor is seen in the healing phase of the traumatic ulcer. Acute ulcers are generally painful and are of short duration. Less painful ulcers with long duration are exemplars of chronic ulcers. Cervical lymph nodes in traumatic oral ulcers are usually not enlarged unless the ulcer is secondarily infected.

Causes include ill-fitting dentures, sharp cusps of teeth or appliances rubbing against the mucosa, and thermal, chemical or radiation burns.

Diagnostic approaches to traumatic ulcers include the following:

1. Determine if there is a history of trauma. A cause of trauma must be identified. The cause must fit the size, site and shape of the ulcer.
2. Remove the suspected cause**.** On removal of the cause, the ulcer must show signs of healing within 7-10 days
3. If the lesion has not resolved within 2-3 weeks (after the removal of the cause), refer the patient for biopsy and microscopic examination.

- **Malignant ulcer**

Malignant ulcers of the oral mucosa are examples of squamous cell carcinomas. Rarely malignant melanomas, Kaposi's sarcomas and salivary gland tumours may show ulcerative changes. Squamous cell carcinoma appearing as an ulcer is discussed here. For other forms of oral cancer, the reader is referred to the standard textbooks on oral pathology or medicine.

Causes: In a majority of cases, causes of squamous cell carcinoma include long term use of tobacco and alcohol. Ultraviolet radiation from sunlight is associated with lip cancer. Human papilloma virus (HPV) has been shown to be causally associated with a subgroup of oral cancers amongst those individuals who generally do not have a history of alcohol abuse and smoking. The majority of these individuals have a history of oro-genital sex.

Clinical features: The malignant ulcer is usually single. Favoured sites include the floor of the mouth, tongue, buccal mucosa, lip and alveolar ridge. A malignant ulcer is of variable size, shape and depth. It is painless in its early stages. Malignant ulcers show raised, rolled and everted margins. The floor of the malignant ulcer is of variable depth and the base is indurated. Malignant ulcers fail to heal even after the cause (if known) has been eliminated. Cervical lymph nodes are not tender, but are enlarged and fixed to the underlying structures (depending on the stage of the malignant ulcer).

Diagnostic approaches to oral malignant ulcers include the following:

- Take a thorough history (medical/family/social, etc).
- Look for the possible causative and /or predisposing factors.
- Examine the ulcer noting its size, shape, margins, etc. Rule out other possible causes of long standing oral ulceration (TB ulcer, for example).
- Offer a provisional diagnosis.
- Refer the patient to a specialist for a biopsy/microscopic evaluation.

Rule of thumb: Any oral ulcer that refuses to heal, even after the elimination of the cause, should be biopsied and histologically examined.

- **Recurrent aphthous ulcer (RAU)**

Recurrent aphthous ulcer (RAU) is also known as recurrent aphthous stomatitis. RAUs are common causes of oral ulceration. Nearly 20% of the healthy population are affected by RAU.

Causes of RAU are uncertain. Immunologic defects have been suspected. Precipitating factors for RAU include trauma, stress, menstruation, food allergies, iron, vitamin B_{12} and folate deficiencies. Genetic predisposition has also been reported. RAU is more common in non-tobacco users (negative association between smoking and RAU).

Clinical features include the following:
Recurrent aphthous ulcers (RAU) are classified into three types based on the size:

1. Minor (80%) <1 cm
2. Major (10%) >1 cm
3. Herpetiform RAUs (10%): 1-2 mms in crops.

Features of minor RAU:

- Painful and recurrent ulcers
- Round, 1-5 ulcers in number, <1 cm in diameter
- Mainly occur on non-keratinised mucosa with occasional exceptions
- Present a red halo surrounding the ulcer and a shallow, yellow floor

Minor RAUs heal within 7 to 10 days leaving no scar.
Features of major RAU:

- They are recurrent and severely painful
- Major RAUs are large, usually >1 cm in diameter and found on the posterior part of the mouth (including the keratinised part)
- Major RAUs are single or multiple, round/irregular with a deep, yellow floor and red halo. Their edges may be raised (confused for Ca)
- Their healing time is from two weeks to three months. They heal leaving scar. Major RAUs are common in HIV patients.

Features of herpetiform RAU:
These ulcers are less common and resemble ulcers of primary HSV infection.

- They are painful and recurrent
- They are present in crops and each ulcer may be 1-3 mm in diameter; when they coalesce, ulcers are larger
- They are round/irregular and present a grey, shallow floor
- Favoured sites include the floor of the mouth, ventral tongue and buccal mucosa
- These ulcers heal within two weeks leaving no scar.

Diagnosis and Investigations

History and clinical examination of ulcers provide sufficient clues to diagnosis. Major ulcers lasting for more than two or three weeks should be biopsied and microscopically examined in order to rule out malignancy or tuberculosis. A full blood count (FBC),

estimation of iron, haemoglobin, vitamin B^{12} and folate are useful in identifying the underlying predisposing factors.

Management of RAU

- Initial visit: History/clinical examination, chlorhexidine mouth wash and dietary advice, topical local anaesthetic gel for symptomatic relief.
- Review 1, after four weeks. If no improvement:

–Topical steroid applications (Triamcinalone, for example).

- Review 2, after a further 4-6 weeks. If symptomatic improvement is seen, maintain topical steroids as required.
- If there is no improvement, consult a physician. Further haematological investigations may be necessary. Biopsy and microscopic examination of the lesion is necessary at this stage. Systemic steroids may be required, and refer the patient to a specialist.

If your patient with recurrent oral ulceration complains of gastrointestinal and other systemic problems, consider the following:

- **Behcet's syndrome**. Ask your patient if he/she has genital ulcerations, painful eyes, abdominal pain, nausea, diarrhoea and joint pain.
- **Coeliac disease.** Ask your patient if he/she has gluten sensitivity, loose greasy stools, abdominal pain (after eating a wheat-rich meal), itchy skin rashes and fatigue
- **Crohn's disease.** Ask your patient if he/she has diarrhoea, frequency of bowel movement, abdominal pain, eye pain and joint pain.
- **Ulcerative colitis.** Ask your patient if he/she has diarrhoea, mucus and blood in stools and arthritis.

Referral to a physician is essential in these cases.

Erosions/Ulcers of Uncertain Aetiology or Immunologic Background

- **Oral lichen planus (OLP)**

Lichen planus (LP) is a chronic mucocutaneous disorder of uncertain etiology.

LP affects 2% of the population, particularly the 30-50 year age groups, and is more common in females. LP affects skin and oral mucous membrane (70% with skin lesions have oral lesions).

Different clinical forms of LP occur. These include plaque-like, reticular, papular, erosive, atrophic or bullous lesions.

Cause of LP in unknown. Immunologic background has been suggested.

Erosive Oral Lichen Planus

- Erosive lesions are generally painful. Favoured sites include buccal mucosa. Lesions are bilateral. They present as shallow, irregular erosions and/or ulcers of varying sizes. The floor of the lesion is yellow with a layer of fibrin. Erosive lesions present an erythaematous border and the area surrounding these lesions may be white or pigmented.

Diagnosis is based on history and clinical examination. Histopathology is confirmatory. A small percentage of oral lichen planus lesions may be potentially malignant.

Management includes topical steroids or topical tacrolimus (an immunosuppressant). Severe forms need systemic treatment with steroids.

Referral to a skin specialist is necessary if dermatologic manifestations are present.

- **Erythema multiforme (EM)**

Erythema multiforme is an immunological disorder of a hypersensitivity type.

Cause: EM is an acute condition and may be recurrent depending on the precipitating factors involved. EM is drug induced or associated with HSV infection.

Symptoms and signs: "Target lesions" on the skin are common. Genital and ocular lesions may also be present. Constitutional findings are present in some cases. The severe form of erythema multiforme is called Stevens-Johnson syndrome.

Mouth ulcers/erosions of EM include multiple, irregular, shallow, painful, haemorrhagic and crusted erosions (on the lips) or ulcers.

Referral to a physician or specialist is necessary.

- **Pemphigus vulgaris**

- Pemphigus vulgaris is an autoimmune disease characterised by the development of blister forming, erosive, ulcerative lesions. This disorder involves skin and mucous membranes and is common in females between 40 and 60 years of age.

Oral Lesions in Pemphigus Vulgaris

- Oral blisters occur on buccal mucosa, the palate, and gingivae. They rupture, resulting in erosions or ulcers. Lips present with crusted lesions.
- Ulcers are of variable size and shape with ragged borders. They are shallow ulcers covered with blood stained exudates.

Referral to a physician or specialist is necessary.

- **Mucous membrane pemphigoid (MMP)**

This is an autoimmune disease which occurs mainly in patients over 50 years of age and is four times more common in females.

MMP is a blistering disorder and affects the eyes, skin and oral mucosa.

Oral Ulcers in MMP

- Soft palate/gingivae are the favoured sites for blood filled blisters of varying sizes. Lesions present irregular, well defined margins and an inflamed base.
- Eye involvement is serious and can cause blindness.

Referral to a physician or specialist is necessary.

- **Bullous pemphigoid (BP)**

- This disorder is common in the elderly and is characterised by large, tense blisters on the skin, especially on the upper arms and thighs.
- **Oral lesions** are similar to those of pemphigus but less frequent.

Referral to a physician or dermatologist is recommended.

Oral Ulcers Due to Bacterial Infections

- **Acute necrotising ulcerative stomatitis**

ANUG is characterised by progressive ulceration of the interdental papillae (punched out craters). Bleeding and tender gums are common, and halitosis is intense. Young and middle aged individuals are commonly affected. In these patients, host resistance is generally poor.

Cause: ANUG is caused by fusobacteria and spirochaetes in individuals with underlying predisposing factors.

Management includes maintenance of good oral hygiene and the use of antibiotics (metronidazole and ampicillin). Nutritional supplements are recommended.

- **Tuberculous ulcer**

Primary tuberculous oral ulcers are rare. Though uncommon, oral tuberculous ulcers are secondary lesions derived from the primary source in the lungs.

Clinical features: General symptoms include weakness, loss of weight, cough, night sweats, and haemoptysis.

Oral tuberculous ulcers are usually seen on the dorsum of the tongue. Ulcer characteristics include:

- Angular or stellate in shape
- Pale with mucus-like material on the base of the ulcer floor
- Undermined edges or margins
- Cervical lymph nodes: Enlarged or firm, sometimes with fistulae
- Fails to heal unless systemic anti-tuberculous therapy is instituted.

Referral to a physician is required.

- **Syphilitic ulcer**

Acquired syphilis is a sexually transmitted bacterial disease caused by *Treponema pallidum*.

Oral ulcers are seen in primary, secondary and tertiary forms of the disease.

Ulcers in the primary stage are called *chancres*.

- A chancre is usually single and painless.
- Favoured sites for chancres include the lips, and the tip of the tongue.
- The chancre is of variable (5 mm - several cms) size, and round in shape. Its edges are raised and indurated (clinically resembling a malignant ulcer).
- Chancres are highly infectious. They heal spontaneously (with or without treatment). They do not leave any scars.
- Ulcers of secondary syphilis are called *snail track ulcers.*
- The tongue, palate and tonsils are the favoured sites. They are flat, irregular, linear ulcers covered by a grey membrane.

In tertiary syphilis, ulcers are punched out in appearance. They are called gummatous ulcers and are extremely rare. These ulcers may cause perforations of the palate or tongue.

Referral to a specialist is necessary.

Ulcers Due to Viral Infections

- **Primary herpetic gingivostomatitis**

Primary herpetic gingivostomatitis is common in children. This is caused by herpes simplex virus type 1 (HSV-1) in the majority of cases (90%) and HSV-2 in a minority of patients (10%).

Symptoms and signs include: Constitutional symptoms such as fever, malaise and lethargy.

Cervical lymph nodes are palpable and tender.

Initially, oral involvement develops as viral blisters and these rupture leaving shallow ulcers. Gingiva, tongue, and hard palate are the favoured sites (predominantly in keratinised areas). Ulcers are multiple and painful, 2-3 mm in diameter, round and shallow with yellow/grey floors and red/inflamed margins.

Diagnosis is based on the history and clinical examination. Cytological examination of the vesicular fluid may be carried out to detect the cytopathic effects of the virus; however, this is not specific to HSV. Treatment is symptomatic since the condition is self-limiting.

Secondary infection of the HSV is common on the lips. This is called herpes labialis. Vesicular lesions develop on the upper lip and around the ala of the nose. These rupture leaving crops of tiny ulcers. Generally, there is a prodromal period of about 48 hours before vesicles appear. This period is characterised by a tingling or itching sensation in the area of the developing labial vesicular lesions. Intraoral secondary (recurrent) herpes virus infection is uncommon in the general population. This can occur in immunocompromised patients.

- **Herpangina**

Coxsackie virus causes herpangina. Constitutional symptoms such as fever and malaise are present. Herpangina is uncommon in children. Multiple vesicles rupture leaving ulcers. The soft palate and tonsillar regions are the favoured sites. Ulcers are 1-2 mm in diameter, round and shallow with surrounding red and inflamed mucosa.

Coxsackie virus also causes hand, foot and mouth (HFM) disease in children.

- **Shingles (herpes zoster)**

Reactivation of the varicella-zoster virus in adults (the elderly in particular), or immunocompromised patients, causes shingles.

Shingles affects sensory nerves and causes painful unilateral vesicular/ulcerative lesions. Diagnosis is by history and clinical examination. Antiviral treatment is recommended in severe cases. Symptomatic treatment is necessary for all patients.

Oral Ulcers Due to Fungal Infections: (Deep Mycoses)

Oral ulceration may be seen in deep mycotic infections such as histoplasmosis, mucormycosis, aspergillosis, cryptococcosis and blastomycosis.

Immunocompromised patients (with HIV, for example) are at a greater risk for the development of oral ulcers due to deep mycoses.

Oral ulcerations in superficial fungal infection (oral candidiasis, for example) are extremely rare.

Oral Ulcers Due to Systemic Disorders

Oral ulcers in haematological diseases include

- **Neutropenia.** In neutropenia, an abnormally low number of neutrophils occur. Individuals with this disorder are susceptible to infections and mouth ulcers.
- **Cyclic neutropenia** (or cyclical neutropenia) is a form of neutropenia that tends to occur every three weeks, lasting from three to six days at a time, due to changing rates of cell production by the bone marrow.

- **Leukaemia:** Acute and chronic forms of leukaemia occur. Oral symptoms include petechiae, gingival swelling, oral ulceration and infections. These are discussed elsewhere in the book.

Other causes of oral ulceration include drugs, and a condition called necrotising sialometaplasia which may result from tissue ischaemia as seen occasionally on the palate and secondary to local anaesthetic infiltration.

Gingival swellings

The majority of oral soft tissue lesions are gingival swellings. These may present as localised (focal) or generalised (diffuse) enlargements of the gingival tissues. Often gingival swellings are termed as gingival hyperplasia. Since the term hyperplasia refers to a pathological increase in the size of tissue due to an increase in the number of constituent cells, it is necessary to understand that not all gingival swellings are examples of hyperplastic change.

Causes of gingival swellings include infection, reactive proliferations secondary to irritation, drug induced lesions and rarely, neoplasms. Usually localised gingival swellings present with a broad base (sessile). Occasionally, lesions may be pedunculated. The surface of the lesions may be smooth, rough or ulcerated. Rarely, lesions may be malignant at the time of presentation.

- **Diagnostic steps**

History, clinical examination and investigations (where relevant) are essential diagnostic steps in the diagnosis of gingival swellings.

History should include:

Onset and duration, size and growth characteristics, and a history of constitutional symptoms (fever, loss of weight, weakness, etc.) should also be sought.

Examination of gingival swellings should include inspection of the swelling for its location (localised/generalised), size, shape, colour and surface characteristics (smooth, rough, papillary, etc.). Palpation for determining the swelling's consistency (soft, firm, or hard), surface characteristics, mobility and fixation (if any) to the underlying tissues, is carried out following the inspection.

Investigations employed in the diagnosis of gingival swellings include a smear for microscopy, culture, biopsy, radiography, and blood tests where relevant.

- Localised gingival swellings: The majority are due to local causes such as low grade irritation, trauma or infection of the gingival tissues. Rarely, these are due to primary or secondary (metastatic) neoplasms.
- Generalised gingival swellings: These include swellings due to inflammation (plaque-induced), effects of drugs (drug–induced), deposits of leukaemic-infiltrate, hormonal imbalances and hereditary causes.

Only a brief description of localised and generalised gingival swelling is given in this chapter. The reader is referred to texts on periodontics or oral medicine or pathology for a detailed description.

Localised Gingival Swellings

- **Fibrous epulis**

The term epulis literally means 'on the gingiva'. Fibrous epulis therefore refers to a localised fibrous growth on the gingiva.

Cause: The cause of fibrous epulis is hyperplasia of the fibrous tissue as a result of the tissue's response to mild and chronic local irritation or trauma.

Clinical features: Fibrous epulis affects both genders, with a higher predilection for females. This is common in the 4th-6th decade of life. Commonly located on the gingiva between two teeth, the fibrous growth is a pink-white firm nodule, sessile or pedunculated.

Diagnosis: History and clinical examination. Biopsy and histological examination is recommended for confirmation.

Treatment: Conservative surgical excision.

- **Pyogenic granuloma**

Pyogenic granuloma is a highly vascular localised inflammatory response to low grade irritation resulting in a localised collection of granulation tissue. The term 'pyogenic' is a misnomer, since this lesion does not produce pus.

Cause: Pyogenic granuloma is an exuberant tissue response to local irritation or trauma. The lesion may also be related to effects of female hormones in pregnancy.

Clinical features: Pyogenic granuloma occurs commonly in females. In pregnant women, it is called pregnancy epulis. Facial surfaces of gingival tissues are commonly involved. The lesion is painless or mildly painful or tender. The surface is smooth or lobulated, and red or purple in colour. It may show ulceration and in most cases the lesion is pedunculated.

Diagnosis: History and clinical examination provides clues to diagnosis. Biopsy and histology is confirmatory.

Treatment: Conservative surgical excision is the treatment of choice. Scaling adjacent teeth is required in order to eliminate sources of irritation or infection. Multiple recurrences are common.

- **Peripheral giant cell granuloma (Giant cell epulis)**

Peripheral giant cell granuloma (PGCG), also called giant cell epulis, is a reactive lesion on the gingiva characterised by the presence of granulation tissue and multinucleated giant cells.

Cause: Lesions occur as a result of a reactive response to irritation or trauma.

Clinical features: This lesion has a gender predilection in favour of females and is more prevalent in the 5th-6th decades of life. Gingiva or alveolar ridges are commonly involved, with a predilection for maxilla. Peripheral giant cell granuloma is a bluish-purple nodule and sessile or pedunculated. It may show signs of surface ulceration.

Diagnosis: Clinical examination is useful in arriving at a diagnosis of epulides, which include pyogenic granuloma (pregnancy epulis), fibrous epulis, peripheral ossifying fibroma

(ossifying fibroid epulis), and other localised gingival lesions. Biopsy and histological examination is necessary to confirm the diagnosis. PGCG lesions are characterised by the presence of multinucleated giant cells, ovoid-spindle stromal cells, RBCs, haemosiderin and occasionally, reactive bone.

Treatment: Conservative surgical excision is the treatment of choice. Adjacent teeth should be scaled, and patients need to be followed up as 10% show recurrence.

- **Peripheral ossifying fibroma (Ossifying fibroid epulis)**

Peripheral ossifying fibroma, also known as ossifying fibroid epulis, refers to a gingival lesion that is characterised by the presence of fibroblastic proliferation and bone or cementum-like mineralised structures. Fibroblasts are derived from the periosteum or periodontal ligament.

Cause: This is the result of a reactive response to low grade irritation.

Clinical features: Peripheral ossifying fibroma shows gender predilection in favour of females. Lesions are found exclusively on the gingiva with a predilection for maxilla, particularly in the incisor-canine region. The lesion is a pink or red nodule, and either sessile or pedunculated.

Diagnosis: Clinical examination, and biopsy for confirmation.

Treatment: Conservative surgical excision is the treatment of choice. Adjacent teeth should be scaled, and patients need to be followed up as 16 % show recurrence.

- **Denture granuloma (Epulis fissuratum, denture epulis, denture hyperplasia)**

Denture granuloma, also called epulis fissuratum, is a hyperplastic lesion caused by chronic irritation derived from a denture flange.

Clinical features: This is a pink hyperplastic lesion creased by a trough which is produced by the denture flange. The lesion is firm, persistent and painless. It occurs commonly on the labial surface of the maxillary alveolar ridge and the lingual surface of the mandibular alveolar ridge.

Diagnosis: History, clinical appearance and microscopic examination to confirm the diagnosis.

Treatment: Surgical removal of the hyperplastic tissue is required, and denture relining or obtaining new dentures.

- **Gingival cyst of the adult**

This is a rare cystic lesion located on the gingival caused by a cystic change in the remnants of the dental lamina.

Clinical features: Gingival cyst of the adult is a painless, well-circumscribed, bluish and sessile swelling which occurs in adults over the age of 40 years. The mandibular premolar and canine area are the favoured sites.

Clinical diagnosis should be confirmed by microscopic examination.

Treatment includes surgical removal.

- **Congenital epulis of the newborn (Gingival granular cell tumour)**

This is a rare granular cell tumour seen in newborns. This nodular lesion is either pedunculated or sessile with a gender predilection in favour of females (with a ratio of F:M 10:1). Lesions are usually found in the anterior maxillary region. Diagnosis is confirmed by histopathology and treatment is surgical excision.

- **Gingival cyst of the newborn**

This is a rare cyst that arises from the rest cells of the dental lamina. Gingival cyst of the newborn occurs on the alveolar ridge of the jaws as a white papule measuring approximately 1 - 2 mm in diameter. This lesion usually sheds itself and no treatment is necessary.

- **Melanotic neuroectodermal tumour of infancy**

A rare developmental disorder present at birth, this is an aggressive tumour of neural crest origin. Occurring in the anterior maxilla, this tumour rapidly expands. It is a destructive bony lesion which may appear blue/black on the surface. X-rays of the lesion show an ill-defined radiolucency, and the tooth buds appear to be "floating". This tumour mimics malignant neoplasm.

Diagnosis: Histopathology and detection of vanillylmandelic acid (VMA) are confirmatory as in other neural crest lesions.

Treatment is by surgical excision. Prognosis: 20% of melanotic neuroectodermal tumour of infancy recurs.

Generalised Gingival Swellings

Causes include hormonal changes, such as occur in puberty and pregnancy, bacterial plaque-induced chronic inflammation, systemic use of certain drugs (drug-induced), deposits in the gingival of leukaemic infiltrate, and hereditary transmission. Some conditions are briefly discussed here.

- **Pregnancy gingivitis**

Pregnancy gingivitis refers to gingival inflammation during pregnancy and which can result in generalised gingival enlargement. This occurs in 60-75% of pregnant women.

Cause: Increased progesterone alters the microcirculation in the gingival tissues in the presence of bacterial plaque and subgingival microflora.

Clinical features: Pregnancy gingivitis commences in the second month of pregnancy and peaks in the eighth month. Clinically, the condition presents gingival swelling, redness (generalised/localised), and bleeds easily.

- **Pubertal gingivitis**

In pubertal gingivitis hormonal changes magnify the tissue response to local irritants. This is characterised by gingival swelling with bleeding, interdental sites. Males and females are affected. Gingivitis resolves partially after puberty.

- **Inflammatory gingival enlargement**

Cause: Bacterial plaque is responsible for inflammatory gingivitis.

Clinical features: Generalised gingival enlargement with the disappearance of stippling is seen. Gums are shiny. Halitosis is present. Gingival bleeding on tooth brushing is a common complaint. This condition responds to mechanical debridement of the teeth and gums.

- **Drug-induced gingival hyperplasia**

Etiology: This is characterised by an abnormal gingival response to the use of certain systemic medications**.** The condition has strong associations with phenytoin (an anticonvulsive agent), cyclosporine (an immunosuppressant) and nifedipine (calcium channel blocker for hypertension).

The incidence of gingival hyperplasia in phenytoin users is 50%, in cyclosporine users 25%, and in nifedipine users 25%. The anterior gingival facial surface is commonly involved. The degree of enlargement is dependent upon a patient's level of oral hygiene.

Clinical features: Gingival hyperplasia typically begins 1-3 months after starting treatment. The gingiva is pink and firm. If inflamed, gingiva is red and oedematous and may completely cover the crowns of the teeth.

Diagnosis and investigations: History and clinical examination offer sufficient clues to the diagnosis. Consultation with the patient's physician is necessary for treatment, as it may possibly involve changing medications.

Treatment: Prophylaxis, gingivectomy and periodic re-evaluation are recommended.

- **Leukaemic gingival enlargement**

In acute myelogenous leukaemia (AML), gingival enlargement with leukaemic cell infiltrate is a common feature.

Clinical features: Gingival enlargement is generally boggy and non-tender. Ulceration of the gingiva and adjacent oral mucosa due to neutropenia is frequently present. Constitutional symptoms include fatigue and fever. Infection and bleeding are common in these patients.

Diagnosis: A complete blood count (CBC) should be ordered - typically, elevated WBC count is present. Other investigations include peripheral blood smear and bone marrow aspiration.

Treatment for leukaemia includes chemotherapy, radiation therapy and stem cell transplant. This is undertaken by a haematologist or oncologist. Gingival enlargement responds to chemotherapy.

- **Hereditary gingival fibromatosis**

This is an inherited disorder characterised by an increased synthesis of collagen and a decreased degradation or alteration in fibroblast proliferation, resulting in diffuse fibrous enlargement of gingival tissues. Both sexes are affected.

Gingival enlargement presents normal colour and is firm. Buccal and lingual aspects are involved, often covering the entire dentition. Gingival enlargement starts as permanent teeth erupt.

Diagnosis is based on the history and biopsy findings. Gingivectomy is commonly done, but recurrence is common.

Diseases and Disorders of the Oesophagus, Stomach and the Intestines

- **Dyspepsia**

Definition/Description: Dyspepsia is a collective term suggestive of indigestion or "upset stomach" and is due to disorders of the stomach.

Causes: Any of the following causes should be considered:

peptic ulcer, oesophagitis, gastric carcinoma, pancreatic disease, Crohn's disease, malignancy of the colon, liver and kidney failure, drugs, alcohol, pregnancy, depression and anxiety neuroses.

Symptoms: These include: discomfort or burning pain in the upper part of the abdomen related to eating, flatulence (burping, belching, excessive wind), heartburn, water brash, loss of appetite, nausea, vomiting, and abdominal bloating.

Diagnosis/Investigations: In a majority of cases, history and clinical features provide clues to diagnosis. However, to determine the underlying causes of dyspepsia, investigations such as full blood count (FBC), erythrocyte sedimentation rate (ESR), stool examination for occult blood, liver function tests for alcoholism, endoscopy and/or a barium meal to exclude organic disease may be carried out..

Management: Reassurance, stress relief by counselling, avoidance of smoking and alcohol, use of antacids, and H2 receptor antagonists for night pain are some of the measures to treat dyspepsia. Medical or surgical approaches to treating organic disease involving the gastrointestinal tract are also important measures.

- **Dysphagia**

Definition/Description: Difficulty in swallowing food or liquid is called dysphagia.

Causes: Causes of dysphagia include the following:

peptic oesophagitis, reflux disease, swallowed foreign body, severe iron deficiency anaemia (as in Plummer–Vinson syndrome or Patterson–Brown-Kelly syndrome), bulbar palsies (refers to the lower brain stem that controls the functions of cranial nerves VII-XII) and pseudo-bulbar palsy, myasthenia gravis, achalasia (failure of the lower oesophageal sphincter to relax due to improper innervation), anxiety (globus hystericus), oesophageal spasm, stomatitis, pharyngitis and tonsillitis.

Some extrinsic, less common causes include pressure from mediastinal lymph nodes, bronchial carcinoma, thyroid enlargement, aneurysm of the aorta, pharyngeal diverticula filled with food or liquid, strictures of the oesophagus and Barrett's oesophagus, (characterised by the replacement of squamous epithelium by columnar and goblet cells). Patients with this change (metaplasia) in Barrett's oesophagus are at a higher risk of adenocarcinoma.

Symptoms: Common symptoms of dysphagia include difficulty in swallowing food or liquids, complaining of a 'lump in the throat' when not swallowing, pain or burning sensation while swallowing, bulge or gurgle in the neck when drinking, and regurgitation of food.

Diagnosis/Investigations: Full blood count (FBC), erythrocyte sedimentation rate (ESR), x-ray with barium swallow and/or endoscopy (with or without biopsy), CT scan of the thorax, and oesophageal motility studies.

Management: Depending on the identified causes of dysphagia, treatment modalities include endoscopic or pneumatic dilatation of the oesophagus, the correction of iron deficiency, surgical resection, and use of muscle relaxants, antacids or proton pump inhibitors.

- **Gastro-oesophageal reflux disease (GORD)**

Definition/Description: Gastro-oesophageal reflux disease (GORD) is a condition characterised by inflammation of the oesophagus, usually due to reflux of acid from the stomach.

Causes/predisposing factors: These include obesity, big meals, tight clothing, fatty meals, alcohol, smoking, pregnancy, hiatus hernia, and drugs such as tricyclics (antidepressants) and anticholinergic agents (Buscopan, for example). Anticholinergic agents are parasympathetic nerve impulse inhibitors which inhibit involuntary movements of the smooth muscles of the GIT and other sites.

Symptoms: Dull, retrosternal ache (heart burn) is often triggered by food, coffee, or alcohol, and aggravated by bending, lying flat, lifting weights or straining. Pain may radiate to the throat and to the back. These symptoms often mimic angina. Regurgitation of stomach contents into the mouth, and transient or permanent dysphagia in relation to solids, arecommon.

Signs: No identifiable signs are evident in the early stages of GORD. In advanced cases, signs of haematemesis (vomiting of blood), anaemia, aspiration pneumonia, peptic stricture, and Barrett's oesophagus may be present. Barrett's oesophagus is a risk factor for the development of adenocarcinoma.

Oral Manifestations and Dental Management Considerations

- Regurgitation of gastric contents in patients with GORD (and in pyloric stenosis) may cause dental erosion on the palatal aspects of the maxillary anterior teeth and premolars.
- Oral adverse effects of medication for GORD and peptic ulcer disease may include dry mouth from proton pump inhibitors (PPI), and sucralfate (a cytoprotective agent that protects gastric mucosa). Sucralfate causes constipation, so the use of codeine should be avoided.

- PPIs interfere with calcium absorption. X–rays of the jaw bones should be periodically checked for any changes in bone density.
- Erythema multiforme from ranitidine, (a histamine H_2-receptor antagonist that inhibits stomach acid production) and loss of taste from omeprazole (a PPI) are often reported. Erythema multiforme is a skin/mucocutaneous condition which usually occur secondary to drug use or viral infections.
- If patients are on antacids containing aluminium hydroxide (Mylanta or Gelusil, for example), tetracyclines, metronidazole, erythromycin and ciprofloxacin should be avoided because they are not absorbed adequately.

Diagnosis/Investigations: Endoscopy with or without biopsy, barium meals, and oesophageal motility studies are commonly used investigations for GORD. Resting ECG is helpful to rule out ischaemic heart disease.

Management: Depending on the severity and causes of GORD, management protocols include weight reduction, small volume frequent meals, avoidance of alcohol, fatty foods, smoking, caffeine and late night meals. Other measures include elevating the head end of the bed, use of antacids and H2 receptor antagonists, oesophageal dilatation, the surgical resection of strictures, and surgical treatment of hiatus hernia.

- **Gastritis**

Definition/Description: Gastritis is characterised by acute or chronic inflammation of the gastric mucosa which is sometimes accompanied by erosions.

Causes: In a majority of cases, over-indulgence in alcohol or drugs (aspirin or anti-rheumatic drugs, for example) cause gastritis. Chronic forms may be caused by *H. pylori* infection, or from autoimmune processes.

Symptoms and signs: These include epigastric pain after eating, vomiting, indigestion, loss of appetite and weight loss. In autoimmune disorders, pernicious anaemia is common.

Diagnosis/Investigations: No special investigations are necessary in most cases. When erosions are suspected, endoscopy is recommended. If pernicious anaemia is present, haematological examination is necessary. A urea breath test, rapid urease test, culture and histology tests for *H. pylori* are recommended, particularly for chronic gastritis.

Management: Avoidance of causative factors is essential. H2 receptors are useful in reducing acid production and *H. pylori.*

- **Hiatus hernia**

Definition/Description: Hiatus hernia is a protrusion of the stomach through a hiatus (aperture) in the diaphragm. There are two types: sliding (most common) and paraesophageal hernias. In the sliding type, the gastroesophageal junction and a portion of the stomach are above the diaphragm, whereas in paraoesophageal hiatus hernia, the gastroesophageal junction is in the normal location, but a part of the stomach is adjacent to the oesophagus.

Cause: The exact cause of hiatus hernia is not known. Stretching of the fascial attachments between the oesophagus and the diaphragm at the hiatus appears to be a feature of its development. Gastroesophageal reflux disease is associated with hiatus hernia in a considerable number of patients.

Symptoms and signs: Many patients with hiatus hernia are asymptomatic. Often hernias are noted as incidental findings on x-rays. Chest pain may be a feature in some patients. Strangulation of the hernia is a complication.

Diagnosis/investigations: Chest x-rays and barium swallows confirm a clinical suspicion of hiatus hernia.

Management: Small hernias do not need specific therapy. If reflux disease is present, it should be treated. Paraoesophageal hernia is reduced surgically to avoid strangulation.

- **Gastroenteritis**

Definition/description: Gastroenteritis is the inflammation of the mucosal surfaces of the stomach, and the small and large intestines.

Cause: The majority of gastroenteritis cases are due to infections. Ingestion of chemicals and drugs may also cause gastroenteritis in a minority of patients.

Infectious agents include viruses (rotaviruses and adenoviruses, for example), bacteria (such as *Vibrio cholera species, Escherichia coli, Shigella spp, Salmonella spp, Clostridium deficile*, and *Campylobacter spp*) and parasites (including *Giardia lamblia* and *Cryptosporidium parvum*). In Asian countries, an acute epidemic of gastroenteritis is commonly caused by *Vibrio choleriae*. This condition is known as cholera.

Symptoms and signs: The sudden onset of symptoms, which include nausea, vomiting, rumbling noises in the intestines (borborygmi), abdominal cramps, and diarrhoea with or without blood and mucus. Persistent vomiting and diarrhoea may result in fluid depletion along with tachycardia and hypotension. This may lead to shock in severe cases. Hypokalemia (abnormally low levels of potassium ions in the circulating blood) results if both vomiting and diarrhoea are severe.

Diarrhoea is watery (without mucus or blood) in viral gastroenteritis. Bloody diarrhoea with fever is a feature of bacterial gastroenteritis (caused by *Shigella* or *Salmonella*, for example). Gastroenteritis caused by parasites does not cause bloody diarrhoea (with the exception of amoebic dysentery caused by *E. histolytica)*. Diarrhoea may be of subacute or chronic form.

Diagnosis/Investigations: A history of ingesting contaminated food, recent travel and copious watery diarrhoea may provide sufficient information as to the possible cause. Differential diagnosis of other diarrhoeal disorders, such as appendicitis, ulcerative colitis and cholecystitis, needs to be performed in order to exclude these possibilities.

Stool testing for blood, parasites, WBCs and culture studies are indicated. Serum electrolytes, blood urea nitrogen (BUN) and creatinine are evaluated for hydration and acid-base status.

Management: Bed rest, access to toilets, oral glucose-electrolyte solutions, frequent sips of fluids; antidiarrhoeal agents (loperamide, for example), antiemetic and antimicrobial agents and probiotics are used in the management of gastroenteritis.

- **Appendicitis**

Definition/description: Appendicitis is characterised by acute inflammation of the vermiform appendix, resulting in nausea and acute pain around the umbilicus.

Cause: Obstruction of the appendiceal lumen by faecalith, lymphoid hyperplasia or worms leading to bacterial growth, are common causes of appendicitis. Ischaemia, inflammation and gangrene may cause perforation if appendicitis is not promptly treated.

Symptoms and signs: These include epigastric or peri-umbilical pain followed by nausea and vomiting. A few hours later, pain shifts to the right lower quadrant and the severity of the pain increases with coughing or movement. Low grade fever is common.

Diagnosis/investigations: In most cases, history and clinical presentation provide clues to the diagnosis of appendicitis. A CT scan or ultrasound imaging and laparoscopy are useful diagnostic tools. Blood examination shows leucocytosis in most cases.

Management: An appendectomy followed by intravenous antibiotics is the treatment of choice.

- **Acute Abdomen**

Definition/Description: Acute abdomen is characterised by the sudden onset of acute abdominal pain that causes the patient to be hospitalised within a few hours.

Cause: Acute abdomen can be caused by several conditions. These include: perforation due to duodenal ulcer, rupture or torsion, acute pancreatitis, renal tract disease, acute appendicitis, acute peritonitis, intestinal obstruction, and strangulated hernia.

Symptoms and signs: These include pain, vomiting, fever, furred tongue, and diarrhoea. If pain is due to non-intestinal sources, symptoms of conditions such as renal colic, pain due to myocardial infarction (referred) and pneumonia (referred) may be present.

Diagnosis/Investigations: These include blood counts (a raised WBC in inflammatory conditions), serum amylase (raised in pancreatitis), x-rays (perforation), ultrasound (cholangitis and aortic aneurysm), a CT scan, and laparoscopy.

Management: Hospitalisation and IV fluids with mild analgesics and antibiotics are essential measures. In cases of appendicitis surgery and for obstruction, laparotomies are performed.

- **Peptic ulcer disease (Gastric and duodenal ulcers)**

Definition: Peptic ulcer disease is characterised by well-defined ulcers in the gastrointestinal mucosa. There are two types: gastric ulcers and duodenal ulcers.

Cause: Peptic ulcer disease is often caused by chronic acid-pepsin secretions, the harmful effects of *Helicobacter pylori,* NSAIDs and Crohn's disease.

Symptoms and signs: These include epigastric pain and tenderness. Pain associated with eating points to gastric ulcers. Pain between meals and during the night is suggestive of duodenal ulcers. Pain tends to wax and wane, is aggravated during stress and with the use of drugs and alcohol.

Signs of anaemia due to bleeding from the ulcers may be present. Ingestion of food, milk or antacids provides temporary relief. Protracted vomiting a few hours after meals is a sign of gastric outlet (pyloric) obstruction. Black tarry stools (melena) due to gastrointestinal haemorrhage, and weight loss, are other features of peptic ulcer disease.

- In peptic ulcer disease, oral signs and symptoms of anaemia may be present.
- Aspirin, anti-inflammatory drugs such as NSAIDs, and corticosteroids should be avoided in peptic ulcer disease.
- Antibiotics should be taken 2 hours before or 2 hours after antacids. This is because antibiotics such as ampicillin need the presence of acid for its absorption.
- Long term use of antibiotics taken for peptic ulcers may sometimes promote oral fungal infections.

Diagnosis/Investigations: Endoscopy, barium meal or biopsy (to rule out gastric malignancy) are recommended diagnostic measures. Biopsy is not required for duodenal ulcers as the duodenum is a rare site of malignancy. *H. pylori* status assessment through urease activity, histology, serology and urea breath tests are useful.

Management: *H. pylori* positive ulcers are treated with proton pump inhibitors (omeprazole, for example) and two antibiotics (metronidazole and clarithromycin) for one week, followed by an endoscopy or urea breath test.

Pylori negative ulcers are treated with H2 receptor antagonists (cimetidine, for example) and the use of sucralfate (a mucosa protecting agent).

Avoidance of NSAIDS, smoking, and alcohol is essential. Surgical dissection of the vagus nerve is carried out in some cases to reduce acid secretion**.**

- **Inflammatory Bowel Disease (IBD): (Crohn's disease and Ulcerative colitis)**

Inflammatory bowel disease comprises two clinical entities: Crohn's disease and Ulcerative colitis.

1. Crohn's disease

Definition/description: Crohn's disease is also known as regional enteritis. This is an inflammatory disease of any part of the gastrointestinal tract. The small bowel is involved in 80% of cases.

Cause: Unknown. There is a genetic association with Crohn's disease; siblings of affected individuals are at higher risk of developing this disease. Males and females are equally affected. Smokers are twice as likely to develop Crohn's disease as non-smokers.

Symptoms and signs: These include malaise, anorexia, weight loss, nausea, fever, abdominal pain, diarrhoea, arthralgia, rectal bleeding, pallor, finger clubbing, erythema nodosum (inflammation of fat cells under the skin appearing as nodules, usually seen on both shins) and perianal abscesses.

Oral Manifestations and Dental Management Considerations

Oral manifestations include diffuse labial, gingival or mucosal swelling, cobblestoning of buccal mucosa and gingiva, aphthous ulcers, mucosal tags, angular cheilitis and oral granulomas.

- Antibiotics which promote diarrhoea and NSAIDs should be avoided.
- Avoidance of codeine in patients already on antidiarrhoeal medication is recommended because codeine causes severe constipation in these patients.
- for those with Crohn's disease, assessment of FBC, LFTs, and CRPs prior to invasive dental treatment is recommended.

Diagnosis/Investigations: These include a full blood count (FBC) for anaemia, WBC count (elevated), ESR count (elevated), platelet count (increased), the level of C-reactive protein (a protein in the blood as a marker of inflammation - elevated), plain abdominal x-ray, barium meal endoscopy, radionuclide scanning, and biopsy for histology.

Management: Crohn's disease is treated with corticosteroids (prednisolone), antibiotics (metronidazole) and dietary replacement of fluids and electrolytes. Surgical treatment may be required for strictures.

2. Ulcerative colitis

Definition/description: Ulcerative colitis is the chronic inflammation of all or part of the colon.

Cause: Unknown. Probable factors implicated include genetic, immunological, dietary and psychological.

Symptoms and signs: These include diarrhoea with blood and mucus, malaise, lethargy and anorexia, weight loss, abdominal pain, mouth ulcers, erythema nodosum, arthritis, uveitis, pallor, and weakness.

Oral Manifestations and Dental Management Considerations

Oral manifestations can occur during periods of exacerbation of the disease. These include aphthous ulceration or superficial haemorrhagic ulcers, angular stomatitis, and pyostomatitis vegetans (an inflammatory condition seen in inflammatory bowel disease).

Antibiotics which promote diarrhoea should be avoided. Avoidance of codeine in patients already on antidiarrhoeal medication is recommended because codeine causes severe constipation in these patients.

Diagnosis/Investigations: Blood tests include FBC for anaemia and WBCs (increased), and ESR (elevated). Liver function tests (LFTs) may be abnormal. Colonoscopy, plain x-ray and biopsy for histology may be necessary for confirmation.

Management: Treatment with IV fluids, blood transfusion, parenteral nutrition and steroids are used to treat patients with ulcerative colitis. Surgery may be necessary for some cases.

- **Coeliac disease**

Definition/Description: Coeliac disease is also known as gluten sensitive enteropathy. It is characterised by the atrophy of the jejunal mucosa due to its sensitivity to dietary gluten.

Cause: Gluten in the diet is the cause of coeliac disease. Gluten has two components: glutenin and α-gliadin. The latter is antigenic. Genetic predisposition to coeliac disease exists (HLA B8 and BR3).

Symptoms and signs: These include diarrhoea, steatorrhoea, weight loss, abdominal pain, anaemia, muscle wasting, skin pigmentation, peripheral oedema, and dermatitis herpetiformis (itchy, blistering skin disease).

Oral Manifestations and Dental Management Considerations

- In coeliac disease, glossitis, angular cheilitis, bleeding tendencies, oral mucosal ulcers, dental enamel hypoplasia, delayed eruption, and anaemia may be encountered.
- The status of iron levels, bone density, vitamin K, folic acid and vitamin B_{12} should be determined prior to invasive dental procedures in patients with coeliac disease.
- Consultation with the patient's GP is recommended for patients prior to the commencement of invasive dental procedures.

Diagnosis/Investigations: A full blood count may reveal anaemia and Howell-Jolly bodies. Howell-Jolly bodies are basophilic nuclear remnants in circulating erythrocytes.

Liver Function Tests for hypoalbuminaemia, endoscopy and jejunal biopsy for atrophic mucosa, detection of endomysial antibodies (IgA) and xylose tolerance test are other tests used in identifying coeliac disease.

Management: A gluten-free diet should be adopted. Signs of anaemia need to be treated.

- **Irritable bowel syndrome (IBS)**

Definition/description: Irritable bowel syndrome (IBS) is characterised by constipation, diarrhoea, abdominal pain (in the left iliac fossa) and the frequent passage of stools.

Cause: Psychological and stress related, in most cases.

Symptoms and signs: Symptoms include pain in the left iliac fossa or epigastrium (aggravated by eating and relieved by defecation), abdominal bloating, and alternating diarrhoea and constipation. Signs include passage of mucus in stools, abdominal tenderness and mucus on rectal examination.

Oral Manifestations and Dental Management Considerations

- Psychogenic oral symptoms, such as facial pain and TMD symptoms, may be present in these patients.
- Routine dental treatment can be offered to IBS patients.

Diagnosis/Investigations: These include rectal examination, barium enema and sigmoidoscopy.

Management: An increase in the fibre content of the diet, use of antispasmodics, and antidiarrhoeal agents are advised. Antidepressants are recommended for IBS.

- **Haemorrhoids (Piles)**

Definition: Haemorrhoids are dilated veins of the haemorrhoidal plexus in the lower rectum. Two types exist: internal and external haemorrhoids.

Cause: Increased intra-abdominal pressure, particularly from constipation, is the major cause of haemorrhoids.

Symptoms and signs: Haemorrhoids are often asymptomatic. Internal haemorrhoids are painless but can cause fresh bleeding during or after bowel movements. Blood typically covers the normally-coloured stools. This condition is called haematochezia. Sometimes blood is observed on the toilet paper or toilet bowl. Faecal incontinence, mucus discharge, and itchiness around the anus may occur.

External haemorrhoids show a swelling protruding from the anus. If thrombosed, these can be painful. Pruritus ani may be a feature in some cases.

Strangulated haemorrhoids occur if protrusion and constriction cause occlusion of the blood supply. These are painful and sometimes cause ulceration.

Diagnosis/Investigation: History and inspection or anoscopy are adequate for diagnosis of haemorrhoids.

Management: Symptomatic treatment with stool softeners (e.g., psyllium), warm baths, and the application of a local anaesthetic ointment for external haemorrhoids are adequate. Rubber band ligation is effective for large internal haemorrhoids.

- **Dysentery: Amoebic dysentery and bacillary dysentery**

Definition/description: Dysentery is a disease marked by frequent watery stools often with blood and mucus, abdominal pain, tenesmus (spasm of the anal sphincter with an urgent desire to evacuate the bowel), fever and dehydration.

Cause: These include infection with the protozoa *Entamoeba histolytica* (amoebic dysentery, amoebiasis), the bacteria *Shigella dysenteriae* (bacillary dysentery), and *sSchistosoma mansoni* (bilharzial dysentery).

1. Amoebic dysentery

Definition/description: Amoebic dysentery is characterised by diarrhoea resulting from ulcerative inflammation of the colon caused by *Entamoeba hystolytica*.

Cause: *Entamoeba histolytica* (intestinal protozoa) exist as trophozyte and cyst forms. Usually cysts are present in the formed stools of infected persons. They spread directly from person to person, or indirectly via food or water. Rarely, amoebiasis can be transmitted sexually through oral-anal contact.

Symptoms and signs: Most infected persons are asymptomatic. In those with symptoms, diarrhoea, constipation, flatulence and cramping abdominal pain are common.

Amoebic dysentery is common in the tropics. Infected persons complain of semi-solid stools with mucus and blood, abdominal pain and fever. Stools may contain trophozoietes. A tender abdomen often mimics appendicitis. Anaemia due to bleeding is a constant finding. The chronic form of the disease mimics inflammatory bowel disease.

Oral manifestations: There are no specific oral manifestations seen in amoebiasis. Oral symptoms secondary to anaemia are often encountered in patients with long standing amoebiasis.

Diagnosis/investigations: Amoebic dysentery is often misdiagnosed as irritable bowel syndrome, regional enteritis, or diverticulitis. It can also be confused with shigellosis, (bacillary dysentery), salmonellosis and schistosomiasis, or ulcerative colitis. Diagnosis is by identifying *E. histolytica* in the stool specimen, or by serologic tests.

Management: Treatment for the symptomatic disease is metronidazole or tinidazole, followed by drugs active against cysts in the lumen.

2. Bacillary dysentery (Shigellosis)

Definition/description: Bacillary dysentery is an acute infection of the intestine with *Shigella* sp.

Cause: *Shigella* species, particularly the virulent type *S. dysenteriae*, cause bacillary dysentery**.** The source of infection is the faeces of infected people or convalescent carriers. Spread is by the faecal-oral route and by contaminated food and fomites. Flies also serve as vectors.

Symptoms and signs: The incubation period is 1-4 days. Watery diarrhoea is the most common symptom. Gripping abdominal pain and an urgency to defecate are the main symptoms in adults. Stools may contain mucus, pus or blood. In young children, the sudden onset of nausea, vomiting, diarrhoea, fever, anorexia, drowsiness and abdominal pain and distension are common. Within three days mucus, blood and pus appear in the stools. Significant dehydration and the loss of electrolytes may lead to death in debilitated adults and children under two years of age.

Oral manifestations: There are no specific oral manifestations seen in bacillary dysentery. Dehydration may lead to xerostomia which can be corrected with rehydration.

Diagnosis/investigations: Suspected individuals in areas of outbreak should be tested further. Stool examination for WBCs and positive cultures for *Shigella* are diagnostic.

Management: Usually, supportive treatment in the form of rehydration is adequate. Antibiotics (ampicillin) may be required for severe cases, and for children and the elderly.

- **Malabsorption**

Definition/description: Malabsorption syndromes are characterised by the inadequate absorption of dietary substances during digestion, absorption and transport, affecting proteins, carbohydrates, fats, vitamins and minerals, which results in nutritional deficiencies.

Causes: Causes of malabsorption include gastrocolic fistula, gastrectomy, biliary obstruction, chronic liver failure, chronic pancreatitis, alcohol abuse, acute intestinal infections, coeliac disease, amyloidosis, Crohn’s disease, Addison’s disease and radiation enteritis.

Symptoms and signs: The effects of unabsorbed substances include diarrhoea, steatorrhoea with pale, bulky, greasy, foul–smelling stools, abdominal bloating, and gas. Other symptoms result from nutritional deficiencies. Weight loss may be a feature in most patients despite an adequate food intake.

Oral manifestations: When oral lesions are seen in malabsorption syndromes, they usually relate to the causes of malabsorption such as Crohn's disease, coeliac disease, amyloidosis, Addison's disease and other disorders.

Diagnosis/investigations: If history suggests a specific cause (liver failure, pancreatitis, etc.) testing should be directed towards that condition. Chronic diarrhoea, weight loss and anaemia are suggestive of malabsorption. Faecal fat estimation is helpful in determining steatorrhoea. Faecal fat >6 g/day is abnormal. Endoscopy with or without small bowel biopsy, reveals a mucosal disease of the small bowel. The Schilling test assesses malabsorption of the vitamin $B_{12.}$

Management: The causes of malabsorption must be identified and treated.

1. Diarrhoeal disease

Definition/Description: Diarrhoea is a symptom. It is defined as the passage of three or more loose or liquid stools per day (>300 ml of liquid faeces per day). Frequent passing of formed stools is not diarrhoea, nor is the passing of loose, "pasty" stools by breastfed babies. Mechanisms involved in diarrhoea include increased osmotic load, increased secretion, inflammation, and decreased absorption time.

Causes: Diarrhoea can be due to infectious, drug-induced, food-related, surgical, inflammatory, or malabsorptive causes. Infections include non-specific bacteria such as *Campylobactor spp, Salmonella spp, Escherichia coli, Shigella spp, Staphylococci, Vibrio cholera, Clostridium difficile, and Yersinia enterocolitice.* Viral, fungal and protozoal infections also cause diarrhoea. *Entameba histolytica* causes a diarrhoeal disease called amoebiasis, which is common in the underdeveloped tropics. Among the inflammatory causes, ulcerative colitis and Crohn's disease are important entities to be considered. Malabsorption syndromes causing diarrhoea include coeliac disease, short bowel syndrome and radiation. Other intestinal causes of diarrhoea include diverticular disease, irritable bowel syndrome, and bowel cancer. Causes of drug-induced diarrhoea include laxatives, antibiotics, magnesium-containing antacids, and cytotoxic drugs.

Symptoms and signs: Symptoms depend on the cause. Common symptoms of diarrhoea may include frequent loose, watery stools, abdominal cramps, bloating, abdominal pain and tenderness, bleeding, fever, lightheadedness or dizziness from dehydration. Vomiting may be present in bacterial or viral diarrhoea. Steatorrhoea (presence of excess fat in stools) and bloody diarrhoea may be a feature of chronic diarrhoea.

Oral Manifestations and Dental Management Considerations

There are no specific oral manifestations of diarrhoeal diseases. Dehydration causes a dry mouth. Patients may complain of altered taste as well. Oral manifestations of Crohn' disease, ulcerative colitis, and coeliac disease (which can cause diarrhoea) present oral aphthous-like ulcerative lesions.

Investigations: Diagnosis is often made on the history and clinical examination. General investigations include FBC, Hb estimation, WBC count, ESR, urea and electrolytes, stool culture and microscopy, colonoscopy, sigmoidoscopy, barium enema, and faecal fats. Not all investigations are carried out in patients with diarrhoea. Selecting the investigation is based on clinical judgment after considering the history and examination.

Management: Diarrhoeal management includes identifying and treating the cause of diarrhoea, and symptomatic treatment. Severe diarrhoea requires fluid and electrolyte replacement to correct dehydration, electrolyte imbalance and acidosis. Plenty of oral fluids are recommended. Medications which are known to promote diarrhoea should be avoided.

Chapter 7

Diseases of the Liver, Gall Bladder and Pancreas

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Abstract

In this chapter, liver diseases and those of the gall bladder and pancreas are discussed. The chapter begins with the common signs and symptoms of liver disease in general. This is followed by investigations employed in the diagnostic process. Liver diseases of dental interest discussed in this chapter include viral hepatitis, alcoholic liver disease, liver cirrhosis, liver cancer (hepatocellular carcinoma), gallstones (cholelithiasis), cholecystitis and pancreatitis. Other topics discussed of dental interest are jaundice, ascites and hepatomegaly**.** Each disease entity starts with the definition or description followed by its causes, symptoms, signs, the investigations employed, and principles of management. Where relevant, oral and dental aspects of diseases of the liver, gall bladder and pancreas are also described.

Introduction

Liver diseases, and those of the gall bladder and pancreas, are common. From the dental practice point of view, disorders of these organs are important because of their direct and indirect relevance to oral health.

Liver diseases are caused by viral infections, alcohol, autoimmunity, malignancy or genetic disorders. They include the following:

1. **Hepatitis** is inflammation of the liver. This is caused mainly by viruses (viral hepatitis). Hepatitis can also be caused by some liver toxins (e.g., alcoholic hepatitis), autoimmunity (autoimmune hepatitis) or hereditary conditions.
2. **Alcoholic liver disease** is any hepatic manifestation of alcohol overconsumption. This group can also include fatty liver disease, alcoholic hepatitis, and cirrhosis.

3. **Fatty liver disease** (hepatic steatosis) is a reversible condition where large vacuoles of triglyceride fat accumulate in liver cells. Non-alcoholic fatty liver disease is a spectrum of diseases, some of which are associated with obesity and metabolic syndrome.
4. **Liver Cirrhosis** is the formation of fibrous tissue (fibrosis) in the place of liver cells that have died due to a variety of causes, including viral hepatitis, alcohol overconsumption, and other forms of liver toxicity. Cirrhosis causes chronic liver failure.
5. **Primary liver cancer** most commonly manifests as hepatocellular carcinoma or cholangiocarcinoma. Many liver malignancies are secondary lesions that have metastasized from primary cancers in the gastrointestinal tract and other organs, such as the kidneys, lungs, breast, or prostate.
6. **Primary biliary cirrhosis** is a serious autoimmune disease causing inflammation of the biliary system.
7. **Budd-Chiari syndrome** is the clinical picture caused by occlusion of the hepatic vein, which in some cases may lead to cirrhosis.
8. **Hemochromatosis** is a hereditary disorder that causes damage to the liver due to the accumulation of excess iron in the body.
9. **Wilson's disease** also known as hepatolenticular degeneration is a genetic disorder in which copper accumulates in tissues. This disease manifests as neurological or psychiatric symptoms and liver disease.
10. **Glycogen storage disease type II.** Glycogen storage disease type II (GSD-II) is also known as Pompe disease or acid maltase deficiency. This is a fatal genetic muscle disorder caused by a deficiency of acid α-glucosidase (GAA) which is a glycogen degrading lysosomal enzyme
11. **Hereditary amyloidosis.** The most common type of hereditary amyloidosis is transthyretin amyloidosis of the liver.
12. **Gilbert's syndrome** is a genetic disorder of bilirubin metabolism. [3]

In this chapter, from the dental practice point of view, only a few diseases are briefly discussed.

General Signs and Symptoms of Liver Disease

Jaundice: Also called icterus, jaundice is characterised by yellow pigmentation of the skin, oral mucous membrane and sclera due to deposition of bilirubin. Jaundice is detectable when bilirubin is above 30-60 mmol/L. Jaundice is often seen in liver disease such as hepatitis or liver cancer. It may also indicate obstruction of the biliary tract by, for example, gallstones or pancreatic cancer. Less commonly, jaundice is congenital in origin (e.g., biliary atresia).

Spider angioma (spider nevi): These are small arterial dilatations on the skin of the face and neck. They contain a central red spot and reddish extensions which radiate outwards like a spider's web. They occur in healthy adults and young children. Having more than five spider nevi generally indicates liver disease.

Palmar erythema: Redness of the palms is called palmar erythema. These are often referred to as liver palms. Palmar erythema also occurs in pregnancy, polycythemia and thyrotoxicosis.

Dupuytren's contracture: This condition is characterised by fixed flexion of the little, and sometimes ring, finger due to thickening of the palmar fascia. Dupuytren's contracture has a genetic background in some cases. It also occurs in patients with diabetes, alcoholism and epilepsy.

Finger clubbing: Finger (nail) clubbing is characterised by the loss of the normal angle at the bed of the nails. This is a common feature in liver cirrhosis (primary biliary cirrhosis in particular). Other conditions where finger clubbing is a feature include cystic fibrosis of the lungs, lung cancer, pulmonary tuberculosis, suppurative lung disease, congenital cyanotic heart disease, Crohn's disease, subacute bacterial endocarditis and thyrotoxicosis. Idiopathic clubbing is sometimes seen in normal persons.

Ascites: Ascites is characterised by the accumulation of serous fluid in the peritoneal cavity. This causes abdominal distension and bulging flanks. Ascites can be a feature of liver disease, heart failure, protein-calorie malnutrition, nephritic syndrome and metastatic cancers.

Leukonychia: White discolouration appearing on the nails is called leukonychia. Leukonychia occurs in liver disease and in those patients on chemotherapy. In healthy individuals, leukonychia may have a genetic background.

Terry's nail: This is a physical finding in which fingernails and/or toenails appear white with a brown or pink arc at the tip of the nails. This condition is considered to be due to a decrease in vascularity and an increase in connective tissue within the nail bed. It frequently occurs in hepatic failure and cirrhosis. Terry's nails may also occur in patients with renal failure and congestive heart failure, hyperthyroidism, diabetes and malnutrition.

Caput medusae: This is characterised by a pattern of dilated cutaneous veins radiating from the umbilical area. This feature is observed in adults with cirrhosis of the liver with portal hypertension. In newborn babies this feature is common and is not indicative of liver disorder.

Ankle oedema: Abnormal accumulation of interstitial fluid in the tissues around the ankles causes ankle oedema. In addition to liver diseases, causes of ankle oedema include kidney failure, heart failure, varicose veins, deep vein thrombosis and extended periods of standing. Under these circumstances ankle oedema is generally of pitting dependant type. Non-pitting oedema of the lower limbs is seen in hypothyroidism, lymphoedema and allergy.

Petechiae and ecchymosis: Often seen in lever disease are multiple haemorrhagic conditions presenting as petechiae, and ecchymosis on the skin due to an underlying clotting defect. Petechiae are minor haemorrhagic spots on the skin and mucous membranes which are smaller than 3 mm in diameter. They are usually caused by thrombocytopenia or deficiency of clotting factors. Ecchymosis is a subcutaneous collection of blood as a result of haemorrhage. These lesions are larger than petechiae.

Investigations in Liver Disease

Depending on the type and severity of liver disease, any of the following investigations may be necessary:

1. Full Blood Count (FBC) for platelet count and microcytosis
2. Liver Function Tests for liver dysfunction
3. Serological investigations for viral hepatitis:
    a. Anti-HBs Ag (previous HBV infection or immunisation)
    b. IgM Anti-HBcAg - recent HBV infection
    c. IgM Anti-HAV - recent HAV infection
    d. Anti-HDV - previous delta virus infection
    e. Anti-HCV - chronic hepatitis C infection
    f. HCV-RNA - active HCV infection
4. Radiology for detection of air within the biliary tree and for the presence of gallstones
5. CT scan - for lesions within the liver parenchyma
6. MRI - for lesions of the liver, biliary tree and pancreas
7. Angiography of the hepatic artery - for hepatic tumours and portal hypertension
8. Endoscopy for gastroesophageal varices
9. Liver biopsy for the staging of most hepatic disorders

Liver Diseases of Dental Interest

- **Viral Hepatitis**

Viral hepatitis is inflammation of the liver caused by a range of viruses. The disease is seen in acute and chronic forms. Common viruses involved in the causation of hepatitis include hepatitis viruses A, B, C, D and E. Other viruses that can cause hepatitis include Epstein-Barr virus (EBV), herpes simplex virus (HSV), cytomegalovirus (CMV), and yellow fever virus. In this chapter, from the dental point of view, hepatitis caused by the foregoing viruses is briefly discussed.

Hepatitis A virus infection

Hepatitis A viruses are RNA viruses transmitted through the faecal-oral route. Risk groups for hepatitis A virus infection include food handlers and day care workers with poor hygiene. Incubation of the virus is between 15 and50 days. There is no carrier state for this infection. Prophylaxis is through immunoglobulin (Ig) and vaccine. Immunity following infection is probably for life.

Hepatitis B virus infection

Hepatitis B viruses are DNA viruses transmitted through percutaneous, sexual or perinatal routes. Risk groups for hepatitis B virus infections include IV drug users, healthcare workers dealing with blood, haemodialysis patients, male homosexuals, heterosexuals with multiple partners, and recipients of blood transfusions. Incubation of the virus is 30-180 days. There is a carrier state for this infection. Prophylaxis is through Hepatitis B immunoglobulin (HbIg) and vaccine. Immunity following infection is probably lifelong.

Hepatitis C virus infection

Hepatitis viruses are RNA viruses transmitted through percutaneous, and occasionally sexual or perinatal routes. Risk groups for hepatitis C virus infections include IV drug users, healthcare workers dealing with blood, haemodialysis patients, and recipients of blood transfusions. Incubation of the virus is 15-160 days. There is carrier state for this infection (50-80%). There is no prophylaxis for this infection and no vaccine is available.

Hepatitis D virus infection

Hepatitis D viruses are defective RNA viruses transmitted through percutaneous, sexual or perinatal routes. This virus infects with hepatitis B virus. Risk groups for hepatitis B virus infections include IV drug users, healthcare workers dealing with blood, haemodialysis patients, male homosexuals, heterosexuals with multiple partners and recipients of blood transfusion. Incubation of the virus is 21-140 days. There is a carrier state for this infection. Prophylaxis is not available but HBV vaccine offers some immunity for susceptible persons.

Hepatitis E virus infection

Hepatitis E viruses are defective RNA viruses transmitted through faecal-oral routes. Risk groups for hepatitis E virus infections include travellers to endemic areas such as India, Asia, Africa and Central America. Incubation of the virus is 15-64 days. There is no carrier state and no prophylaxis available for this infection. Immunity following this infection may last a lifetime.

Clinical features of viral hepatitis

There are no differences in clinical features between the types of viruses involved. Generally patients complain of ‘flu’ like symptoms in the early phase of infection. Three stages of infection are often noticed:

- **Preicteric phase:** This phase is characterised by anorexia, nausea, vomiting, fatigue, myalgia, malaise and fever (1-2 weeks before the onset of jaundice). With Hepatitis B virus infection, 5-10% of sufferers develop arthralgia, rash and angioedema.
- **Icteric phase:** In this phase, clinical features include the appearance of jaundice and right upper quadrant pain, with anorexia, nausea and vomiting. Hepatomegaly and splenomegaly also become palpable. This phase lasts from between 2 and 8 weeks in 20 to 50% of patients.
- **Posticteric phase:** In this phase, symptoms disappear but hepatomegaly persists for some time. Recovery is achieved in four months after the onset of jaundice.

Oral Manifestations and Dental Management Considerations

- Jaundice of the oral mucosa in the icteric phase.
- Identification of carriers of HBV, HCV and HDV is essential. All patients are to be considered potentially infectious. Standard precautions must be used at all times.

- *Patients with active hepatitis*: Consultation immediately with their physician is essential. No dental treatment should be given unless it is urgent. No hepatotoxic drugs are to be prescribed for these patients. Use an isolated operatory and adhere to standard precautions.
- *Patients with a history of hepatitis:* Obtaining a thorough history and adhering to standard precautions during the treatment are essential.
- *Patients at high risk for HBV infection:* Screening for HbsAg is recommended before dental treatment is undertaken. No modifications in dental treatment are necessary
- *Patients who are known hepatitis carriers (HbsAg positive):* Adhere to infection control protocols during the treatment. No modification in dental treatment is necessary.
- *Dentists who are hepatitis virus carriers:* Dentists should adhere strictly to professional ethics and practice guidelines, and to standard precautions in the operatory. Periodically, the dentist should test his/her HBsAg status. Obtaining informed consent from patients is essential. Until seroconversion has occurred, dental practice should be discontinued.

Diagnosis/investigations: History, physical findings and blood tests for liver enzymes (elevated) bilirubin (raised), prothrombin time (elevated), alkaline phosphatase (elevated) and WBCs (increased) are helpful in the diagnosis of the disease.

- **Alcoholic liver disease (ALD)**

Alcoholic liver disease (ALD) refers to liver damage and its function as a result of alcohol abuse.

Classification of ALD

1. Fatty liver: is the mildest form of reversible liver injury.
2. Alcoholic hepatitis: features hepatocellular damage. Jaundice, fever and ascites are common at this stage.
3. Perivenular sclerosis: leads to liver cirrhosis.
4. Cirrhosis: displays fibrosis and nodule formation. Cirrhosis is irreversible and accompanies portal hypertension in most cases.

Symptoms and Signs: Suggestive of ALD, these include oedematous puffy face, traumatic or unexplained injuries and scars, memory deficits, slurred speech, jaundice of sclera and oral mucosa, spider angiomas, ascites, white nails or transverse pale bands on nails, ankle oedema, petechiae, ecchymoses, prolonged bleeding, parotid gland enlargement and a sweet, musty breath odour.

Oral Manifestations and Dental Management Considerations

Jaundice of the oral mucosa, advanced periodontal disease, parotid enlargement, sweet and musty oral malodour, and bleeding tendencies are common in ALD.

Detect ALD by history, clinical examination, alcohol on the breath and obtaining information from family members. Other considerations include:

- Referral or consultation with a physician to check current health status, medications, laboratory values and to discuss management issues.
- Laboratory screening for FBC, AST, ALT, bleeding time (BT), thrombin time (TT) and prothrombin time (PT)
- Avoidance of drugs metabolised by the liver is essential.
- If laboratory values are abnormal for surgical procedures, give consideration to antifibrinolytic agents, fresh frozen plasma, vitamin K, or platelets.
- Alcohol prevention information must be given to patients.
- Patients are to be directed to healthcare providers for rehabilitation.

Diagnosis/Investigations: History and physical examination are suggestive of alcohol abuse.

Laboratory findings will vary depending on the stage of ALD. These include elevations of bilirubin, alkaline phosphatase, aspartate transaminase (also called serum glutamic oxaloacetic transaminase) and alanine transaminase (also called serum glutamic pyruvic transaminase), amylase, uric acid, triglycerides and cholesterol.

Deficiencies in clotting factors lead to elevations in prothrombin time (PT) and partial thromboplastin time (PTT). Thrombocytopenia results in increased bleeding time.

Ultrasounds and CT scans can also be used to assess liver damage. In some cases, a liver biopsy is useful in assessing the extent of liver damage.

Management: Cessation of alcohol use is the main form of management of ALD. This would reverse the damage, or prevent further damage leading to liver cirrhosis. A calorie rich diet, the use of antioxidants, anticytokines, and steroids in severe cases, are used to treat ALD. A liver transplant is the ultimate measure when all other modalities have failed.

- **Liver cirrhosis**

Definition/description: Liver cirrhosis is a consequence of chronic liver disease characterised by replacement of liver tissue by fibrosis, scar tissue and nodules leading to loss of function

Causes: Liver cirrhosis is caused by a host of factors. They include infection (Hepatitis B and C virus infections), metabolic (alcohol, haemochromatosis, Wilson's disease, galactosaemia), immunological (Primary biliary cirrhosis), drugs (methotrexate, methyldopa, isoniazid), veno-occlusive disease, constrictive pericarditis and sarcoidosis).

Symptoms: These include lethargy, itching (because of deposition of bile salt products in the skin), ankle oedema and abdominal oedema.

Signs: Hepatomegaly, splenomegaly, jaundice, spider nevi, palmar erythema, Dupuytren's contracture, finger clubbing, ascites, Terry's nails, reduced body hair,

oesophageal varices, dark urine, caput medusae and bruising are seen in patients with cirrhosis.

Oral manifestations and dental management considerations

- Jaundice of the oral mucosa, bleeding tendencies and poor oral hygiene are common in patients with liver cirrhosis.
- Hepatotoxic drugs are to be avoided in these patients.
- Consultation with a physician is required before the commencement of any invasive procedure.

Diagnosis/Investigations: Liver biopsy is the gold standard for the diagnosis of cirrhosis. Blood tests include the estimation of mean corpuscular volume **(**MCV), serum ferritin, viral serology, auto antibodies, clotting studies, albumin, platelets and bilirubin. Endoscopy is done for oesophageal varices.

Management: Cirrhosis is irreversible. Absolute abstinence from alcohol for alcoholic cirrhosis, and steroids for chronic active hepatitis, are useful in arresting the progress of the disease. Venesection for haemochromatosis may retard the progression. Complications should be treated appropriately. A liver transplant is possible for those with end-stage cirrhosis.

- **Liver cancer (Hepatocellular carcinoma)**

Definition/description: A malignant tumour of the hepatocytes is hepatocellular carcinoma. This is one of the most common tumours worldwide.

Causes: These include chronic HBV or HCV carriage, and cirrhosis from any cause. Hemochromatosis (excess iron absorption and storage, a genetic disorder) is also known to cause liver cancer/

Symptoms and signs: Male to female ratio: 3:1. Symptoms include abdominal pain, weight loss, ascites, fever, jaundice and hepatomegaly. Paraneoplastic signs include hypoglycaemia, erythrocytosis, hypocalcaemia, and ectopic gonadotrophin production.

Oral manifestations: Oral mucosa may show signs of jaundice. Rarely, metastasis from the liver to oral tissues may occur. Consultation with a physician, surgeon or oncologist is required.

Diagnosis/Investigations: History and clinical findings may lead to a provisional diagnosis which needs to be confirmed by liver biopsy. Other tests include the estimation of alpha-fetoprotein (increased in 85% of patients), ultrasounds and CT scan.

Management: Treatment includes resection or liver transplant, chemotherapy and opiates for pain.

Diseases of the gall bladder

- **Cholelithiasis (Gallstones)**

Definition/description: Presence of stones in the gall bladder is referred to as cholelithiasis.

Cause: Low fibre and high cholesterol diets, rapid weight loss, constipation, eating fewer meals per day, and low intake of nutrients such as folate, magnesium, calcium, and vitamin C may be associated with the formation of gallstones. Among drugs, long term use of proton pump inhibitors can potentially lead to gallstone formation.

Symptoms and signs: Gallstones may be asymptomatic (silent gallstones). Symptoms commonly begin to appear once the stones reach 8 mm or more. A characteristic symptom of gallstones is intense pain in the upper right side of the abdomen (a "gallstone attack"), often accompanied by nausea and vomiting. Pain steadily increases for approximately 30 minutes to several hours. Referred pain to a shoulder may also be felt by the patient. Often, attacks occur after a particularly fatty meal and almost always happen at night.

Diagnosis/investigation: History and clinical features, x-rays of the abdomen, ultrasounds, and magnetic resonance imaging are used in the diagnosis of gallstones,

Management: Laparoscopic cholecystectomy is the treatment of choice for symptomatic cholelithiasis.

- **Cholecystitis**

Definition: Cholecystitis is inflammation of the gall bladder. It presents in acute and chronic forms.

Causes: Gallstones cause acute cholecystitis in a majority of cases. In some cases, prolonged starvation, burns, shock and vasculitis are the risk factors for acalculous cholecystitis, a condition which is devoid of gallstones.

Symptoms/signs: These include upper right quadrant pain and tenderness, nausea, vomiting and fever.

Oral manifestations: There are no specific oral manifestations seen in cholecystitis. Taste disturbances may occur in some patients.

Diagnosis/Investigations: History and clinical findings are helpful in arriving at a provisional diagnosis. Abdominal ultrasounds detect the gallstones and inflammation of the gall bladder.

Management: Treatment includes use of antibiotics and cholecystectomy.

Diseases of the Pancreas

- **Pancreatitis**

Definition/Description: Pancreatitis is inflammation of the pancreas. Two forms of pancreatitis exist: acute and chronic.

Causes: In acute pancreatitis, release of the activated pancreatic enzymes cause acute inflammation which may result from biliary tract disease or heavy chronic alcohol intake. In the chronic form of the disease, chronic heavy alcohol intake is the major cause. In some cases it may be idiopathic.

Symptoms and signs: Nausea, vomiting and steady pain in the upper abdomen radiating to the back are common symptoms of the acute form of the disease. Often, pain is less severe in a sitting up or leaning forward position. Coughing, deep breathing and vigorous movements increase the intensity of pain. Rapid but shallow respiration, tachycardia and abdominal tenderness are present. Chronic pancreatitis may be asymptomatic. Malabsorption and steatorrhoea may be present in some patients. When symptomatic, pain is epigastric, severe and may last several hours or days.

Oral manifestations: If malabsorption is a feature of pancreatitis, oral manifestations may include those of dehydration.

Diagnosis/Investigations: Clinical history and clinical features are useful indicators. Since symptoms and signs of pancreatitis mimic some other gastrointestinal and cardiovascular diseases, a broad range of tests are required. Investigations for acute pancreatitis include full blood count, electrolytes, calcium, magnesium, glucose, BUN, creatinine, amylase, lipase, ECG, urine for trypsinogen and abdominal x-rays. Imaging studies such as Endoscopic Retrograde Cholangiopancreatography (ERCP) and secretin pancreatic function tests are useful for chronic pancreatitis.

Management: Treatment for acute pancreatitis includes intravenous fluids, fasting and analgesics (opioids). Dietary modifications, analgesics and enzyme supplements are adequate for chronic pancreatitis. Surgery may be necessary in some cases.

Other Topics of Dental Interest

- **Jaundice**

Definition/description: Jaundice is yellow discolouration of the tissues. This is noticed especially in the skin and sclera**.** Jaundice is due to the deposition of bilirubin and becomes clinically visible when the circulating bilirubin levels exceed 35 µmol/L.

Causes: Jaundice may be due to haemolysis (prehepatic jaundice), liver disease (hepatic jaundice), biliary obstruction or intrahepatic cholestasis (cholestatic jaundice).

Prehepatic jaundice can occur due to congenital or acquired causes: These include:

- Gilbert's disease
- Criggler-Nijjar syndrome
- Hereditary spherocytosis (congenital disorder)
- Sickle cell disease
- G6PD deficiency
- Thalassaemia
- Malaria
- Incompatible blood transfusion
- Haemolytic disease of the newborn
- Autoimmune disorder

- Hypersplenism

Hepatic jaundice can occur due to acute or chronic hepatocellular disease. Causes include:

- Viral hepatitis: hepatitis A, B or C, Epstein-Barr virus and Cytomegalovirus
- Drugs: paracetamol and halothane
- Toxins: carbon tetrachloride
- Autoimmune disorders
- Chronic viral hepatitis
- Chronic hepatitis due to autoimmune disorders
- End stage liver disease due to alcohol, cirrhosis, haemochromatosis and Wilson's disease

Cholestatic jaundice can occur due to intrahepatic and extrahepatic causes. These include:

- Primary biliary cirrhosis
- Drugs such as chlorpromazine
- Viral hepatitis
- Gallstones
- Infestations such as schistosomiasis
- Cholangitis
- Carcinoma of the head of the pancreas
- Carcinoma of the ampulla of Vater (formed by the union of the pancreatic duct and the common bile duct).
- Chronic pancreatitis

Symptoms and signs: Yellow colouration of the skin and sclera is the hallmark of jaundice.

In the newborn, physiological jaundice is common due to immature liver function. This generally settles within the first week of life.

In prehepatic jaundice, a family history of jaundice, history of haematuria and drug history offer some clues to the diagnosis. Often jaundice is mild.

In hepatic jaundice, a history of alcohol abuse, travel, sexual activity, drug history, and blood transfusions offer useful hints. The liver is tender in hepatitis. Signs of liver failure such as spider nevi, palmar erythema, leukonychia, finger clubbing, gynaecomastia, ascites, peripheral oedema, bruising tendencies, Depuytren's contracture, Caput medusae, and haemetemesis may be seen.

In cholestatic jaundice hepatomegaly, palpable gall bladder (in the presence of obstructive jaundice) and epigastric mass (carcinoma of the pancreas, for example), may be seen. A history of severe upper abdominal pain preceding jaundice may be a common symptom. Dark urine and pale stools are also common in this condition.

Oral Manifestations and Dental Management Considerations

- Oral mucosa may show signs of yellow colouration in patients with jaundice.
- In patients with jaundice, bleeding tendencies may be present. In such cases, post-operative haemorrhage due to clotting factor deficiencies need to be identified and managed appropriately.
- Hepatotoxic drugs must be avoided.
- If the jaundice is due to viral hepatitis, the cross infection risk is to be borne in mind and appropriate infection control measures must be invoked.
- In all cases of jaundice regardless of the type, a physician should be consulted before any invasive dental treatment is carried out.

Investigations: The following investigations are recommended for a patient with jaundice:

- Full blood count (FBC) for haemolysis, malignancy and infections,
- Erythrocyte sedimentation rate (ESR) for infections and malignancy,
- Blood film for spherocytosis, reticulocyte count for haemolysis,
- Urea and electrolytes (U&Es) for hepatorenal syndrome,
- Liver function tests (LFTs),
- Clotting screen,
- Viral antibodies for hepatitis,
- Ultrasound for gallstones and dilated biliary tree,
- CT scan for carcinoma of the head of the pancreas,
- Endoscopic retrograde cholangiopancreatography (ERCP) for stones of the common bile duct and periampullary carcinoma, and also for biopsy,
- Urine and blood biochemistry for bilirubin, and
- Blood for alkaline phosphatase and transaminases.

Management: Since jaundice is a symptom, its management includes identification and elimination of its cause.

- **Ascites**

Definition/description: Ascites is the accumulation of excess free fluid in the peritoneal cavity called.

Causes: Several causes are associated with ascites. These include: liver cirrhosis, hepatic tumours, primary or secondary pelvic or abdominal tumours, cardiac failure and constrictive pericarditis, nephrotic syndrome, peritonitis, hepatic portal vein or inferior vena cava obstruction, malabsorption, and pancreatitis.

Symptoms and signs include abdominal discomfort, increasing abdominal girth, weight gain, and ankle swelling. Shortness of breath may also be present due to splinting of the diaphragm by ascites, or to cardiac failure as the underlying cause.

Inspection of patients with ascities may reveal jaundice, spider nevi, palmar erythema, or caput medusae. In such cases liver disease may be the cause. Shifting dullness and fluid thrill are also present on physical examination. Hepatomegaly and splenomegaly are felt on

palpation in patients with ascites. In these patients, portal hypertension and haematological malignancies may be the cause. Patients with ascites showing evidence of cirrhosis, cardiac failure, malabsorption, nephrotic syndrome and lymphatic flow obstruction due to intra-abdominal or pelvic tumours may present pedal oedema as well. In cardiac failure causing ascites, JVP is raised and systolic murmurs are present. Crepitations due to pulmonary oedema may also be present in patients with ascites.

Oral Manifestations and Dental Management Considerations

There are no specific oral manifestations of ascites. Since underlying causes of ascites include diseases of cardiac, hepatic, renal and other systems (cardiac failure, liver disease, nephrotic syndrome and malabsorption, for example), appropriate dental management considerations specific to these conditions must be taken into account before offering dental treatment. Infection control measures need to be strictly followed and appropriate positioning of the dental chair for patient comfort must also be considered. Prior consultation with a physician is essential.

Investigations: These include:

Urine dipsticks for protein in patients with nephrotic syndrome, a full blood count including a differential white cell count for infective aetiology, urea and electrolytes for renal causes, liver function tests for liver disease, chest x-ray for cardiomegaly in heart failure, and ultrasounds for confirmation of ascites and presence of intra-abdominal masses. Aspiration of ascetic fluid (abdominal paracentesis) is also helpful in determining the underlying cause of ascites.

Management of ascites includes the identification and elimination of the underlying cause, and treatment for symptoms.

- **Hepatomegaly**

Definition: Enlargement of the liver is called hepatomegaly.

Causes: There are hepatic and non-hepatic causes associated with hepatomegaly. Some of these include: hepatitis due to viruses, bacteria, parasites, protozoa, alcohol, fatty liver, hepatoma, biliary tract diseases, cardiac failure, constrictive pericarditits, leukaemia, myeloproliferative diseases, metastasis, glycogen storage disease and haemochromatosis.

Symptoms: General malaise, fever, weight loss and jaundice may be present in hepatomegaly due to infections. Upper abdominal pain may be present due to stretching of liver capsule in congestive cardiac failure. Metastasis from a known malignancy from GIT or bronchus is common.

Signs of liver disease (cirrhosis, for example) may be present. In cardiac causes (congestive cardiac failure, for example), peripheral oedema and raised JVP is present. Deep jaundice in biliary tract disease and palpable mass in primary malignancies of the liver can occur. The gall bladder is palpable in patients with carcinoma of the head of the pancreas (Courvoisier's law). Skin pigmentation and portal hypertension is evident in haemochromatosis. Jaundice and ascites are present in malignancy. In addition to

hepatomegaly, lymphadenopathy and splenomegaly may be present in myeloproliferative disorders.

Oral Manifestations and Dental Management Considerations

Oral symptoms and signs of the underlying disease which caused hepatomegaly may be present. A physician must be consulted before commencing dental treatment.

Investigations: Depending on the suspected cause of hepatomegaly, some of the following investigations may be necessary:

A full blood count, erythrocyte sedimentation rate, urea and electrolyte estimation, liver function tests, a clotting screen, hepatitis serology serum iron estimation, bone marrow aspiration, ultrasounds and CT scans of the liver, and liver biopsy.

Management of hepatomegaly includes the identification and elimination of the cause, and appropriate treatment for associated symptoms.

Chapter 8

Disorders of the Cardiovascular System

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Abstract

This chapter deals with diseases of the cardiovascular system. Beginning with common cardiovascular symptoms, the chapter looks at clinical examination methods and investigations employed in assessing the status of the cardiovascular system. This is followed by individual cardiovascular disease entities which include angina, myocardial infarction, heart failure (cardiac failure), cardiac arrhythmias, mitral stenosis, mitral regurgitation, aortic stenosis, aortic regurgitation, atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), coarctation of aorta (COA), tetralogy of Fallot (ToF), rheumatic fever (RF), infective endocarditis (IE) and hypertension. Each disease entity starts with a definition or description followed by its causes, symptoms, signs, investigations employed, and principles of management. Where relevant, oral and dental aspects of cardiovascular diseases are also described.

Introduction

The components of the cardiovascular system are the heart, blood vessels and blood. From a dental management point of view, dental practitioners are expected to have an adequate knowledge of some of the more common manifestations of cardiovascular diseases and their impact on oral health. In this chapter, disorders of the heart and blood vessels, and their oral aspects are briefly discussed.

Common Cardiovascular Symptoms

Chest pain: Chest pain is one of the major symptoms of heart disease. Pain can also originate from lungs, oesophagus and thorax. A practitioner should therefore be able to identify the exact source of chest pain. Chest pain of cardiac origin occurs in angina pectoris,

myocardial infarction, thoracic aortic dissection, pericarditis and myocarditis. Non-cardiac causes of chest pain include tension pneumothorax, oesophageal rupture, pulmonary embolism, pneumonia, pancreatitis, thoracic malignancies, gastroesophageal reflux, peptic ulcers, costochondritis, biliary tract disorders, herpes zoster infection involving the thorax, and chest trauma.

Differential diagnosis of chest pain due to cardiac and other causes:

Quality of the chest pain due to:

- Angina and myocardial infarction, feels tight and crushing.
- Dissection of an aortic aneurism has a tearing quality.
- Pericarditis and pulmonary in origin is sharp and worse on inspiration (pleuritic pain).
- Gastroesophageal reflux disease (GORD) has a burning quality.
- Peptic-acid disorders are deep and of a biting, gnawing or chewing quality.

Location of the Chest Pain

- Pain of IHD and GORD are retrosternal in location and can radiate to the left arm and the jaw.
- Pericarditis pain may radiate to the shoulders,
- Pain due to aortic dissection may radiate to the back.
- Pain of pulmonary origin can be located anywhere in the thorax.

Precipitating Factors

- Angina is precipitated by exercise, emotion, heavy meals or cold weather.
- If pain occurs at rest for more than 30 minutes, it should be considered as a pain of myocardial infarction until proven otherwise.
- Pain of GORD is associated with meals and a change in posture (bending down, for example).
- Pulmonary pain worsens with movements of the thorax.

Relieving Factors

- Both angina and pain due to oesophageal spasm are relieved by glyceryl trinitrate (GTN) due to its action on smooth muscles.
- GORD pain is relieved by antacids. Antacids have no effect on pain of cardiac origin.
- Pericarditic pain is relieved by sitting forwards.

Breathlessness: Breathlessness (short of breath, or dyspnoea) is a normal symptom after heavy exertion, but becomes pathological if it occurs in clinical situations. In congestive heart failure and cardiac ischaemia, breathlessness is common. Non-cardiac causes of breathlessness include asthma, pneumonia, interstitial lung disease, chronic obstructive pulmonary disease, or psychogenic factors.

Palpitations: A patient's perception of cardiac activity as a racing, fluttering or skipping sensation is generally referred to as palpitations. Anxiety, exercise and febrile illness often cause heightened awareness of normal cardiac activity, however, these experiences are not to be regarded as examples of palpitations. Palpitations generally result from cardiac arrhythmia caused by premature atrial or ventricular contractions, and these are harmless. Some clinical conditions which cause arrhythmias include paroxysmal supraventricular tachycardia, atrial fibrillation (flutter), ventricular tachycardia, myocardial ischaemia, valvular heart defects, thyrotoxicosis (which causes increased myocardial contractility), anaemia, hypoxia, and electrolyte imbalances. Coffee, alcohol, epinephrine, and theophylline are known to trigger palpitations.

Postural hypotension: Postural hypotension (orthostatic hypotension) results in faintness, dizziness and blurred vision within a few seconds of standing due to an excessive fall in blood pressure. Some patients may experience syncope. Elderly patients and those with hypertension are vulnerable to orthostatic hypotension. This can also occur due to vagal stimulation after urination or defecation.

Syncope: Syncope is characterised by a brief loss of consciousness with a loss of postural tone followed by spontaneous revival. The patient becomes pale, motionless and hypotensive with a weak pulse, cool extremities and shallow respiration.

Ankle swelling: There are a number of diseases that can cause swelling in the ankles. Right heart failure is one of the major cardiac causes of ankle swelling. Other conditions which cause ankle swelling include varicose veins, venous insufficiency, lymphatic obstruction, surgery of the foot, burns, and insect bites or stings. Hypertension is also believed to be one of the factors that can cause swelling in the feet and ankles. Certain birth control pills, calcium channel blockers (e.g., nifedipine, amlodipine, diltiazem, felodipine, and verapamil) prescribed for lowering blood pressure, steroids and antidepressants such as phenelzine and tranylcypromine may trigger ankle swelling. Ankle oedema during pregnancy is also common. Long periods of standing and long aeroplane flights are also known to cause ankle oedema.

- **Examination of the cardiovascular system**

The major elements of a cardiovascular examination include observation, palpation and auscultation. Percussion of the chest was previously used to determine the size of the heart. This test was found to be unreliable and was abandoned. X-rays are now used to determine the size of the heart.

Methods

Ask the patient to strip to the waist. Female patients can cover up until that part of the chest needs to be examined. Inform the patient of what you are about to do. The patient should rest in a supine position with the upper body elevated at an angle of 30-45 degrees.

Assessment of the pulse and blood pressure are essential elements of the CVS examination. Methods of recording the pulse and blood pressure are described in Chapter 3.

Inspection: Look for tachypnoea (heart failure), malar flush showing a bluish tinge on the cheeks (mitral stenosis), xanthelasma and corneal arcus (hyperlipidaemia), cyanosis (heart failure), forceful carotid pulsations (aortic regurgitation), ankle oedema (heart failure), splinter haemorrhages in the nail bed under the nail (infective endocarditis), clubbing (congenital cyanotic heart disease, respiratory disease), pallor, (anaemia, congenital heart disease), surgical scars on the chest and chest deformities.

Palpation: Palpate the right radial pulse (see Chapter 2). Assess the rate (60-100 beats per minute) and rhythm. Bradycardia is a feature of heart block, beta blocker use and hypothyroidism whereas tachycardia can occur in patients with anxiety, fever, hyperthyroidism and arrhythmia. Is the rhythm irregular or regular (irregularly irregular, as in atrial fibrillation, or regularly irregular as in missing every 4^{th} beat)? Ventricular ectopic beats disappear on exercise. The pulse deficit (atrial fibrillation) is the difference between the auscultated heart rate at the apex and the palpated pulse at the wrist. Palpation of the right and left pulse together is important. The difference can be detected in atherosclerosis and stricture of the axillary artery. Radial and femoral pulse should be recorded to detect if there is any delay, as found in coarctation of the aorta. Carotid pulse should be palpated for its character. Abnormal pulse characteristics can indicate valvular disease. Collapsing pulse is a sign of aortic regurgitation. This is assessed by raising the patient's left arm and with both hands, holding the forearm (one on the wrist and the other lower down) with your fingers on the ulnar side of forearm. If you feel the pulse vibrating back down your arm, it is indicative of collapsing pulse.

In addition to the above, basic cardiovascular system examinations include the following:

1. Jugular venous pressure (JVP) assessment,
2. Inspection and palpation of the chest and precordium and
3. Auscultation of the heart.

Jugular venous pressure (JVP)

Jugular venous pressure (JVP) is the blood pressure in the jugular vein, which reflects the volume and pressure of venous blood. Sometimes this is referred to as jugular venous pulse. JVP has a wave form which correlates to various parts of the cardiac cycle.

In the case of normal JVP (<3 cm), when the patient lies at an angle of 45 degrees, the jugular vein should be visible at the level of the clavicle between the heads of the sternocleidomastoid muscle. When JVP is elevated, the pulsation is seen higher up the neck.

Causes of raised JVP include right heart failure, fluid overload as seen in kidney failure, tricuspid valve regurgitation, obstruction of the superior vena cava and atrial fibrillation.

Method

1. Seat the patient reclining supine at 45 degrees.
2. Ask the patient to look slightly to his/her left.
3. Look just above the clavicle between the heads of the sternocleidomastoid muscle. If there is pulsation visible, it could be JVP (in which case, it is raised). It is important

to distinguish carotid artery pulsations from jugular venous pulsations. JVP has a double waveform. Look at the ear lobe as well, which may also show pulsation in raised JVP.

4. Look for and identify the internal jugular pulsation. Right and left internal jugular veins pass medial to the clavicular heads of the sternocleidomastoid muscles and run behind the angles of the mandible to the ear lobes.
5. In order to clearly identify the internal jugular veins, compress the abdomen. A rise in venous pulsation is seen in the neck on both sides. In many patients JVP may not be seen - this is normal. In heart failure, when the liver is pressed, the JVP increases and stays raised for longer than a few seconds. Arterial pulsations are not affected by pressing on the abdomen.
6. Measure the vertical height in centimetres between the mid-point of the venous pulsation and the sternal angle. A ruler is placed at the upper level of the distended vein parallel to the ground, and another ruler is placed perpendicular to the first ruler up to the sternal angle and the length is measured. This is the jugular venous pressure.

Normal JVP is less than 3 cms. In a healthy individual reclining at 45 degrees, JVP is not (or maybe just) visible above the level of clavicle. Pulsation generally falls during inspiration. A persistent elevation of the JVP is the earliest sign of right ventricular failure.

Examination of the Chest And Precordium

The precordium is the area of the chest directly covering the heart. There are four regions to be considered:

1. Apex/mitral area: around the 5^{th} left intercostal space; the mid-clavicular line.
2. Tricuspid area: around the 4^{th} intercostal space just lateral to the sternum.
3. Pulmonary area: the 2^{nd} left intercostal space, just lateral to the sternum.
4. Aortic area: The 2^{nd} right intercostal space, just lateral to the sternum.

Inspection

The patient is to be positioned semi-recumbent.

Inspect the chest wall for any deformities.

The precordium: look and feel for the apical impulse and abnormal pulsations.

The apex beat may be visible on inspection, usually in the 5^{th} left intercostal space medial to the mid-clavicular line.

- Parasternal movement can be seen in right ventricular hypertrophy.
- A pulsation from an enlarged pulmonary artery can also be seen in the left 2^{nd} intercostal space.
- Pulsation high in the epigastrium is due to the expansion of the abdominal aorta.

Palpation

The precordium is palpated for pulsations and thrills. The apex beat may be palpated by placing your right hand on the left chest of the patient. Start at the 2nd intercostal space and move down to the manubriosternal junction and laterally in relation to the mid-clavicular line. Usually, the apex beat is felt in the 5th intercostal space medial to the mid-clavicular line.

- **Apex beats** are not palpable in nearly 80% of patients. The most common cause of this is a thick chest wall or obesity. Other causes include emphysema and pericardial effusion. A 'tapping' apex beat may be felt in mitral stenosis. Displaced apex beats may be felt in aortic stenosis, mitral and aortic regurgitation, left ventricular dilatation and hypertension, where the apex beat may be slightly displaced downwards and outwards.
- **Thrill:** A palpable vibrating sensation called is thrill. This is feels like a cat's purr, due to the presence of a murmur. It is present in the aortic area in patients with aortic stenosis.
- **Heave:** The apical impulse, when it feels abnormally forceful and strong, is called a heave. Parasternal heave can be felt by placing the palm firmly on the sternum with your fingers pointing upwards. In patients with heave, your fingers will be lifted off the patient's chest.

Auscultation

Use a stethoscope with both a bell (which is better for low pitched sounds) and a diaphragm (used for high pitched sounds). Always feel the carotid pulse during auscultation of the heart. This will give you an idea of the part of the cardiac cycle (systole or diastole) in which the murmur occurs.

There are normally two heart sounds: the first heart sound (S1) and the second heart sound (S2).

Assess their intensity and splitting character. Also auscultate the interval between the heart sounds for additional sounds.

- **The first heart sound** (due to the closure of the mitral and tricuspid valves) is best heard at the apex. This signals the start of a systole. Auscultate the apex (using the bell) in the left lateral position.
- **The second heart sound** (due to the closure of the aortic and pulmonary valves) signals the start of the diastole. (Generally diastole is longer than systole. The carotid pulse will be present between S1 and S2 during systole). The second heart sound can be best heard at the left sternal edge in the second intercostal space.
- The first heart sound (S1) is generally lower pitched than the second heart sound (S2). S1 is the 'lubb' and S2 is the 'dup'.
- Sometimes splitting of the second heart sound (S2) can occur in inspiration (due to delayed closure of the pulmonary valve).

Altered heart sounds

Loud S1: In mitral stenosis, the mitral valve shuts rapidly causing a louder sound.

Soft S1: When the mitral valve does not close fully, S1 is softer in mitral regurgitation.

Soft S2: Due to reduced aortic valve movement in aortic stenosis, S2 is softer.

In patients with prosthetic valves, the prosthetic heart sounds are often audible without the aid of a stethoscope.

Added heart sounds: These can be heard as third and fourth heart sounds, an opening snap and pericardial friction.

- The third heart sound is heard at the apex during early ventricular filling in children and young adults.
- The fourth heart sound is heard preceding the first heart sound and is coincidental with atrial contraction.
- An opening snap of the mitral valve (soon after the second sound) is heard in mitral stenosis.
- Pericardial friction (a creaking and rustling noise) is heard in pericarditis. It is best heard when the diaphragm is pressed against the chest.

Murmurs result from turbulent blood flow which can occur in a diseased valve or due to the flow of large amounts of blood through a normal valve. Murmurs can be systolic or diastolic. Systolic murmurs are present in mitral regurgitation and aortic stenosis. Diastolic murmurs are audible in aortic regurgitation and mitral stenosis. Mitral systolic murmurs (in mitral regurgitation) are best heard at the apex whereas aortic systolic murmurs (in aortic stenosis) are heard at the second intercostal space (aortic area). In mitral stenosis, diastolic murmurs can be heard at the mitral area with the patient lying on his/her left side. Diastolic murmur of the aortic regurgitation can be heard at the left sternal edge, with the patient sitting up and holding their breath at the end of expiration. Ensure that the stethoscope's diaphragm is pressed firmly against the skin.

Examination of the Peripheral Arterie

Detection of the peripheral pulse is an important aspect of cardiovascular examination.

Dorsalis pedis pulse should be detected using the finger tips placed over the artery immediately proximal to the first metatarsal space.

Posterior tibial pulse can be felt behind the malleolus.

The femoral pulse can be detected at the mid-inguinal point

Popliteal pulse can be detected in the popliteal fossa with the knee slightly flexed.

Bruits can be detected using a stethoscope. Femoral, (abdominal) aortic and internal carotid arteries are sites of major artery bruits. Bruits are signs of turbulent blood flow.

Common Investigations in Cardiology

Common investigations in cardiology include chest x-rays, electrocardiograms (ECGs), exercise ECGs, ambulatory ECGs and BP monitoring. Echocardiography and angiography are also used in detecting coronary artery disease and other disorders involving the heart muscles and valves.

Chest x-ray: In cardiology, an x-ray of the chest is useful in identifying cardiomegaly.

Electrocardiogram (ECG): The record obtained from the depolarisation and repolarisation voltages of the heart muscle is called an electrocardiogram or ECG. An ECG is used to measure the rate and regularity of heartbeats as well as the size and position of the chambers, the presence of any damage to the heart, and the effects of drugs or devices used to regulate the heart, such as a pacemaker. Electrical activity starts in the sinoatrial node, passes across the atria (detected as the P wave) then travels across the atrioventricular node before causing ventricular depolarisation.

Ambulatory electrocardiography (AECG): An ambulatory electrocardiogram records the electrical activity of the heart during usual activities. The most common type is the continuous recorder (such as the Holter monitor) which provides a 24- to 72-hour record of the electrical signals from the heart. Ambulatory electrocardiography is used to detect, characterise and document cardiac arrhythmias.

Echocardiography (cardiac ultrasound scanning): This is useful in assessing ventricular function and valvular abnormalities. Echocardiography uses sound waves to create images of the heart. It provides information about the size and shape of the heart, and the level of function of its chambers and valves. It can detect blood clots inside the heart, pericardial fluid build-up, and problems with the aorta. A type of echocardiography called Doppler ultrasound provides information on the blood flow through the chambers and valves of the heart.

Angiography: Angiography is an imaging technique used to visualise the lumen of arteries, veins and the heart chambers. This is done by injecting a radio-opaque contrast agent into the blood vessels and imaging using x-ray based techniques such as fluoroscopy. One of the most common angiograms performed is to visualise the blood in the coronary arteries (coronary angiography). Coronary angiography is performed to outline coronary circulation in atheromatous disease prior to angioplasty, and isotope scans are carried out for ventricular function and myocardial infarction.

Estimation of cardiac enzymes: These include creatinine kinase, transaminases and lactate dehydrogenase. These enzymes are useful in evaluating the status of cardiac disease. Troponin levels provide useful information on cardiac muscle damage, as seen in myocardial infarction.

Cardiovascular Diseases of Dental Interest

Ischaemic Heart Disease

- **Angina (Angina pectoris)**

Definition/Description: Angina, also called angina pectoris, is a common symptom characterised by pain of cardiac origin as a result of ischaemic heart disease (IHD). IHD causes an imbalance between the myocardial oxygen supply and demand.

Causes: A reduction of the coronary arterial luminal diameter (by 70-90%) due to ischaemic heart disease (IHD) causes angina. Non-IHD diseases that can cause angina include coronary artery spasm (Prinzmetal's angina), aortic stenosis, and cardiomyopathy. Major risk factors for IHD include cigarette smoking, hypertension, male gender, family history,

diabetes mellitus, and hyperlipidaemia. High blood levels of low density lipoprotein (LDL) cholesterol and low levels of high density lipoprotein (HDL) cholesterol are associated with IHD.

Symptoms and signs: These include severe central chest pain (gripping, constricting, crushing or tightness) often with shortness of breath, faintness and pain radiating to the left (and sometimes right) arm, and into the neck and jaw. Pain is typically induced by exercise, emotion, a heavy meal and cold weather, and is relieved by rest and nitrates. This is called **stable angina**. If angina is of recent onset, severe and rapidly worsening on minimal or no exertion, and lasts longer than a few minutes, it is considered as **unstable angina**. This is usually a forerunner of myocardial infarction (MI). The pain of myocardial infarction is of similar character and site to stable angina, but is more prolonged, more severe, and accompanied by nausea, vomiting, breathlessness, sweating, and abnormal heart rate and rhythm. This pain is not relieved by nitrates. Signs of angina are sometimes absent. Risk factors such as hypertension may be identified in a majority of cases.

Oral Manifestations and Dental Management Considerations

- During the attack, the patient may feel acute pain in the jaw.
- Patients with stable angina can be treated with LA containing 2% lidocaine and adrenaline 1:80,000. Good practice is to take GTN before the commencement of treatment.
- If a patient with history of angina experiences chest pain during dental procedures:
  - Treatment must be stopped immediately and the patient must be seated upright, given GTN (sublingual) and administered oxygen. Vital signs should be monitored.
  - Pain should be relieved within 3-4 minutes. **If pain persists (a symptom of MI), medical help must be summoned.**
  - Patient should be given 300 mg aspirin to chew and the administration of oxygen continued.

Investigations: Electrocardiogram (ECG). This reading is abnormal during an attack, and often normal at rest. If the ECG has been recorded as normal during rest (in a patient with suspected angina), a treadmill exercise ECG or radionuclide scanning is usually considered. ECG and BP are monitored during the recovery period after exercise. Coronary angiography is considered for those who may require angioplasty or bypass surgery.

A full blood count (FBC) and erythrocyte sedimentation rate (ESR) are required to exclude non-atheromatous causes of angina.

Management: Recognition and correction of risk factors such as hypertension, smoking, obesity, diabetes and hyperlipidaemia are the key factors in the management of angina.

Drug therapy for angina includes aspirin (75 mg daily), nitrates (isosorbide mononitrate 20 mg bd), β-blockers (atenolol 50 mg daily), and calcium channel antagonists (nifedipine 10 mg tid or amlodipine 5-20 mg/day, or ATP-sensitive potassium channel activators (nicorandil 10-20 mg bd).

Surgery includes coronary angioplasty revascularisation (with stents) for proximal arterial stenosis, and coronary artery bypass surgery for triple coronary artery disease.

- **Myocardial infarction (MI)**

Definition/description: Death (necrosis) of a part of the heart muscle due to total occlusion of the coronary artery results in myocardial infarction.

Cause: Embolism following rupture of atheromatous coronary artery plaque is the major cause of myocardial infarction. MI is common in the winter months, and may be precipitated by vigorous exercise, major surgery or infections.

Symptoms: These include severe, crushing, central chest pain, often radiating to the neck, left arm and mandible, and not relieved by nitrates. Pain is usually associated with nausea, sweating, breathlessness and vomiting. In some cases symptoms do not occur and infarction is discovered incidentally when an ECG is performed at a later date. In this situation the infarct is called a **silent infarct.** Sudden death is due to ventricular fibrillation in 50% of heart attack patients.

Signs: Common signs of myocardial infarction include pallor, circulatory shock, tachycardia, low blood pressure, cyanosis and gallop rhythm. Gallop rhythm usually refers to the abnormal rhythm of the heart on auscultation. It includes three or four sounds, thus resembling the sound of a galloping horse.

Oral Manifestations and Dental Management Considerations

- During the attack, patients may feel acute pain in the jaw
- Dental intervention in patients with myocardial infarction can precipitate arrhythmias. For those patients who have had a recent MI attack (in the last 6 months), elective surgical procedures are to be deferred. Simple emergency procedures under LA may be undertaken in consultation with the cardiologist.
- Patients who have had recent angioplasty, or a Coronary Artery Bypass Graft (CABG)
  - Elective procedures need to be modified. LA with adrenaline/epinephrine is contraindicated for CABG patients, as arrhythmias may be precipitated by these agents. Emergency procedures need to be carried out in a hospital setting.
  - Consultation with the cardiologist or cardiac surgeon is essential.

Investigations: These include abnormal ECG findings and plasma cardiac enzymes such as creatine kinase, transaminases and lactate dehydrogenase (elevated). The troponin level is also elevated in MI. Chest x-rays are used to identify pulmonary oedema, and aortic dissections are required.

Management: Therapeutic management of MI includes aspirin (300 mg soluble) as soon as possible and opiates (diamorphine 2.5 mg IV with an antiemetic (metoclopramide 10 mg IV). Immediate transfer of the patient to ICU is required. ECG recording, bed rest with high

flow oxygen administration, thrombolytic therapy (subcutaneous heparin 5000 IU eight-hourly) and monitoring blood cardiac enzymes are other essential measures.

Complications: Tachyarrhythmias, bradyarrhythmias, cardiogenic shock, pulmonary oedema, and pericarditis may occur as complications.

Prevention of Ischaemic Heart Disease (IHD):

The risk factors for IHD need to be targeted in a prevention strategy, and these include smoking, hypercholesterolemia, hypertension, obesity, alcohol abuse and diabetes. Contraceptive pills, cold climates, a Type A personality and socio-economic factors also have a role in the causation of IHDs.

Heart failure (cardiac failure)

Definition/description: Heart failure is a clinical syndrome characterised by a change in the pumping function of the heart accompanied by typical symptoms such as shortness of breath or weakness.

Cause: These include ischaemic heart disease, hypertension, valvular heart disease, arrhythmias, pulmonary embolism, anaemia, thyrotoxicosis, myocarditis, cardiomyopathy, infective endocarditis, and thiamine deficiency.

Symptoms: Dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, ankle swelling, fatigue and lethargy are common symptoms of cardiac failure.

Signs: These include ankle and sacral oedema, jugular venous distension, basal crepitations, hepatomegaly and gallop rhythm.

Investigations: Chest x-ray, ECG, echocardiography, and radionuclide imaging are used in the diagnosis of heart failure. Blood tests such as a complete blood count, blood creatinine, blood glucose, albumin, liver function tests, and thyroid function tests are also necessary.

Management: Management includes dietary salt restriction, diuretics, digitalis preparations (digoxin), vasodilators, β-blockers, and ACE inhibitors. If thyrotoxicosis is the cause, treatment is directed to correct it.

Cardiac arrhythmias

Definition/description: Loss of rhythm resulting in irregularity of the heartbeat is called arrhythmia. Cardiac arrhythmias are divided in to two categories: (1) supraventricular and ventricular arrhythmias, and (2) bradyarrhythmias and tachyarrhythmias.

Cardiac arrhythmias include isolated ectopic beats, bradycardia and tachycardia. Cardiac arrest can occur as a result of arrhythmias.

Causes: Cardiac arrhythmias are associated with a range of diseases of cardiac and non-cardiac origin. These include ischaemic heart disease, myocardial infarction, rheumatic heart disease, congestive heart failure, pneumonia, obstructive lung disease, thyrotoxicosis, systemic infections, drug-related side effects, and electrolytic imbalances.

Symptoms and signs: Cardiac arrhythmias can cause palpitations and collapse. Irregularly irregular pulse and rapid atrial rhythm can be considered as signs of atrial fibrillation. The rate at the apex is faster than that at the radial artery.

A sinus rate greater than 100 beats per minute is considered as tachycardia. A sinus rate of less than 60 bpm is considered to be bradycardia.

Valvular Heart Disease

There are four valves in the heart: two atrioventricular valves and two semilunar valves. Atrioventricular (AV) valves are located between the atria and the ventricle. These are the mitral valve and the tricuspid valve. The two semilunar (SL) valves, which are located in the arteries leaving the heart, are the aortic valve and the pulmonic (pulmonary) valve. Valvular heart diseases can involve any of these valves. Commonly these disorders include mitral stenosis, mitral regurgitation, aortic stenosis and aortic regurgitation.

- **Mitral stenosis**

Definition/description: The mitral valve (also known as the bicuspid valve) separates the upper and lower chambers on the left side of the heart. Mitral stenosis is a condition in which the valve does not open fully, thus restricting blood flow.

Cause: In the majority of cases, rheumatic fever is the cause of mitral stenosis. Marked thickening of the leaflets (the flaps of the bicuspid or tricuspid valves), or occasionally calcifications around the orifice of the mitral valve, may also cause stenosis.

Symptoms: Breathlessness, cough, palpitations and haemoptysis are common symptoms in mitral stenosis.

Signs: These include malar flush, atrial fibrillation, peripheral embolism, a tapping apex beat, a loud first heart sound, opening snap and a low-pitched diastolic murmur.

Investigations: Chest x-rays, ECGs, echocardiography, and cardiac catheterisation are performed in evaluating mitral stenosis.

Management: Medical treatment includes the use of diuretics, digoxin, and warfarin. Definitive treatments of mitral stenosis include mitral valvotomy, balloon valvuloplasty or valve replacement.

- **Mitral regurgitation**

Definition/description: Mitral regurgitation is a disorder in which the mitral valve does not close properly, resulting in the backwards flow of blood into the atrium when the heart contracts.

Cause: These include myxomatous degeneration, rheumatic heart disease, or mitral valve prolapse due to post-MI.

Symptoms: Breathlessness, fatigue and palpitations are commonly experienced by patients with mitral regurgitation.

Signs: Large apical murmurs radiating towards the axillae, and large pulse pressure are common in mitral regurgitation.

Investigations: These include chest x-rays, ECGs, Doppler studies, or cardiac catheterisation.

Management: Valve replacement or repair becomes necessary in severe cases while control of arrhythmias is required in milder cases.

- **Aortic stenosis**

Definition/description: The aortic valve guards the opening between the left ventricle and the aorta. The aortic valve opens as the left ventricle begins to pump, allowing blood to eject out of the heart and into the aorta. When the ventricle has finished beating, the aortic valve closes to keep blood from washing back into the left ventricle. In aortic stenosis, the aortic valve becomes partially obstructed, leading to significant heart problems.

Cause: These include congenital and rheumatic heart disease, calcification of the congenital bicuspid valve in younger age groups, and degenerative valves in the elderly.

Symptoms: Dyspnoea, angina and syncope are commonly encountered symptoms. Sometimes sudden death occurs in these patients.

Signs: Small pulse pressure and ejection murmur are features of aortic stenosis.

Investigations: These include ECG, chest x-rays, echocardiography, and Doppler echocardiography measurements.

Management: Cardiac failure and angina need to be treated. Valve replacement or balloon valvuloplasty, are other options available. Patients with aortic stenosis require antibiotic prophylaxis.

- **Aortic regurgitation**

Definition/description: Aortic regurgitation is the abnormal retrograde flow of blood through the aortic valve during cardiac diastole.

Causes: These include congenital bicuspid valve defects due to fusion of the leaflets, endocarditis, rheumatic heart disease, connective tissue disease and aortic dissection.

Symptoms and signs: Patients with aortic regurgitation may be asymptomatic. Symptomatic patients may complain of dyspnoea, palpitations and angina.

Signs: These include a collapsing pulse, wide pulse pressure, cardiac apex displacement, early blowing diastolic murmur and systolic flow murmur.

Investigations: These include ECGs, chest x-rays, echocardiography, Doppler studies and cardiac catheterisation.

Management: Treatment of cardiac failure, valve replacement and antibiotic prophylaxis are management options for aortic regurgitation.

Oral Manifestations and Oral Relevance of Valvular Disorders

There are no specific oral manifestations of the mitral or aortic valve disorders. For any invasive dental treatment, however, a clearance from the cardiologist must be sought to establish the patient's ability to withstand the procedures and stress. In patients with aortic stenosis, dyspnoea, angina and syncope may occur during dental procedures. The dental practitioner should be equipped with the knowledge and skills to manage these patients should such emergencies occur.

Congenital Heart Disease

Definition/description: Congenital heart disease refers to congenital malformations of the heart involving the heart chambers, heart valves or major blood vessels.

Cause: There are two types of congenital heart diseases: Acyanotic (non-cyanotic) and cyanotic congenital heart defects. Acyanotic defects include atrial septal defect (ASD), ventricular septal defect, (VSD), patent ductus arteriosus (PDA), and coarctation of the aorta. The cyanotic form of congenital heart disease includes tetralogy of Fallot, transposition of great vessels, tricuspid atresia, and pulmonary atresia.

- **Atrial septal defect (ASD)**

Definition/description: Atrial septal defect is characterised by a defect in the interatrial septum allowing pulmonary venous return from the left atrium to pass directly to the right atrium. Often this condition is not suspected until adulthood.

Symptoms and signs: These include palpitations and breathlessness, and the presence of tricuspid diastolic murmurs.

Investigations: Tests include chest x-rays, ECGs, echocardiography, and Doppler studies.

Management: Treatment of pulmonary hypertension, and where indicated, surgical repair are available treatment options.

- **Ventricular septal defect (VSD)**

Definition/description: Ventricular septal defect (VSD) is a hole in the wall between the right and left ventricles of the heart. This abnormality usually develops before birth and is found most often in infants.

Symptoms/signs: These include breathlessness, fatigue, systolic thrill, a loud systolic murmur and a diastolic mitral flow murmur.

Diagnosis/Investigations: Chest x-rays, ECGs, echocardiography, and Doppler studies are used in the diagnosis of VSD.

Management: Surgery is the treatment of choice.

- **Patent ductus arteriosus (PDA)**

Definition/description: Patent ductus arteriosus (PDA) refers to the persistence of the foetal connection (ductus arteriosus) between the aorta and pulmonary artery after birth. This results in left to right shunt. The ductus arteriosus is a normal connection between the pulmonary artery and the aorta which is necessary for foetal circulation. At birth, usually within the first 10-15 hours of life, this connection closes due to the decline in prostaglandin. If it does not close, PDA occurs.

Cause: This is a congenital heart anomaly.

Symptoms and signs: PDA is common among premature children. Infants with small PDA are asymptomatic. With large PDA, symptoms include tachypnoea, dyspnoea with

feeding, and failure to thrive. A continuous murmur is common at the upper left sternal border.

Diagnosis/investigations: History and clinical examination provide clues to the diagnosis. X-rays of the chest, ECGs and echocardiography with colour flow and Doppler studies, or with CT scans or magnetic resonance angiography (MRA) confirm the clinical diagnosis.

Management: Administration of indomethacin (a prostaglandin synthesis inhibitor) is sometimes effective in closing the PDA in premature infants. In full term infants, indomethacin is ineffective. Surgical or catheter-based corrections are the treatments of choice. Endocarditis prophylaxis is recommended before, and for between 6 and 12 months after, correction.

- **Coarctation of the aorta**

Definition/description: Coarctation of the aorta is a congenital anomaly of the heart which refers to the localised narrowing of the aortic lumen.

Cause: This condition is a congenital heart anomaly.

Symptoms and signs: Significant coarctation of aorta results in circulatory shock, with renal insufficiency and metabolic acidosis during the neonatal period. In children with less severe coarctation, symptoms include headaches, chest pain, and fatigue and leg claudication during physical activity. Signs include hypertension in the upper extremities, diminished femoral pulses, and low blood pressure in the lower extremities. Left ventricular hypertrophy and malperfusion of the abdominal organs are the other features encountered in coarctation of the aorta. A soft bruit may be heard over the coarctation site.

Diagnosis/investigations: Diagnosis is arrived at by clinical examination, chest x-rays, ECGs, and 2-dimensional echocardiography with colour flow and Doppler studies, or with CT scans or MR angiography.

Management: Treatment is balloon angioplasty with stent placement, or surgical correction. Endocarditis prophylaxis is recommended.

- **Tetralogy of Fallot**

Definition/description: A congenital heart disorder in children characterised by pulmonary stenosis, ventricular septal defect, dextral position of the aorta and ventricular hypertrophy causing cyanosis (‘blue baby’ syndrome) and other serious clinical features.

Cause: This is a congenital defect.

Symptoms and signs: These include dyspnoea (due to the large amount of deoxygenated blood shunted into the systemic circulation), weakness, clubbing of fingers, faintness, dizziness, syncope and coma. Blood tests show polycythemia.

Oral Manifestations and Dental Management Considerations

- Delayed tooth eruption, frequent positional anomalies of teeth, hypoplastic enamel, more dental caries and an increase in the occurrence of periodontal disease are encountered in patients with congenital heart disease.

- Prevention of infective endocarditis should be the main focus while planning dental procedures. Prophylactic antibiotic cover may be needed for patients with congenital heart defects. In this regard, the patient's physician or cardiologist should be consulted prior to commencing invasive procedures.
- Patients with tetralogy of Fallot are susceptible to infective bacterial endocarditis. Dental procedures may need to be carried out with antibiotic cover.
- A physician or surgeon should be consulted.

Diagnosis/Investigations: These include chest x-rays, echocardiography (definitive), CT scans and MRI.

Management: Surgical repair is the treatment of choice. Prognosis is usually poor.

Rheumatic Fever (RF)

Definition /description: Rheumatic fever is an acute inflammatory disease of the joints and heart caused by an autoimmune disorder which is preceded by a streptococcal infection of the throat. In RF, heart valves can be damaged and become vulnerable to infective endocarditis.

Cause: β-haemolytic streptococcal infection of the throat results in producing cross-reacting antibodies which then attack various normal body tissues including the heart valves, joints and skin. Rheumatic nodules called Aschoff's nodules can occur on heart valves (the mitral valve in particular) causing valve incompetency.

Symptoms and signs: These include fever, polyarthritis, pericarditic pain, heart murmurs and heart failure.

Diagnosis/Investigations: These include microbiological confirmation of the causative organism via a throat swab. Other investigations include detection of the antistreptolysin antibody, ECGs, and ESR.

Management: Bed rest, analgesia and appropriate antibiotics are recommended.

Infective Endocarditis

Definition/description: Bacterial infection of the cardiac valves or endocardium is called infective endocarditis. This condition affects several organs and three types of infective endocarditis are known:

1. Subacute infective endocarditis,
2. Acute (or fulminant) endocarditis and
3. Right-sided endocarditis.

1. Subacute infective endocarditis

Cause: Causative organisms include *Streptococcus viridians, Strep. faecalis*, staphylococci and coliforms.

Symptoms and signs: Subacute infective endocarditis is of insidious onset. Symptoms include pyrexia, night sweats, fatigue, joint pains and haematuria. Signs include retinal infarcts, Osler's nodes, changing murmur, and splenomegaly. The valves at most risk of abnormalities are the bicuspid aortic valves, rheumatic valves, and prosthetic valves.

2. Acute endocarditis:

Cause: The causative organism is *Staphylococcus aureus*.

Symptoms and signs: These include pyrexia, retinal haemorrhages, petechiae, and peripheral emboli.

3. Right-sided endocarditis

Symptoms and signs: These include pyrexia, fatigue, breathlessness and pleuritic chest pain. The tricuspid valve is involved. Intravenous drug users are prone to be affected.

Investigations for all three types of infective endocarditis: These include blood cultures, transoeasophageal echocardiography for vegetations, full blood count (leucocytosis), and ESR (elevated).

Management and prevention: Antibiotics after the organism has been identified in the culture. Prevention strategies include antibiotic prophylaxis for patients with abnormal or prosthetic valves or VSDs before receiving procedures (such as dental extraction) that are likely to produce bacteraemia. Recommended prophylaxis includes 3 gm oral amoxicillin one hour before, and for penicillin –sensitive patients, erythromycin 1.5 gm one hour before the procedure, followed by 0.5 gm six hours later.

Oral Manifestations and Dental Management Considerations

Many clinicians believe that patients with Rheumatic fever (RF) may have an increased risk of developing infective endocarditis following invasive dental treatment. They recommend that these patients are given antibiotic prophylaxis prior to invasive dental procedures such as tooth extraction, implant placement, subgingival probing and scaling, endodontic treatment, placement of matrix and orthodontic bands, and intraligamentary local anaesthetic injections.

Recommended prophylaxis includes 3gm oral amoxicillin 1 hour before, and for penicillin–sensitive patients, erythromycin 1.5gm one hour before the procedure followed by 0.5gm six hours later. In some countries prophylactic treatment is not recommended.

This is a controversial issue! In the UK, antibiotic prophylaxis is not recommended for patients at risk of endocarditis undergoing dental procedures. [4]

In many other countries there are national guidelines on the use of antimicrobial prophylaxis against infective endocarditis. It is to be noted, however, that patients at risk of developing infective endocarditis should receive intensive preventive oral care in order to minimise the need for dental intervention.

- **Hypertension**

Definition/description: Hypertension is characterised by the sustained elevation of resting systolic blood pressure (BP ≥140 mmHg), diastolic blood pressure (BP ≥90 mmHg) or both. Two types of hypertension are known: primary (85-95% of cases) and secondary hypertension.

Cause: Although in the majority of cases no obvious cause can be found, obesity, dietary salt, stress, and hereditary factors are frequently associated with hypertension. This is called **essential or primary hypertension**.

Secondary hypertension, on the other hand, is associated with glomerulonephritis, pyelonephritis, Cushing's syndrome, primary aldosteronism, hyperthyroidism, myxoedema, coarctation of the aorta, alcohol abuse, contraceptive pills, pregnancy and drugs such as prednisolone.

Symptoms and signs: The majority of patients with hypertension are asymptomatic. Occasionally, patients may complain of headache, dizziness, flushed face, fatigue, nose bleeds, and nervousness.

Complications may include cardiac failure, myocardial infarction, renal failure and retinal symptoms and signs. High BP is a consistent sign in these situations.

'White coat hypertension' is to be considered for those whose BP is elevated when measured in the physician's office, but is normal when measured at home or by ambulatory BP monitoring.

Oral Manifestations and Dental Management Considerations

- There are no specific oral manifestations of hypertension.
- The practitioner should be aware of oral side-effects of antihypertensive drugs. These include xerostomia, gingival hyperplasia (with nifedipine), salivary gland swelling (with clonidine), and increased post-operative bleeding (for those patients on aspirin).
- Patients have an increased risk of cardiovascular disease such as angina and MI.
- There are no dental treatment restrictions (including the use of vasoconstrictors) for those patients with mild to moderate but controlled hypertension. For those with severe hypertension, only emergency dental procedures can be undertaken. Consultation with the physician is necessary.
- Stressful situations should be avoided. Vasoconstrictors in LA need to be avoided for patients with severe hypertension (180/100 mmHg and above). Referral to a GP or hospital for severe and uncontrolled hypertension is essential.
- Drugs taken for angina or hypertension may cause oral adverse effects. An example is calcium channel blockers, as these cause mucosal lichenoid reactions and gingival swellings.

Diagnosis/investigations: Physical examination includes measurements such as weight, height, waist circumference, fundoscopic examination for retinopathy, auscultation of the neck for bruits, and a full cardiac, respiratory and neurological examination. If the blood pressure is severe and diagnosed for the first time in a young patient, testing for target organ

damage should be carried out. Tests include urinalysis, spot urine albumin/creatinine ratio, lipid profile, blood glucose estimation, and ECGs.

Management: Primary hypertension has no cure! Causes of secondary hypertension can be corrected. Treatment is aimed at reducing BP lower than 140/90 mmHg for those without signs of target organ disorders, and 130/80 for those with renal disease or diabetes.

Life style modifications include regular physical exercise (30 minutes a day most days of the week), weight loss down to a body mass index between 18.5 and 24.9, smoking cessation, a diet rich in fruits and vegetables and low-fat dairy products, a low salt intake (NaCl less than 6 g/day), and alcohol less than 30 ml/day for men and 15 ml/day for women. These life style modifications may be adequate for those who have no signs of target organ manifestations of hypertension. No medications are required as long as their BP is controlled.

Antihypertensive drugs are required for patients whose BP remains above 140/90 after 6 months of life style modifications.

Antihypertensive drugs include diuretics, β-blockers, calcium channel blockers, ACE inhibitors, and angiotensin-II receptor blockers.

Chapter 9

Diseases of the Respiratory System

S. R. Prabhu

Abstract

This chapter deals with respiratory diseases of dental interest. Starting with common symptoms of the respiratory disease, the chapter presents essential details of the clinical examination and investigations employed in identifying respiratory diseases. This is followed by respiratory diseases of dental interest which include the common cold, sinusitis, laryngitis, influenza, bronchitis, asthma, chronic obstructive pulmonary disease (COPD), pneumonia, lung abscess, bronchiectasis, pulmonary embolism, pulmonary tuberculosis, lung cancer, obstructive sleep apnoea (OSA) and cystic fibrosis. Other related disorders associated with respiratory disease discussed in this chapter include pleural effusion and pneumothorax. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of respiratory diseases are also described.

Introduction

Respiratory diseases are common. Some respiratory diseases are chronic in nature and require treatment for a long time. Patients with chronic respiratory diseases such as asthma require special considerations in the management of dental conditions. It is essential therefore that practicing dentists should possess a basic knowledge of respiratory diseases in order to take appropriate steps in managing dental procedures.

Common Symptoms of Respiratory Diseases

Cough: A cough is an explosive expiratory act that is reflexively or deliberately intended to clear the airways. In most instances, the presence of mucus or foreign material in the

airway causes coughing. A persistent cough is an indication of chronic irritation of the pulmonary airway.

Coughs may be acute or chronic. Acute cough is a feature of the common cold. Other causes include pneumonia, postnasal drip and exacerbations of chronic pulmonary obstructive disease (COPD). Pulmonary embolism and cardiac failure in the elderly can also cause acute episodes of coughing. A chronic productive cough is common in smokers as a result of chronic bronchitis. Other common causes of chronic cough include postnasal drip syndrome, gastroeosophageal reflux disease, asthma, and use of ACE inhibitors. Chronic cough with haemoptysis may be a feature of tuberculosis or bronchogenic carcinoma. In bronchiectasis and lung abscess, chronic cough with purulent sputum is intense. The green or yellow colour of the sputum is suggestive of pyogenic infection. Dry spasmodic coughing associated with wheezing is a sign of asthma.

Dyspnoea: Dyspnoea is characterised by shortness of breath or breathlessness.

Pulmonary causes of dyspnoea include pneumothorax, pulmonary embolism, bronchospasm due to asthma, foreign body and toxic inhalation, acute bronchitis, pneumonia, obstructive lung disease and pleural effusion. Cardiac causes of dyspnoea include acute myocardial ischaemia or infarction, ventricular dysfunction and cardiogenic pulmonary oedema. Other causes include anxiety disorders and anaemia. Anxiety related dyspnoea is called hyperventilation syndrome.

Chest pain: Pulmonary and pleural causes of chest pain include pneumonia, pulmonary embolism, pleuritis, lung cancer and rib fractures. Cardiac causes of chest pain include angina, myocardial infarction, acute aortic dissection and pericarditis. Gastric causes of chest pain include gastroesophageal reflux disease, peptic ulcer disease and oesophageal spasm. Herpes zoster (involving thoracic dermatome) and emotional factors (depression, for example) also cause chest pain.

Haemoptysis: Haemoptysis is the coughing up of blood or blood stained sputum.

Respiratory causes of haemoptysis include bronchial carcinoma, pulmonary tuberculosis, pulmonary embolism, chronic bronchitis, pneumonia, brochiectasis, pulmonary oedema and Wagener's granulomatosis. Other causes include mitral stenosis and clotting disorders.

Wheezing: Wheezing results from a narrowing of the airways. Causes include asthma, COPD, heart failure, vocal cord dysfunction and anaphylaxis.

Stridor: Stridor is a high pitched inspiratory sound formed by extrathoracic upper airway obstruction. Causes of stridor include foreign body aspiration, epiglottitis, croup, vocal cord dysfunction, laryngeal tumours, allergic reactions and retropharyngeal abscesses.

Examination of the Respiratory System

General Observation

Look for evidence of respiratory distress such as breathlessness, wheezing, cough and exhalation with pursed lips. Also look for signs of cyanosis, finger clubbing, and tobacco staining on the fingers. The radial pulse should be recorded. Tachycardia suggests significant respiratory distress.

Examination of the face: Look for signs of anaemia (pale conjunctivas), central cyanosis (bluish tinge of the tongue or lips) and Cushingoid appearance as a result of long-

term steroid use. Neck examination may indicate an increased jugular venous pressure (right heart failure), goitre, or lymph node enlargement.

Physical Examination of the Chest

Methods include inspection, palpation, percussion and auscultation.

Inspection of the chest: With patient sitting or standing ask the patient to fully expose the chest and upper abdomen. Look for:

- **Chest wall abnormalities:** kyphosis (abnormal anterior–posterior curvature of the spine), scoliosis (abnormal lateral curvature of the spine), barrel chest (chest wall increased anterior-posteriorly), pectum excavatum (sternum sunken in to the chest), pectus carinatum (sternum protruding from the chest, also known as pigeon chest), scars, and skin lesions need to be noted.
- Check for: respiration frequency (normal adult 14 breaths per minute), respiratory depth (hyper- or hypo-ventilation), maximum chest expansion **(**measuring the inspiratory and expiratory difference; > 4 cm is normal), and mode of breathing, either thoracic or abdominal (in normal subjects it is mainly abdominal).
- Look for the nature of the breathing: 1. Kassmaul's breathing is deep and laboured, often associated with severe metabolic acidosis. 2. Cheyne-Stokes' breathing is progressively deep followed by temporary apnoea (heart failure, or carbon monoxide poisoning).
- Abnormal inspiratory movements can occur in cases of fractured ribs and sternum, whereas an abnormal expiratory movement can occur in asthma and acute bronchitis.

Palpation of the Chest Wall

- Position of the trachea: insert the tip of the index finger into the suprasternal notch to detect any deviation of the trachea**.** In pneumothorax, the trachea is deviated away, as it is also in the presence of masses and enlarged lymph nodes.
- Chest expansion: should be symmetrical, and normally 4-5 cm. Symmetrical reduction occurs, for instance, in bronchial asthma, emphysema, and stiff lungs (pulmonary fibrosis).
- Feel for vibrations: place both palms on the posterior lung fields, and ask the patient to count from one to ten. Feel for vibrations and compare the right and left lung fields. Increased vibration can be felt in pneumonia. Decreased vibration is a feature of pleural effusion. A "crackling" sensation on palpation is an indication of subcutaneous emphysema. Sometimes, pleural rub is also palpable.
- Test for tectile fremitus, the vibration felt by a hand placed on the chest during vocal fremitus. Ask the patient to say "*ninety nine"* and use the ulnar aspect of the palm to feel changes in sound conduction. Tactile fremitus is increased over the areas of lung consolidation.

Percussion of the Chest Wall

- The physician attempts to examine changes in the density of lung fields by noting their resonance. Steps include:

- Placing the left hand on the patient's chest wall, palm downwards and fingers slightly apart so that the second phalanx of the middle finger is precisely over the area to be percussed.
- Pressing the finger firmly against the chest wall along an intercostal space.
- Performing percussion with the middle finger of the free hand striking the middle phalanx of the middle finger of the stationary hand.
- Performing this action symmetrically on all lung fields, and on both anterior and posterior chest walls.
- Comparing both sides of the chest.

The percussion note over a normal lung is resonant. The percussion note is dull when the lung is separated from the chest wall by pleural fluid (stony, dull note), or in cases of pulmonary consolidation or collapse. The percussion note is tympanic (hyper resonance) in emphysematous lungs, and in pneumothorax.

Auscultation for Respiratory Sounds

Auscultation can detect respiratory sounds produced by vibrations of the vocal cords caused by the passage of air through the larynx during inspiration and expiration. These sounds are transmitted along the trachea and bronchi through the lungs to the chest wall. Respiratory (breath) sounds may be normal, vesicular (diminished or prolonged with expiration), or bronchial.

- Place the stethoscope over each of the five lobes of the lungs and on the front and back of the chest. Ask the patient to take deep breaths in and out with their mouth open. Auscultate over the apices and the axillae as well.
- **Normal breath sounds** (also called vesicular breath sounds) are quiet and gentle without any gaps between the inspiratory and expiratory phase sounds.
- **Bronchial breath sounds** are heard in pneumonic consolidation of the lung and also in pulmonary fibrosis and cavitation.
- **Added breath sounds** of importance include rhonchi (wheezes), crepitations (crackles) and pleural rub (friction).

Rhonchi are musical sounds of high, medium or low pitch. In asthma, they are of high or medium pitch and audible on expiration. In bronchitis, rhonchi are audible during inspiration and expiration, and are of medium or low pitch.

Crepitations are non-musical sounds with a crackling quality audible during inspiration. Excess secretions (due to oedema or viscid exudate) in small airways can cause these sounds. These may disappear after prolonged coughing.

Pleural rub is a creaking sound produced by the movement of the visceral over parietal pleura when both surfaces have been roughened by fibrinous exudates. These are heard at the end of inspiration and just after the beginning of expiration. It can be best heard when the patient takes a deep breath.

Common Investigations in Respiratory Diseases

Common investigations in respiratory disease include the chest x-ray, sputum microscopy, sputum culture, flow rate and lung volume measurements, arterial blood gas analysis, spirometry, pulmonary gas exchange, pulse oximetry, bronchoscopy, ventilation and perfusion scans, thoracentesis, pleural biopsy, CT scans, positron emission tomography scans (PET), magnetic resonance imaging (MRI) and ultrasonography.

Plain chest x-rays are most useful in identifying abnormalities in the heart, lung parenchyma, chest wall, pleurae, diaphragm, mediastinum and hilum. Usually, a chest x-ray is the initial test performed to evaluate the lungs.

Sputum microscopy and serology are useful for the diagnosis of infections of the lungs.

Flow rate and lung volume measurements are used to differentiate obstructive from restrictive pulmonary disorders, to characterise disease severity and to measure responses to therapy. Obstructive lung disorder is characterised by a decrease in the flow rate, whereas restrictive lung disorder is a reduction in lung volume.

Arterial blood gas exchange analysis is useful in assessing accurate measures of PaO2, PaCO2 and blood pH. These also measure carboxyhaemoglobin and methaemoglobin levels.

Spirometry: Spirometry is the most common of the pulmonary function tests (PFTs), measuring lung function. It is an important tool used for generating pneumotachographs, which are helpful in assessing conditions such as asthma, pulmonary fibrosis, cystic fibrosis, and COPD.

Pulmonary gas exchange: This refers to the process by which oxygen is extracted from inhaled air into the bloodstream, and, at the same time, carbon dioxide is eliminated from the blood and exhaled.

Pulse oximetry: Transcutaneous pulse oximetry estimates O_2 saturation (spO_2) of capillary blood based on the absorption of light from a light-emitting diode positioned in a finger clip or adhesive strip probe. Results may be less accurate in patients wearing nail polish; and patients with arrhythmias, hypotension, or profound systemic vasoconstriction, in whom the amplitude of the signal may be dampened. Also, pulse oximetry is able to detect only oxyhaemoglobin or reduced haemoglobin, but not carboxyhaemoglobin or methaemoglobin.

Bronchoscopy: Bronchoscopy is a technique of visualising the inside of the airways for diagnostic and therapeutic purposes. A bronchoscope is inserted into the airways, usually through the nose or mouth, or occasionally through a tracheostomy. This allows the practitioner to examine the patient's airways for abnormalities such as foreign bodies, bleeding, tumours, or inflammation. Specimens may be taken from inside the lungs. The construction of bronchoscopes ranges from rigid metal tubes with attached lighting devices to flexible optical fibre instruments with video equipment.

A Ventilation and perfusion scan, also called a v/q lung scan, is a type of medical imaging using scintigraphy and medical isotopes to evaluate the circulation of air and blood within a patient's lungs in order to determine the ventilation/perfusion ratio. The ventilation part of the test looks at the ability of air to reach all parts of the lungs, while the perfusion part evaluates how well blood circulates within the lungs. This test is performed in cases of COPD and pneumonia.

Thoracentesis: Thoracentesis is a chest wall puncture for the aspiration of pleural fluid. It is used to determine the cause of pleural effusion (diagnostic thoracentesis) and also used to relieve dyspnoea caused by pleural fluid (therapeutic thoracentesis).

Pleural biopsy: A pleural biopsy is performed to determine the underlying cause of an exudative pleural effusion when repeated thoracenteses are non-diagnostic.

CT scan: CT scans of the chest define intrathoracic structures and abnormalities more clearly than do chest x-rays. CT angiography using a bolus of intravenous contrast is employed to highlight pulmonary arteries, useful in the diagnosis of pulmonary embolism.

Positron emission tomography scanning (PET scans): PET scanning uses radioactively labeled glucose to measure metabolic activity in tissues. In pulmonary medicine, PET is useful for determining if a tumour is present in the mediastinal lymph nodes or lung nodes (metabolic staging).

Magnetic resonance imaging: MRI is not extensively used in pulmonary medicine. It is useful in patients with pulmonary embolism in whom IV contrast cannot be used.

Ultrasonography: Ultrasonography is generally used to facilitate procedures such as thoracentesis and central venous catheter insertion.

Respiratory Diseases of Dental Interest

- **Common cold (Acute coryza)**

Definition/description: The common cold is an upper respiratory tract viral infection.

Cause: Viruses include rhino-, corona-, entero- and adenoviruses and respiratory syncytial virus.

Symptoms and signs: sneezing, dry sore throat, headache and rhinorrhoea. Colds are usually self-limiting. Rarely, complications include otitis media, sinusitis and pneumonia.

Management: No treatment is required.

- **Sinusitis**

Definition/Description: Sinusitis (particularly an acute episode) is due to bacterial infection which often follows an upper respiratory tract viral infection.

Cause: *Haemophilus influenzae*, *Strep. pneumoniae* and *Strep. pyogenes.*

Symptoms and signs: pain and tenderness over the involved sinuses, headache and mild fever.

Investigation: Diagnosis is usually on clinical grounds. A sinus x-ray may reveal sinus mucosal thickening or fluid levels.

Management: Antibiotics, nasal vasoconstrictors and analgesics/antipyretics. If not treated, it may lead to chronic sinusitis, or occasionally to meningitis or venous sinus thrombosis.

- **Laryngitis**

Definition/description: Inflammation of the larynx as a complication of the common cold.

Causes include viral or bacterial agents.

Symptoms and signs include non-productive cough, hoarse voice and sore throat.

Investigations: No investigations are required.

Management: Laryngitis is usually self-limiting. Based on the severity, symptomatic treatment and antibiotics may be necessary for some patients. Repeated attacks may lead to chronic laryngitis.

- **Influenza**

Definition/description: An upper respiratory tract viral infection which often spreads widely, resulting in epidemics.

Cause: Myxoviruses and influenza viruses A and B are the causative agents.

Investigations: None required

Symptoms and signs include headache, fever, myalgia and anorexia.

Management: Influenza is self-limiting. Symptomatic treatments include paracetamol for adults. Vaccination in autumn is recommended (a detailed description is given in Chapter 5).

- **Bronchitis**

Definition/description: Bronchitis is a post-viral bacterial infection of the bronchus.

Cause: These include infection with pneumococcus, *H. influenzae* or *Strep. aureus.* Predisposing factors include cigarette smoking, damp, or dusty conditions.

Symptoms and signs: These include cough, retrosternal pain, wheeze, rhonchi, coarse crepitations and productive sputum.

Investigations: Chest x-ray and the culture of organisms from the sputum.

Management: Antibiotics and cough linctus.

- **Asthma**

Definition/description: Asthma is characterised by bronchial inflammation leading to bronchial constriction, oedema and mucous plugging.

Cause: Sensitivity of airways to antigens such as house-dust mite, animal dander and pollens are considered as extrinsic factors. Atopy with raised IgE levels, asthma gene (chromosome 11) and bronchial hyper-reactivity are intrinsic factors. Environmental and life style factors include exposure to cold weather, stress, exercise, use of β-blockers, smoking, environmental pollution, viral infections of the upper respiratory tract and aspirin.

Symptoms and signs include expiratory wheeze, breathlessness, cough (especially in children), chest hyperinflation, tachypnoea, prolonged expiration and audible expiratory wheezing. In severe cases, tachycardia, restlessness, pulsus paradoxus (an abnormally large

decrease in systolic blood pressure and pulse wave amplitude during inspiration), and cyanosis are present. A silent chest and coma may occur in very severe attacks.

Dental Management Considerations for Asthma

- Advise asthma patients to bring their inhalers.
- Routine dental procedures can be carried out under local anaesthesia in asthma patients.
- Non-steroidal anti-inflammatory drugs may precipitate acute attacks of asthma**.** If exacerbations occur, treatment should be discontinued.
- Steroid inhalers may cause oral candidal infections on the palate. Patients should be advised to use spacers and rinse their mouth after using a steroid inhaler.
- Patients should be advised to use their inhalers before each dental treatment session.
- Those on steroids do not generally need a steroid cover during dental treatment under LA.
- Reduce stress.
- Antihistamines should be avoided, since they may cause dryness of the mucosa which may precipitate the formation of tenacious mucous in the event of an asthmatic attack.
- Patients taking theophylline preparations should not be given erythromycin since it may cause toxic levels of theophyllin.
- If an acute attack of asthma occurs in the dental clinic, subcutaneous injection of epinephrine (0.3 to 0.5 ml, 1:1000) should be given and oxygen should be administered.

Investigations: These include chest x-rays, pulmonary function tests, full blood count for eosinophil count, sputum examination for eosinophilia and for casts of small airways, arterial blood gas estimation, and hypersensitivity skin tests for identifying atopic individuals.

Management: Bronchodilators (salbutamol inhalers and steroid inhalers, such as beclometasone) using spacers or dry powder preparations, long acting β-agonist (salmeterol) inhalers, and theophylline are effective.

- **Chronic Obstructive Pulmonary Disease (COPD)**

Definition/description: A chronic, slowly progressive respiratory disorder characterised by airflow obstruction resulting, in most cases, in irreversible lung function impairment. Pathological changes occur in large airways (chronic bronchitis), small airways (bronchiolitis) and lung parenchyma (emphysema).

Causes: Cigarette smoking and exposure to coal dust are the major causes of COPD. In patients with subclinical lung disease, an acute episode of infections may precipitate respiratory failure. Low birth weight and poor socioeconomic status may predispose individuals to COPD.

Symptoms and signs: Productive cough, breathlessness, cyanosis, peripheral oedema, coarse precipitations, cor pulmonale, respiratory failure and polycythemia may develop in patients with COPD. These patients are known as ‘blue bloaters’. Those COPD patients who

develop breathlessness with signs of tachypnoea (rapid breathing), chest hyperinflation and reduced breath sounds are known as 'pink puffers'.

Oral Manifestations and Dental Management Considerations in COPD

- Blue bloaters may show signs of cyanosis of the lips or intra-oral mucosal structures.
- Patients with low risk of COPD can receive dental treatment with minor modifications.
- Medical consultation is required for those with moderate or high risk of COPD.
- Early morning or early afternoon appointments are recommended.
- Diazepam and midazolam are contraindicated as they may act as respiratory depressants.
- Patients need to be seated upright during treatment (to avoid dyspnoea)

Investigations: These include chest x-rays, spirometry, sputum microscopy and culture.

Management: Cessation of smoking, use of bronchodilators, antibiotics, inhaled corticosteroids, flu vaccination, pulmonary rehabilitation and domiciliary oxygen are used in the management of COPD.

- **Pneumonia**

Definition/description: Infection of the parenchyma of the lung is due to microorganisms with a special affinity for pulmonary tissues (primary pneumonia) or due to aspiration (secondary pneumonia). Based on the structures involved, pneumonia can be classified as lobar pneumonia, bronchopneumonia and interstitial pneumonia. Based on the mode of acquisition, pneumonia is referred to as community acquired, hospital acquired or nursing home acquired.

Causes: Microorganisms responsible for pneumonia include *Streptococcus pneumoniae, Strep. aureus, Strep. pyogenes, Legionella pneumophilia, Pneumocystis carini* and influenza viruses A and B.

Predisposing factors for secondary pneumonia include chronic illness, anaesthesia, vomiting, gastroesophageal reflux disease, drugs that depress respiration and immunocompromised patients.

Symptoms and signs: These include productive cough (rusty sputum), rigors, breathlessness, chest pain, haemoptysis, pyrexia, dull percussive notes, crepitations, bronchial breathing and tachypnoea.

Oral manifestations: There are no specific oral manifestations in pneumonias.

Investigations: Chest x-rays (segmental or lobar consolidation), blood examination for leukocytosis, sputum microscopy and culture studies are routinely carried out.

Management: Methods of management include antibiotics (after the collection of blood and sputum for culture tests), administration of oxygen, and rehydration. Pneumococcal vaccination is recommended for those prone to lung infections.

- **Lung abscess**

Definition: Lung abscesses are a necrotising infection of the lung characterised by localised collections of pus.

Cause: Most lung abscesses develop after aspiration of oral secretions by patients with gingivitis or poor oral hygiene. These patients generally have impaired consciousness or are obtunded from alcohol, illicit drugs, anaesthesia, sedatives, or opioids. The most common pathogens involved are anaerobic bacteria. Less frequently, mixed (aerobic and anaerobic) bacteria are also involved in the causation of lung abscess.

Symptoms and signs: Symptoms of lung abscess include fever, productive cough, sweats and loss of weight. Sputum may be purulent or blood streaked with a foul smell and taste. Signs include decreased breath sounds, raised body temperature, crackles over the affected area, egophony (an increased resonance of voice sounds heard during auscultation of the lungs), and dullness to percussion in the presence of effusion.

Oral manifestations: There are no specific oral manifestations in lung abscess. Signs of periodontal disease are common.

Diagnosis and investigations: History, physical examination and chest x-rays are important diagnostic steps. Chest x-rays show consolidation with a single cavity containing an air-fluid level in portions of the lung that are dependant when the patient is recumbent. CT scans are useful but not used as a routine test. Culture methods are useful, but anaerobic organisms are hard to culture. Rarely, bronchoscopy is indicated to rule out malignancy.

Management: Usually, antibiotics such as clyndamycin or a combination of beta-lactam /beta lactamase inhibitors (ampicillin/sulbactum, for example) are effective. Metronidazole with penicillin is also useful. Treatment is carried out until x-rays show complete resolution. For large lesions, surgery (lobectomy) may be necessary.

- **Bronchiectasis**

Definition: Abnormal permanent dilatation and destruction of larger bronchi due to infection and inflammation is referred to as bronchiectasis.

Cause: Cystic fibrosis, immune defects and infections can be causes of bronchiectasis.

Symptoms and signs: Symptoms include chronic cough, haemoptysis, and large volumes of thick, tenacious and purulent sputum. Signs include abnormal breath sounds and finger clubbing.

Oral manifestations: There are no specific oral manifestations in bronchiectasis, however, halitosis is a common feature.

Investigations: Chest x-rays show scattered irregular radiopacities (caused by mucous plugs), honeycombing and "tram lines". A CT scan is also useful in the diagnosis of bronchiectasis. Pulmonary function tests are recommended to document baseline function and for following the progression of the disease over time. Microscopic and cultural studies of sputum can establish the role of causative organisms.

Management: Management of bronchiectasis includes treatment and prevention of acute exacerbations with antibiotics, drainage of secretions and management of complications. Identified underlying causes should also be treated**.**

- **Pulmonary embolism**

Definition: Pulmonary embolism is the occlusion of one or more arteries by thrombi which originate elsewhere in the body, typically in the large veins of the lower extremities or pelvis (deep vein thrombosis, or DVT).

Cause: Venous thromboembolism, air embolism, septic embolism, amniotic fluid embolism, tumour embolism, fat embolism or foreign body embolism are the causes of pulmonary embolism. Thromboembolism due to DVT is the major cause.

Predisposing factors include cigarette smoking, atrial fibrillation, heart failure, trauma to the extremities, hypercoagulability disorders, immobilisation, indwelling venous catheters, malignancy, pregnancy and postpartum sickle cell anaemia, and obesity.

Symptoms and signs: Small pulmonary emboli do not cause symptoms. Larger emboli cause acute dyspnoea, pleuritic chest pain, cough and haemoptysis. Massive pulmonary embolism causes hypotension, tachycardia, syncope and cardiac arrest. Signs include tachycardia and tachypnoea, hypotension, wheezing and crackles.

Oral manifestations: There are no specific oral manifestations in pulmonary embolism.

Diagnosis/Investigations: Diagnosis is challenging because symptoms and signs are often non-specific. Initial investigations include pulse oximetry, ECG, and chest x-rays. Pulse oximetry is helpful in revealing hypoxemia; ECG reveals tachycardia and chest x-rays may show atelectasis (collapse or closure of the lung, resulting in reduced or absent gas exchange), focal infiltrates, an elevated hemidiaphragm, and pleural effusion. V/Q scanning (detects the area of the lung that is ventilated), duplex ultrasonography of the lower extremities to detect femoral vein DVT, arterial blood gas analysis, pulmonary angiography, CT scanning, and echocardiography to detect right ventricular dysfunction are also carried out.

Management: Hospitalisation and immediate administration of oxygen (for hypoxemia), intravenous normal saline and vasopressors (for hypotension) are required. Intravenous thrombolytic therapy with streptokinase and heparin is the mainstay of treatment.

- **Pulmonary tuberculosis**

Definition: A chronic infectious disease caused by *Mycobacterium tuberculosis.*

Cause and pathogenesis: *Mycobacterium tuberculosis,* an acid fast bacillus, can gain entry to the body via the skin, respiratory tract or gastrointestinal tract. Individuals at high risk include those with poor standards of living, are HIV positive, and alcoholics. In the majority of cases, the primary infection is via the lungs as a result of droplet infection.

A primary focus of infection is formed in the lung. This is called the Ghon focus. A caseous involvement of mediastinal lymph nodes occurs. The Ghon focus and the caseous involvement of the lymph nodes are collectively called a ‘primary complex’. The primary complex heals and calcifies in a large majority of cases and the individual remains asymptomatic. If the healing is not complete, progressive tuberculosis may result. Haematogenous spread of infection, called miliary tuberculosis, can occur involving several other organs including the bone marrow, lungs, kidney, joints, heart and brain.

Symptoms and Signs

Constitutional findings include weight loss, tiredness, night sweats, loss of appetite and fever.

Pulmonary involvement causes productive cough, haemoptysis, breathlessness, hoarseness, and chest pain of pleuritic type. Crepitations in the apical area are common. The trachea is pulled to one side, rendering the sternocleidomastoid muscle prominent (Trail's sign). Drooping of the shoulder to the affected side is present. On percussion, impaired dullness on the affected side is apparent. Pleural effusion may also be present.

Oral Manifestations and Dental Management Considerations in Pulmonary TB

Primary tuberculosis infection of the oral soft tissues is extremely rare, however, secondary involvement of oral tissues can occur. These conditions include the appearance of painless tuberculous ulcers on the dorsum or lateral borders of the tongue. These ulcers are chronic and do not respond to routine antibiotics. The edges of tuberculous ulcers are undermined.

Cervical lymphadenopathy is present.

Maxillary or mandibular involvement may result in tuberculous osteomyelitis.

Salivary gland infection can also occur in rare cases.

Biopsy of the lesions stained with Ziehl-Neelsen reagents show typical features of tuberculosis with giant cells, macrophages and acid fast bacilli.

Elective dental care should be deferred in patients with active tuberculosis. Emergency treatment can be given in a hospital setting under strict infection control protocols. Dental personnel should adhere to all infection control measures.

Investigations include chest x-rays (to show non-homogeneous opacity in the upper lobe), chest tomography, CT scans, bacteriological examination (Ziehl-Neelsen stain) and culture studies of the sputum, laryngeal secretions and gastric aspirate, and blood examination for ESR (raised), and FBC and differential count (lymphocytosis).

Mantoux test: An intradermal injection of one tuberculin unit of purified protein derivative (PPD) is given on the forearm using a tuberculin syringe. A positive test shows an indurated area at the injection site of more than 10 mm after 72 hours. A positive test in adults means that the individual has been exposed to TB in the past (but not suffering from the disease at the time of the test). A negative test rules out TB. It is customary that all TB patients are also tested for HIV status and vice versa.

Management: Anti-tuberculosis chemotherapy with rifampicin (also known as rifampin), isoniazid and streptomycin is commonly used. Drug resistance and adverse drug reactions are common. Surgical treatment on the lung may be required for cavities and severe haemoptysis.

Prevention of infection is through BCG vaccination.

- **Lung cancer**

Definition/description: Lung cancer (carcinoma) is a malignant lung neoplasm. This is categorised into two forms: small cell and non-small cell carcinomas.

Causes: Worldwide, cigarette smoking is the major cause of lung cancer of both types. Environmental risk factors among non-smokers include exposure to radon in uranium miners, asbestos in construction workers, silica in miners, arsenic in copper smelting plants, pesticide manufacturing workers and nickel in battery and stainless steel manufacturing plants. Susceptibility to lung cancer may be higher in COPD and pulmonary fibrosis patients. A combination of smoking and exposure to other risk factors puts the individual at greater risk of developing lung cancer.

Symptoms and signs: In early stages, lung cancer may be asymptomatic. As cancer advances, symptoms and sign include cough, dyspnoea, chest pain, haemoptysis, pleural effusion, hoarseness of voice (due to tumour encroachment on the recurrent laryngeal nerve), diaphragmatic paralysis (due to involvement of the phrenic nerve), supine breathlessness, facial and upper extremity oedema, Horner's syndrome (characterised by ptosis, myosis, enophthalmos, and anhydrosis), and symptoms related to the metastasis of the tumour.

Investigations: These include chest x-rays, CT scans, CT guided needle biopsy, sputum and pleural fluid cytology, and bronchoscopy.

Management: Treatment includes surgery, chemotherapy and/or radiation depending on the tumour type. Smoking cessation is an effective preventive measure.

Oral Manifestations and Dental Management Considerations

- Before chemotherapy or radiotherapy is administered, dental caries control measures need to be undertaken.
- Xerostomia and mucositis are common in those on radiation therapy or chemotherapy. Symptomatic treatment of xerostomia includes the use of commercially available artificial saliva substitutes, and for mucositis a lidocaine, Benadryl and Maalox rinse is recommended. An antifungal rinse is also advised to prevent fungal infections.
- Maintenance of good oral hygiene is important.
- Myelosuppression caused by chemotherapy and radiation therapy may result in pancytopenia with increased bruising tendencies and susceptibility to infections. For this reason, dental invasive procedures within the first two weeks of chemotherapy and radiotherapy are contraindicated. Treatment during weeks 3 and 4 is preferred when the blood count begins to rise.
- Frequent consultation with the oncologist is necessary in order to determine the treatment schedule. Obtaining platelet count and the absolute neutrophil count prior to invasive procedures is necessary.

- **Obstructive Sleep Apnoea (OSA)**

Definition: A condition characterised by repetitive pauses in breathing during sleep.

Cause: OSA is caused by obstruction of the upper airway. Old age, brain injury, decreased muscle tone (due to drugs or alcohol), increased soft tissue around the airway (sometimes due to obesity), and structural features that give rise to a narrowed airway. In Down syndrome and in those with enlarged tonsils, OSA may be common.

Symptoms and signs: In individuals with OSA, pauses in breathing typically last 20 to 40 seconds. OSA is usually accompanied by snoring, and is associated with a reduction in blood oxygen saturation. Unexplained daytime sleepiness and restless sleep are other features of OSA. Obesity is common among OSA sufferers.

Investigations/Diagnosis: Diagnosis of OSA is often based on a combination of patient history and tests (lab- or home-based). Polysomnography and home oximetry are used as diagnostic tools.

Management: Physical intervention, which includes 1. Continuous positive airway pressure (CPAP) in which a computer-controlled air flow generator delivers an airstream at a constant pressure; 2. Variable positive airway pressure (VPAP); 3. Automatic positive airway pressure (APAP); and 4. A mandibular advancement splint (MAS) is also useful. In rare cases, tracheostomy is an effective surgical treatment for OSA. Adenoidectomy and tonsillectomy have been tried with variable success.

- **Cystic fibrosis (CF)**

Definition/description: Cystic fibrosis (CF) is an inherited disorder of the exocrine glands affecting primarily the gastrointestinal and respiratory systems. It leads to COPD, exocrine pancreatic insufficiency and abnormally high sweat electrolytes.

Cause: CF is a genetic disorder.

Symptoms and signs: Chronic pulmonary disease leads to periodic exacerbations of lung infections, inflammations and a decline in respiratory function. The majority of patients present in infancy with symptoms. Cough and wheezing are common. A barrel-chest deformity, digital clubbing and cyanosis are common as the disease progresses. In adults, pneumothorax, haemoptysis and right heart failure may occur as complications of the disease. Pancreatic insufficiency is common in children, characterised by bulky foul-smelling stools, poor growth and abdominal protuberance. Due to pancreatic insufficiency, malnutrition is a common feature of CF.

Excessive sweating in hot weather (or with fever) may lead to episodes of hypotonic dehydration and circulatory failure.

Oral Manifestations and Dental Management Considerations

Disorders of the salivary glands in CF can give rise to xerostomia. Gingivitis and swelling of the lips are reported in these patients. In CF, altered amounts of calcium and phosphates in saliva are present. This may promote higher calculus formation and enamel defects.

Dental management in CF patients does not generally demand special considerations. The oral cavity should be examined for candidal infection. This may occur due to inhaled or systemic steroid therapy and xerostomia. A double dose of steroids may be necessary for invasive procedures in some patients. The patient's physician should be consulted prior to any surgical procedures.

Diagnosis: Clinical features and sweat tests are suggestive of the disorder. Identification of two known CF mutations is confirmatory. In addition, measurement of higher electrolyte levels in sweat, particularly chloride, is diagnostic. CF is diagnosed in infancy or childhood.

Management: An interdisciplinary team approach to the management of CF patients is necessary. Control of pulmonary infections, maintenance of adequate nutrition, steroid therapy and symptomatic treatment of other symptoms are required. Chest physical therapy and postural drainage several times each day need to be carried out to loosen mucus and to make it easier to expectorate. Dietary measures include high protein and calories, and vitamins. Lung transplantation may be necessary for some patients.

Other Related Topics

- **Pleural effusion**

Definition: A pleural effusion is an abnormal buildup of fluid between the layers of tissue that line the lungs and chest cavity.

There are two types: transudative and exudative pleural effusions.

- Transudative pleural effusion is caused by fluid leaking into the pleural space. This is caused by increased pressure in the blood vessels or a low blood protein count. Congestive heart failure is the most common cause.
- Exudative effusion is caused by blocked blood or lymph vessels, inflammation, lung injury, and tumours.

Symptoms and Signs

Sometimes there are no symptoms. Chest pain is usually sharp and is worse with cough or deep breaths. Cough, fever, hiccoughs, rapid breathing, and shortness of breath are other associated features.

Investigations: The following tests may help to confirm a diagnosis:

Chest x-ray, chest CT scan, kidney and liver function tests, pleural fluid analysis to identify bacteria, the amount of protein, or the presence of cancer cells, thoracentesis (a sample of fluid is removed with a needle inserted between the ribs) and ultrasound of the chest and heart.

Management Treatment depends on the underlying cause of the pleural effusion. Therapeutic aspiration may be sufficient in most cases. Larger effusions may require insertion of an intercostal drain. Repeated effusions may require chemical (talc, bleomycin, or tetracycline/doxycycline) or surgical pleurodesis. This is a procedure in which the two pleural surfaces are scarred to each other so that no fluid can accumulate between them.

- **Pneumothorax**

Definition/Description: Air in the pleural spaces, causing partial or complete lung collapse, is called pneumothorax.

Cause: Pneumothorax can occur spontaneously, from an underlying pulmonary disease, trauma, or from medical procedures. Respiratory causes include COPD, the HIV-related fungus *Pneumocystis jirovecii*, and cystic fibrosis. Traumatic causes include blunt injuries to the chest. Transthoracic needle aspiration, thoracentesis and CPR may cause iatrogenic pneumothorax.

Symptoms and signs: These include dyspnoea, anxiety and chest pain.

Diagnosis/investigations: Chest x-rays reveal radiolucent air in the absence of radiographic signs of any lung disease.

Management: Oxygen administration to accelerate reabsorption of pleural air, catheter drainage and thoracostomy are used in the management of pneumothorax.

Chapter 10

Diseases of the Renal System

S. R. Prabhu

Abstract

This chapter deals with renal diseases. Beginning with common symptoms and investigations used in nephrology, the chapter discusses individual renal diseases and disorders such as renal calculi, urinary tract infections, nephritic syndrome and renal failure. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of renal diseases are also described.

Introduction

Renal diseases are common. Infections, autoimmune disorders, diabetes, hypertension and other diseases can cause kidney damage. These patients require special dental management. Some common kidney diseases are briefly discussed in this chapter.

Common Symptoms of Renal Disease

Common symptoms of kidney disease may include any of the following:

Nocturia, loin pain, polyurea, oliguria, fatigue, mild fever, incontinence, chills, altered mental states, peripheral neuropathy, nausea, vomiting, anorexia, pruritus, oedema, hypertension and anaemia.

Examination of the Kidneys

Kidneys are physically examined using a bimanual palpation technique. This aspect is described in Chapter 6, which deals with gastrointestinal disorders.

Common Investigations in Renal Disease

Common investigations for renal diseases include the following:

- **Urinalysis -** This test is done by stick testing for proteins, blood, glucose, ketones and pH.
- **Microscopy** for bacteria, white blood cells and casts.
- **Biochemistry** for creatinine, urea, calcium, urate and glucose.
- **Autoantibodies** (ANCA, ANF) can also be detected in the renal involvement of vasculitis.
- **Ultrasounds** are carried out for kidney size and to detect any obstructions.
- **X-rays** for size, shape and position of the kidneys and suspected kidney stones.
- **Pyelography**.
- **Radionuclide scans** are done to asses asymmetrical kidney function.
- **Kidney biopsies** are done for final diagnosis.
- **Renal angiography** is carried out for renovascular disease

Renal Diseases of Dental Interest

- **Urinary tract infections (UTIs)**

Definition/description: A common bacterial infection of the urinary tract which sometimes can involve the kidneys (pyelonephritis), bladder (cystitis) or prostate (prostitis). Fifty percent of women are infected with urinary tract infections and become symptomatic at some time during their lives.

Cause: Bacteria involved include *E. coli, Enterobacter spp, Klebsiella spp, Proteus spp, Pseudomonas aeroginosa, Staph spp*, enterococci, streptococcus groups B, D, and G and *Strep. viridians.*

Symptoms and signs: These include fever, incontinence, dysuria, chills, frequent urination, suprapubic tenderness (cystitis) or tenderness over the renal angle (pyelonephritis), and haematuria.

Investigations: Urinalysis for microscopy and culture. Blood pressure recording and levels of plasma electrolytes and creatinine are commonly investigated in UTIs.

Management: Plenty of water to drink, appropriate antibiotics and symptomatic treatment for fever are the routine management protocols undertaken in UTIs.

- **Acute glomerulonephritis (GN)**

Definition/description: Glomerulonephritis (GN) is a complex inflammatory disease of the glomeruli which can be caused by several factors and may manifest as acute GN, nephrotic syndrome and chronic GN.

Causes: Acute GN is caused by a preceding infection of *Streptococcus pyogenes* (presenting as a sore throat in children, for example). Occasionally this may follow viral infections (including hepatitis B virus infection) and renal involvement in multisystem disorders.

Symptoms and signs: Acute GN is of sudden onset. Symptoms and signs include headache, hypertension, vomiting, loin pain, facial oedema in the morning, haematuria, proteinuria, and uraemia and a reduced amount of urine.

Investigations: Investigations for acute GN include the following:

1. Urine examination includes microscopy for RBC casts and dysmorphic RBC, urine 24-hour protein excretion, and creatinine clearance.
2. Blood examination includes serum for urea and electrolytes (U&E), full blood count (FBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), albumin, antinuclear antibody (ANA), antineutrophil cytoplasmic antibody (ANCA), DNA-binding, complement (C3 and C4), antistreptolysin O (ASO), and HBsAg.
3. Culture is carried out from samples of blood, throat swabs, ear swabs (if otitis is present) and skin swabs (if cellulitis is present).
4. Chest x-ray
5. Renal ultrasound.
6. Renal biopsy.

Management: Bed rest, fluids, antibiotics, and management of hypertension are essential measures. As a complication, acute GN may progress to chronic GN. In chronic GN, the kidneys are small, scarred and unable to function adequately. Clinically, symptoms and signs include those of chronic renal failure, including hypertension and anaemia.

- **Nephrotic syndrome**

Definition/description: Nephrotic syndrome is a form of glomerulonephritis characterised by the heavy leaking of plasma proteins into the urine, resulting in hypoalbuminaemia.

Cause: Glomerulonephritis (GN), diabetes, SLE, infections, amyloidosis, drugs such as NSAIDs or penicillamine, and malignancies (such as lymphoma or leukaemia) may cause nephrotic syndrome.

Symptoms and signs: These include peripheral oedema and swelling of the eyelids, ascites, pleural effusion and frothy urine due to the presence of protein (proteinuria).

Investigations: These include:

1. A 24-hour urine sample for creatinine clearance and protein.
2. Microscopy for RBC and casts

3. Blood examination for U&E and RBC, cholesterol, FBC, ESR, CRP, Ig level, ANA, ANCA, C3and C4.
4. Renal venogram (a diagnostic procedure that uses x-rays and intravenous (IV) contrast dye to visualise the veins within the kidneys and those carrying blood away from the kidneys) or Doppler ultrasound to assess the size, shape and location of the kidneys.

Management: Diuretics for oedema, a high protein diet, infusions of salt–poor albumin to raise serum albumin levels, and corticosteroids or immunosuppressive drugs. Venous thromboses and pulmonary embolism may occur as complications of nephrotic syndrome. Anticoagulation (with streptokinase) may become necessary.

- **Renal Failure (RF): acute and chronic RF**

Definition/description: Renal failure is the loss of renal function leading to uraemia. Two forms exist: acute and chronic RF. chronic RF is characterised by a gradual and permanent loss of renal function.

Cause: These include diabetes mellitus, GN, pyelonephritis, hypertension, renal stones, bladder outlet obstruction, and connective tissue disease.

Symptoms and signs: Acute renal failure (ARF) is also called acute kidney injury (AKI). This is characterised by a rapidly progressive loss of renal function resulting in oliguria, and fluid and electrolyte imbalances. Acute RF involves rapid deterioration of renal function within hours or days. Causes include injuries (due to accidents), complications of surgeries (such as bypass surgery on the heart restricting blood flow to the kidneys for extended periods of time), chemicals and accidental overdoses of drugs.

When kidneys fail to filter properly, waste accumulates in the blood and the body. This condition is called azotemia. This stage may be asymptomatic in some patients. Renal failure accompanied by noticeable symptoms is termed uraemia which is characterised by high levels of urea in the blood. The term uraemia is used for the illness accompanying kidney failure.

In chronic RF any of the following symptoms and signs may be encountered:

Apathy, confusion, drowsiness (due to accumulation of nitrogenous end products), ammoniacal breath odour, brown-coated tongue, metabolic acidosis leading to over-breathing, anorexia, nausea, vomiting, bleeding or bruising tendencies, anaemia, polyuria, peripheral oedema, increased pigmentation, ascites, pleural effusion and pericarditis.

Investigations: Biochemical: increased urea and creatinine, hyperkalaemia (abnormally high levels of potassium in the circulating blood), hypocalcaemia (abnormally low concentrations of calcium in the circulating blood), hyperphosphataemia (abnormally high levels of phosphates in the circulating blood), and hyponatraemia (abnormally low concentrations of sodium ions in the circulating blood). Urine examination includes microscopy for casts, protein analysis, specific gravity and creatinine clearance. Radiology and biopsy includes plain x-ray, retrograde pyelography and renal biopsy.

Management: Management of BP is an essential part of the treatment. BP should be maintained at less than 130/80 mmHg in chronic renal failure (CRF), and for those CRF patients with diabetes, BP should be maintained at less than 120/70mmHg. Vitamin D, avoidance of nephrotoxic drugs such as tetracyclines, a diet low in protein and salt, and renal

dialysis (haemodialysis or chronic ambulatory peritoneal dialysis) are the recommended management strategies for CRF.

Oral Manifestations and Dental Management Considerations

In chronic renal failure and nephrotic syndrome patients may present any of the following oral symptoms and signs:

- Mucosal pallor due to anaemia
- Orange colouration of the mucosa due to deposition of carotene-like pigments.
- Xerostomia with or without candidosis
- Metallic taste
- Ammoniacal salivary odour
- Uraemic stomatitis, and in severe cases with a burning sensation and ulceration
- Petechiae and gingival bleeding
- Necrotising ulcerative gingivitis
- Radiological findings, including a "ground glass" appearance of alveolar bone and tooth erosion due to persistent vomiting

The dentist should consult the physician prior to treating patients with chronic renal failure.

Dental extractions should be scheduled to follow recent renal dialysis, after the heparin effect has worn off.

RF patients on immunosuppressive agents and corticosteroids are predisposed to oral infections.

Nephrotoxic agents are to be avoided (tetracyclines, for example).

Those chronic renal failure patients on dialysis often carry hospital viruses. Dentists should be aware of these patients contracting cytomegalovirus (CMV) and Epstein-Barr virus (EBV) infections.

Strict infection control protocols must be followed when patients with dialysis receive dental treatment.

If the patient is treated as an outpatient, BP should be monitored before and during treatment.

Chapter 11

Disorders of the Endocrine System and Metabolism

S. R. Prabhu and David H. Felix

Abstract

This chapter deals with disorders of the endocrine system and metabolism. Starting with symptoms and investigations common to endocrine diseases, this chapter then deals with individual entities. These include hyperparathyroidism, secondary hyperparathyroidism, hypoparathyroidism, pseudohypoparathyroidism, thyrotoxicosis, myxoedema, diabetes insipidus, acromegaly, Cushing's syndrome (adrenocortical excess), Addison's disease (adrenal insufficiency), diabetes mellitus and disorders of calcium metabolism. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, the investigations employed and principles of management. Where relevant, the oral and dental aspects of endocrine and metabolic diseases are also described.

Introduction

Endocrine and metabolic disorders are relevant to dentistry because some disorders present oral manifestations and require dental management modifications. Important endocrine and metabolic disorders of dental interest are briefly discussed in this chapter.

Common Symptoms in Endocrine Diseases

In endocrine diseases, symptoms of over- or under-secretion of hormones are encountered. Some general symptoms include weight loss or gain, excessive hair loss or growth, skin pigmentation, fertility or menstrual problems, dwarfism, slow mental activity, disturbances in the heart rate and rhythm, gigantism, and chronic fatigue.

Common Investigations in Endocrine Diseases

Investigations into endocrine diseases include thyroid function tests, isotope scanning, fine needle cytology, plain x-rays of the appropriate anatomic region, ultrasound, CT scan, MRI, biochemical investigations such as estimation of levels of hormones, blood glucose, serum cortisol levels, urea and electrolytes, vitamin D levels, serum calcium and phosphate levels.

Diseases of the Parathyroid Gland

Diseases of the parathyroid glands include primary hyperparathyroidism, secondary hyperparathyroidism, hypoparathyroidism and pseudohypoparathyroidism.

- **Primary hyperparathyroidism**

Definition/description: High levels of parathyroid hormone (PTH) due to glandular pathology results in primary hyperparathyroidism.

Cause: Adenoma or hyperplasia of the parathyroid gland.

Symptoms and signs: Often, patients are asymptomatic. When symptomatic, features include polyuria, excessive thirst (due to hypercalcaemia), anorexia, weakness, constipation, vomiting, renal colic, backache, hypertension, renal stones, peptic ulceration, giant cell tumour of the bone, and pancreatitis.

Oral manifestations and dental management considerations

Included are brown tumours of the jaw bones and the loss of lamina dura around the roots**.**

Investigations: These include serum calcium (low), phosphate (raised), alkaline phosphatise (raised), bicarbonate, vitamin D, x-rays of the hands and skull (‘pepper-pot’ like erosions of skull), brown tumours of the bone, abdominal x-rays for renal stones, ultrasound of the neck, radioactive thallium-technetium subtraction scan and immunoassays for PTH.

Management: This includes surgery for adenoma and hyperplasia of the parathyroid gland. In addition, administration of vitamin D in severe cases may be necessary.

- **Secondary hyperparathyroidism**

Definition/Description: Prolonged hypocalcaemia associated with renal failure and deficiency of dietary vitamin D can lead to secondary hyperparathyroidism. Stimulated PTH can also result in secondary hyperparathyroidism. Treatment includes correction of the underlying cause.

- **Hypoparathyroidism**

Definition/description: Hypoparathyroidism may be either primary due to autoimmune disease, or secondary as a result of thyroid surgery.

Symptoms and signs include peri-oral and peripheral paraesthesia, cramps, and abnormalities of the hair, nails and teeth in chronic cases. Tetany in acute cases is characterised by tingling in the extremities, spasms in the hands, facial twitching (Chvostek's sign: contracture of the facial muscles on tapping over the facial nerve) and fits.

Oral Manifestations and Dental Management Considerations

Abnormalities of the teeth may occur in these patients. Chvostek's sign can be elicited in some cases. Dental procedures under LA can be carried out without any potential threat to the patient's health.

Investigations include estimation of serum calcium (low), phosphate (raised), and alkaline phosphatase (normal) levels. Skull x-ray shows basal ganglion calcifications, and plasma PTH levels are low.

Management includes IV administration of calcium gluconate and in acute cases, long term alfacalcidol.

- **Pseudohypoparathyroidism**

Definition/description: This is an inherited disorder with resistance to PTH.

Symptoms/signs include a short stature, mental retardation, 'moon' face, cerebral calcifications, short 4^{th} and 5^{th} metacarpals and hypothyroidism.

Management: Treatment is as for primary hypoparathyroidism.

Diseases of the Thyroid Gland

- **Hyperthyroidism (Thyrotoxicosis)**

Definition/description: Also known as thyrotoxicosis, this disorder is characterised by over-production of thyroid hormone.

Cause: A common cause of hyperthyroidism is an autoimmune disorder (Graves' disease), where antibodies stimulate the thyroid to secrete excess hormone. Less often, a nodule within the multinodular goitre, or a thyroid adenoma producing excessive thyroxin is responsible.

Symptoms: Common symptoms include sweating, heat intolerance, sleep disturbances, irritability, amenorrhoea, palpitations, weight loss, increased appetite, and anxiety.

Signs include tachycardia, atrial fibrillation, exophthalmos, fine tremor, goitre, and peritibial myxoedema.

Oral Manifestations and Dental Management Considerations

- There is an increased risk for periodontal disease in hyperthyroidism and for the premature eruption of teeth.

- A complete history and information from the patient and their physician should be sought.
- If hyperthyroidism is present or suspected, and untreated or poorly controlled, , elective dental treatment should be postponed until the condition is managed successfully by a medical practitioner.
- Currently or previously treated hyperthyroid patients are at risk of hypothyroidism which may go undetected. The treating dentist must be aware of this fact.
- Adrenaline containing local anaesthetics can be used in stable hyperthyroid patients, but for those with unstable hyperthyroidism, this must be avoided in order to prevent a thyroid storm (a medical crisis).
- Swelling at the base of the tongue can sometimes be present in these patients. This may be due to accessory or ectopic thyroid tissue.

Investigations: T3 is raised. T4 or (fT4) is also usually raised. TSH may be suppressed in those with multinodular goitre. The presence of thyroid autoantibodies may be detected.

Management includes antithyroid drugs and surgery.

- **Hypothyroidism**

Definition/Description: Underproduction of thyroid hormone results in hypothyroidism.

Causes include **an** iodine deficiency, (the most common cause worldwide) or an autoimmune disorder (Hashimoto's disease). Thyroidectomy or radiation of the gland, and occasionally hypopituitarism, may be etiologically associated.

Symptoms include weight gain, cold intolerance, depression, tiredness and constipation.

Signs include the slow relaxation of tendon reflexes, myxoedema (deposition of subcutaneous mucopolysaccharides), hair loss, a hoarse voice, cold skin and bradycardia.

Oral Manifestations and Dental Management Considerations

- Untreated neonatal (congenital) hypothyroidism may result in an altered development of the jaws, malocclusion, delayed tooth eruption, and a protruding tongue **(cretinism)**
- In older children and adults, hypothyroidism may result in macroglossia, glossitis, salivary gland swelling, an increased risk of dental caries and periodontal disease. In rare instances, oral lichen planus may be seen in hypothyroid patients.
- Tranquilisers and general anaesthetics may precipitate coma in hypothyroid patients. Local anaesthetics are well tolerated by these patients. Hypothyroid children may show delayed dental development and an increase in decay and periodontal disease.
- Adults with hypothyroidism may show delayed tooth eruption, an enlarged tongue, periodontal disease, alteration in taste sensation and delayed wound healing.
- Seek a complete history and information from the patient and the patient's physician**.**

Investigations include estimation of T4 (low) and TSH (high) levels. Tests for cholesterol (hypercholesterolemia) and anaemia are necessary. Thyroid autoantibodies in the autoimmune variant can be detected.

Management: Thyroxin replacement is the treatment of choice.

Diseases of the Pituitary Gland

- **Hypopituitarism**

Definition/description: A condition characterised by the deficiency of anterior or posterior pituitary hormones is called hypopituitarism.

- **Pituitary dwarfism**

Hypopituitarism in children typically results in short stature with normal proportions, and slow growth due to a deficiency in the growth hormone. This condition is called **pituitary dwarfism.**

Causes include anterior pituitary tumours, surgery on the pituitary for tumours, past head injury, tuberculosis, sarcoidosis, and radiation.

Symptoms and signs include myxoedema, infertility, amenorrhoea, depression, signs of hypoglycaemia, muscle weakness, and short stature.

Oral Manifestations and Dental Management Considerations

Delayed eruption of teeth is a feature of hypopituitarism.

In patients with hypopituitarism, general anaesthesia is contraindicated because of the possibility of the patient lapsing into a coma.

Investigations include CT scan, MRI, glucose, T4 and TSH levels, prolactin, gonadotrophins, cortisol and testosterone levels.

Management: Hormone replacement therapy; recombinant growth hormone (GH) is recommended for children with a deficiency of growth hormone.

- **Acromegaly and gigantism**

Definition/description: Acromegaly and gigantism are syndromes that result when the pituitary gland produces excess growth hormone (GH). Nearly always this is due to a pituitary adenoma. If this occurs after epiphyseal plate closure at puberty, the condition is called **acromegaly**; if before the closure of the epiphyses, the result is **gigantism**.

Symptoms and signs in acromegaly: Excess secretion of growth hormone usually starts between the ages of 20 and 40. Headache is common due to the pituitary tumour. Other features include coarsening of the facial features, enlargement of the extremities, a husky voice, excessive sweating and offensive body odour, hypertension, joint symptoms of degenerative arthritis, peripheral neuropathy, impaired glucose intolerance, menstrual irregularities and heart failure. These patients are at a higher risk of developing gastrointestinal cancers.

Symptoms and signs in gigantism: This is a rare condition. Excess growth hormone secretion begins in childhood before the closure of epiphyses. Bone growth is faster but does not show major deformities. Soft tissue swellings are common. Peripheral nerves are enlarged. A eunuchoid habitus (partially resembling, or having the general characteristics of a eunuch) is often present.

Oral Manifestations and Dental Management Considerations

Mandibular prognathism, malocclusion, diastema, an enlarged tongue, and ankylosis of roots are common in acromegaly.

- **Diabetes insipidus (DI)**

Definition: Diabetes insipidus (DI) is characterised by an inability to produce concentrated urine due to the complete or partial deficiency of antidiuretic hormone (ADH), or renal resistance to the ADH action. These are respectively known as cranial diabetes insipidus and nephrogenic diabetes insipidus.

Causes: Causes of cranial DI can be idiopathic, a head injury, or sarcoidosis. Nephrogenic DI may be caused by drugs, renal disease and glycosuria.

Symptoms and signs: These include polyuria resulting in large volumes of pale coloured urine. Frequent urination, nocturia and polydipsia and dehydration are other common symptoms.

Oral Manifestations and Dental Relevance

In patients with diabetes insipidus, osseous infiltrates are often found on the skull and jaws and can be identified on conventional dental radiographs. Loose teeth are another feature of DI. This may be due to Langerhans cell histiocytosis of gingival tissues. Due to excessive thirst, children with DI drink large amounts of water (fluoridated at optimal level) which can result in dental fluorosis. Vinca alkaloid chemotherapy for Langerhans cell histiocytosis of the gingival tissues is effective.

Local curettage for jaw lesions and the extraction of loose teeth are recommended.

Investigations: Estimation of urine osmolality and a 24-hour urine output is required to confirm polyuria.

Management includes administration of desmopressin for cranial DI, and treatment of the underlying cause for nephrogenic DI. Underlying causes should be identified and treated.

Diseases of the Adrenal Gland

- **Adrenocortical Excess (Cushing's syndrome); hyperadrenalism**

Definition/description: Cushing's syndrome refers to the clinical picture resulting from circulating cortisol excess from any cause, whereas Cushing's disease results from hyperfunction of the adrenal cortex from pituitary ACTH excess, usually due to pituitary adenoma.

Cause: Hyperfunction of the adrenal cortex due to ACTH-dependent or ACTH–independent causes. ACTH–dependant causes include hypersecretion of ACTH from the pituitary gland, or from a non-pituitary tumour such as small cell carcinoma of the lung (ectopic ACTH syndrome), or by administration of exogenous ACTH.

ACTH-independent hyperfunction usually results from therapeutic administration of corticosteroids, or from adrenal adenomas or carcinomas.

Symptoms and signs: These include wasting of tissues, myopathy, thin skin, osteoporosis, easy bruising, truncal obesity with prominent supraclavicular and dorsal cervical fat pads, head and neck fat accumulation (buffalo hump), thin extremities, moon facies, hirsutism (in which women have too much unwanted hair), increased susceptibility to infections, poor wound healing and purple striae on the abdomen.

Oral Manifestations and Dental Management Considerations

- Due to exogenous steroid therapy during stressful dental invasive procedures, patients with Cushing's syndrome are susceptible to adrenal crisis. Dental practitioners should be aware of this and procedures should be planned accordingly.
- Patients are also susceptible to oral infections.
- A Cushingoid person presents a moon face and frontal balding.

Investigations: Urine is to be tested for free cortisol and electrolytes (hypocalaemia). Other tests include a glucose intolerance test, measurement of blood pressure (hypertension), chest x-rays (to exclude bronchial carcinoma), estimation of ACTH levels (high in pituitary disease), serum cortisol levels and CT scans of the pituitary and adrenal glands.

Management includes surgery on the pituitary tumours or adrenal glands where appropriate, and drugs to inhibit cortisol levels.

- **Adrenal insufficiency: Primary adrenal insufficiency (Addison's disease) and secondary adrenal insufficiency**

Definition/Description: A disease of the adrenal glands causing primary adrenal insufficiency is called **Addison's disease**. Panhypopituitarism and a lack of ACTH cause secondary adrenal insufficiency.

Cause: Primary adrenal insufficiency involves autoimmune destruction of the glands in about 80% of cases. Other causes include TB, metastatic disease and hypoparathyroidism, diabetes mellitus and Graves' disease.

Secondary adrenal insufficiency results from panhypopituitarism, a lack of ACTH, and in those patients who are receiving steroids.

Symptoms/Signs: In primary adrenal insufficiency (Addison's disease), lack of cortisol and aldosterone exists. Clinical features include nausea, shock, and bowel disturbances. Other features of adrenal insufficiency include weakness, apathy, anorexia, weight loss, abdominal pain, infrequent periods and constipation. Hypotension, vitiligo, hyperpigmentation of mucous membranes (lips, rectum and vagina, for example), and those areas exposed to sunlight (called "bronzing") and pressure (such as bony prominences). Black freckles are common on the forehead, face, neck and shoulders. Increased pigmentation is due to increased levels of ACTH. In secondary adrenal insufficiency, most clinical features are similar to those of primary adrenal insufficiency, however, muco-cutaneous pigmentation is absent.

Oral Manifestations and Dental Management Considerations

- Pigmentation of the oral mucosa is a feature of Addison's disease. This can appear on any intraoral site but in a majority of patients, the dorsum of the tongue and buccal mucosa are involved. Pigmented lesions are brown, diffuse and patchy in distribution.
- If a patient with adrenal insufficiency is suddenly stressed during a surgical dental procedure, adrenal crisis can be precipitated. Immediate management of this patient would include injection of a glucocorticoid, and fluid replacement.
- For planned invasive procedures on these patients (who are on steroids), doubling the normal amount of steroids on the day of procedure is recommended.

Disorders of Carbohydrate Metabolism

- **Diabetes mellitus**

Definition/description: Diabetes mellitus (DM) is a group of disorders characterised by persistent hyperglycaemia due to a deficiency of endogenous insulin, or resistance to insulin action.

Two types of DM exist: type 1 and type 2 DM.

Type 1 is insulin dependent. Usually it is found in children, who are often prone to ketosis.

Type 2 is non-insulin dependent, and usually occurs in obese older adults. There is concordance in identical twins.

Cause: An autoimmune process resulting in β-cell destruction of the pancreas is the cause of type1 DM. Obesity and genetic component are associated with type 2 DM. Other factors associated with type 2 DM include drugs such as corticosteroid therapy and thiazides, as well as pancreatic disease, Cushing's disease, acromegaly and thyrotoxicosis.

Symptoms and signs include irritability, tiredness, thirst, a dry mouth, weight loss, nocturia, blurring of vision, hyperphagia (excessive hunger and eating), dehydration,

ketonuria, hyperventilation, ketone breath, obesity, lethargy, increased susceptibility to infections (such as pruritus vulvae), and delayed wound healing. These are presenting symptoms of type 2 DM. Polypahgia, polydypsia (excessive thirst) and polyuria (the three 'P's) are classic symptoms of Type 1 DM.

Oral Manifestations and Dental Management Considerations

- A thorough medical history should be obtained.
- Poorly controlled diabetics have an increased risk for periodontal disease and impaired salivary gland function.
- Delayed wound healing is a major consequence of diabetes.
- Blood glucose monitoring prior to invasive dental treatment is essential. This protocol includes:
  - Random blood glucose concentration: Normal range is 3-8 mmol/L, and no modifications are required.
  - Random blood glucose concentration: if between 3.5-12 mmol/L, proceed with routine dental treatment.
  - Random blood glucose concentration: if more than 12 mmol/L, consult the physician for an appropriate adjustment of the diabetic medication.
  - Random blood glucose concentration: if less than 3.5mmol/L, the patient may or may not show signs of hypoglycaemia. In any case, administer oral glucose.
- If a patient is on insulin and presents signs of confusion and oral infection, consider the possibility of diabetic ketoacidosis. Consult the physician.
- Ask the patient to bring their glucose monitor with them.
- Make appointments for mid-morning or early afternoon.
- Instruct the patient to maintain their usual diabetic medication and meals,
- Avoid extensive treatments and long appointments.
- If the patient feels ill during treatment, stop the procedure. Check their blood glucose concentration using a glucometer. For hypoglycaemia, give 20-25 mg of glucose or a fast-acting fruit juice, jelly beans or honey, followed by a lower glycaemic load carbohydrate meal such as a sandwich or dry fruit.
- Keep the patient under observation until they are fully recovered.
- Advise the patient not to drive home, and seek medical review.
- If the patient is drowsy and /or unconscious, cease dental treatment and call EMS.
- Institute basic life support if patient is unconscious.
- Well controlled diabetic patients with no complications of the disease can receive dental treatment without any modifications.
- The dentist should identify signs of hypoglycaemia during treatment. These include hunger, a fast heartbeat, weakness, tingling and altered sensations, confusion, disorientation and sweating.
- Hypoglycaemia can result when patients have received their insulin but failed to eat, or if they have received too much insulin.
- Sometimes drug interactions can cause or enhance hypoglycaemia, examples of which include sulfonylureas (used for DM type 2) and aspirin, or sulfonylureas and fluconazole (an antifungal agent).

- It is to be noted that a hyperglycaemic state is safer and preferred to hypoglycaemic shock.

Investigations for DM include:

1. Urine examination by glucose strips or dipstick method. If glycosuria is detected, the patient should be tested for blood glucose levels.
2. Random blood glucose and fasting blood glucose tests. A random blood glucose level of >14 mmol/l and a fasting glucose level of >7 mmol/l (>120 mg/dl) are diagnostic. An oral glucose tolerance test (OGTT) with 75 g oral glucose is indicative in borderline cases. A test showing >11.1 mmol/l (>200 mg/dl) 2 hours after the glucose load is diagnostic.
3. HbAIc (glycosylated haemoglobin) measurements (which provide an accurate measurement of glycaemic control over a period of three months), and
4. Tests for the evaluation of serum cholesterol (for hypercholesterolemia), creatinine, microalbuminuria for renal disease, fundoscopy for diabetic retinopathy, hypertension and peripheral neuropathy.

Management: **Type 1 DM:** If ketoacidosis is present, immediate administration of IV fluids and insulin is essential. In patients without ketoacidosis (with hyperglycemia and other symptoms), insulin therapy is to be given. Short acting insulin preparations (e.g., Humalog) before meals and long acting (e.g., Humulin I) before bedtime are recommended. Insulin pumps are also available. Dietary control and patient education are also important factors.

Type 2 DM: dietary control, physical exercise, reduction in weight and restricted carbohydrate intake are essential. Treatment with metoformin is necessary for those obese patients who do not respond to dietary control. Some type 2 patients may need insulin to achieve adequate glycaemic control.

Complications of DM: DM can damage a variety of organs.

- **Kidney Damage:** microalbuminuria due to kidney damage (glomerulosclerosis or chronic pyelonephritis).
- **Ocular damage**: background microaneurysms (dots) and haemorrhages (blots), cataracts, vitreous haemorrhages, cotton wool spots and neovascularisation are ocular complications of DM.
- **Heart:** chronic heart disease (CHD) is common (diabetic cardiomyopathy).
- **Circulation:** atheroma of large vessels causing intermittent claudication and stroke. Small vessel disease may cause distal gangrene.
- **Nervous system**: peripheral neuropathy is common in a glove and stocking distribution. Mononeuropathy may result in cranial VI nerve palsy and autonomic neuropathy can cause cardiac arrhythmias, postural hypotension, diarrhoea and vomiting.

Transient diabetes as a complication of pregnancy is known as gestational diabetes.

Disorders of Calcium Metabolism

Introduction

Calcium is required for the proper functioning of muscle contraction, nerve conduction, hormone release and blood coagulation. It also helps regulate several enzymes. Calcium metabolism depends upon a host of factors which include the interplay between dietary calcium, its absorption and excretion; and hormonal interactions of parathormone, vitamin D and calcitonin. Maintenance of calcium is dependent on dietary calcium intake, its absorption from the GIT and excretion by the kidneys. Ninety-nine percent of body calcium is in the bones as hydroxyapatite crystals.

The recommended daily allowance of dietary calcium for healthy individuals is 1 gm. Normal total plasma calcium levels range from 8.8 to 10.4 mg/dL.

Disorders of calcium metabolism occur when the body has too little or too much calcium. Two forms of calcium disorders exist: hypocalcaemia and hypercalcaemia.

- **Hypocalcaemia**

Definition/description: Hypocalcaemia refers to total plasma calcium levels below 8.8 mg/dL in the presence of normal protein levels.

Causes: These include: hypoparathyroidism, vitamin D deficiency, renal disease, magnesium depletion, acute pancreatitis, hypoproteinaemia, septic shock, hyperphosphataemia, and drugs such as anticonvulsants (phenytoin) and rifampin.

Symptoms and signs: Hypocalcaemia may be asymptomatic. When symptomatic, neuromuscular irritability resulting in muscle cramps in the back and legs are common. Prolonged hypocalcaemia may cause cataracts. When plasma levels of calcium reach below 7 mg/dL, tetany may develop. Laryngospasm or generalised seizures are also seen in these patients. Tetany is characterised by paraesthesia of the lips, tongue, fingers and feet, carpopedal spasm, generalised muscle aching, and spasms of the facial musculature (such as tetany). Chvostek's sign and Trousseau's signs are positive in severe hypocalcaemia. Chvostek's sign is characterised by involuntary twitching of the facial muscles elicited by a light tapping of the facial nerve just anterior to the external auditory meatus. Trousseau's sign is characterised by carpopedal spasm by reduction in the blood supply to the hand with a tourniquet or blood pressure cuff inflated to 20 mmHg above systolic BP and applied to the forearm for 3 minutes. Arrhythmia is present in patients with severe hypocalcaemia. Other observable changes in hypocalcaemia include dry and scaly skin, brittle nails and coarse hair.

Oral manifestations: Paraesthesia of the lips and tongue are common in severe hypocalcaemia. Chvostek's sign as described above is a facial manifestation in tetany.

Investigations: These include measurement of plasma Ca levels. Hypocalcaemic patients should also be tested for renal function such as BUN and creatinine. Other tests include estimations of serum phosphate, magnesium and alkaline phosphatase levels.

Management: In tetany, intravenous calcium gluconate is administered. In chronic hypocalcaemia, oral Ca and occasionally vitamin D supplements are recommended. Monitoring of plasma Ca levels at regular intervals is necessary in these patients.

- **Hypercalcaemia**

Definition/description: Hypercalcaemia is characterised by plasma calcium levels above 10.4 mg/L.

Cause: Hypercalcaemia results from excessive bone resorption usually caused by cancer. This may include metastasis from carcinoma, leukaemia, myeloma and lymphoma. Parathyroid hormone excess (hyperparathyroidism) also can cause excessive bone resorption and hypercalcaemia. Other causes of hypercalcaemia include excessive absorption of calcium in the GIT and elevated plasma protein concentrations. Immobilisation with prolonged complete bed rest can cause bone resorption and hypercalcaemia.

Symptoms and signs: Mild hypercalcaemia may be asymptomatic. Clinical manifestations include constipation, anorexia, abdominal pain, nausea and vomiting. polyuria, nocturia and polydipsia are common. In severe cases confusion, delirium, psychosis and coma may occur. Renal damage includes acute renal failure or chronic damage due to kidney stones.

Oral Manifestations: Jaw bone demineralisation, loss of lamina dura and osteitis fibrosa cystica (Von Recklinghausen's disease of bone) may be seen in patients with hypercalcaemia.

Investigations include plasma calcium levels, renal function tests, and appropriate tests for hyperparathyroidism.

Management: Management strategies include a decrease in calcium intake, an increase in calcium excretion, decrease in bone resorption, and removal of excess calcium through dialysis. In cancer associated hypercalcaemia, bisphosphonates are used which inhibit osteoclasts and reduce bone resorption. In patients with vitamin D toxicity, the use of corticosteroids is effective in reducing the intestinal absorption of calcium. In severe hypercalcaemia, haemodialysis is needed.

Chapter 12

Disorders of the Nervous System

S. R. Prabhu

Abstract

This chapter deals with diseases of the nervous system. Starting with common symptoms of disorders of the nervous system, the chapter presents essential details of the clinical examinations and investigations employed in nervous system diseases. This is followed by nervous system diseases of dental interest which include headache, migraine, stroke (cerebrovascular accident), transient ischaemic attack (TIA), epilepsy, parkinsonism and Parkinson's disease, multiple sclerosis, myasthenia gravis, motor neurone disease, , trigeminal neuralgia, atypical trigeminal neuralgia, occipital neuralgia, glossopharyngeal neuralgia, superior laryngeal neuralgia, postherpetic neuralgia, meningitis, and coma and examination of the comatose patient. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of diseases of the nervous system are also described.

Introduction

Neurological diseases such as stroke, epilepsy, facial neuralgias and palsies are common in the general population. From a dental practice point of view, these disorders present oral and maxillofacial manifestations and often require modifications in dental management. A few such conditions are discussed in this chapter.

Common Symptoms of the Nervous System Disorders

Some common general symptoms of nervous system disorders include persistent headache, loss of sensation, dizziness, memory loss, loss of muscle strength, tremors, paralysis, seizures, tinnitus, vertigo, and slurred speech.

Examination of the Nervous System

Look for: muscle wasting, visible fasciculations (muscle twitches) in the thighs and calves occurring at rest (which stop during voluntary movements), tremors, and muscle spasticity, and, and decreased tone.

General Neurological Examination

Assess:

- Power and tone of the musculature,
- Coordination (heel-shin test),
- Sensations such as touch or pain (with the patient's eyes closed),
- Vibration sense,
- Tendon reflexes, plantar reflexes, and abdominal reflexes.

Students are expected to refer to standard textbooks for details.
In this chapter, an assessment is undertaken of the cranial nerves only.

Examination and Assessment of the Cranial Nerves and Their Functions

- **Olfactory (first cranial) nerve**

Test for patency of the nasal airway and test each nostril separately for the sense of smell. With their eyes closed, ask the patient to identify common odours.

- **Optic (second cranial) nerve**

Test for visual acuity and visual fields.
Eyes are tested for near and distant vision by:

- Bringing a finger into the field of vision from the periphery at several points on the circumference of the upper and lower, nasal and temporal, quadrants of the visual field. Ask the patient to indicate the point at which the fingertip is first observed.

An optic disc examination is done using an ophthalmoscope.

- **Oculomotor, trochlear and abducent (third, fourth and sixth) cranial nerves**

  - Note any asymmetry in the eyes.
  - Test pupillary reflexes using a bright light in a dark room, approach the eye from the side (to avoid an accommodation reflex). Shine the light into one eye and check for constriction of the pupil.
  - Ask the patient to gaze into the distance, and then to look at fingers placed near the patient's nose; observe the response of both pupils.
  - Examine for any squint or non-alignment (strabismus).
  - Ask the patient to fix their gaze on the examiner's finger, and report if double vision (diplopia) occurs while following the movement of the finger held about 50 cm away. Test this with one of the patient's eyes closed at a time.
  - While examining eye movements, look for any involuntary eye movement (nystagmus).

- **Trigeminal (Fifth Cranial) Nerve**

Assessment of the sensory function

  - Use cotton wool and pin pricks to test sensory function in all three territories of the sensory divisions of the trigeminal nerve.
  - Compare sensations (with the patient's eyes closed) on both sides of the cheek, forehead and jaws.
  - Assess the corneal reflex with a wisp of cotton wool by approaching from the side and gently touching the edge of the cornea.

Assessment of the motor function:

  - Ask the patient to open their jaw against resistance.
  - Palpate the masseter muscles as the teeth are clenched.
  - Elicit the jaw jerk: ask the patient to let their mouth hang open, place the examiner's thumb on the chin and strike the thumb downwards with a tendon hammer.

- **Facial (Seventh Cranial) Nerve**

Motor function:

  - Ask the patient to frown, wrinkle their forehead, close their eyes as tightly as possible, show their teeth, blow out their cheeks, and whistle.

Sensory function

Test the primary tastes: sweet (sugar), salt (salt), bitter (quinine) and sour (vinegar).

  - Place the test substances on each side of the tongue and ask the patient to identify the tastes (have the patient rinse their mouth after tasting each substance).

- **Vestibulotrochlear (Eighth Cranial) Nerve**

  - Test the patient's hearing by rubbing two fingers together close to one of the patient's ears, with the opposite ear occluded by finger pressure over the meatus. Repeat the procedure for the other ear.
  - Place the base of a vibrating tuning fork on the mastoid bone until the sound fades and then place the tip of the tuning fork in front of the meatus. Normally, the sound should be audible. This is called Rinne's test.
  - Place the base of a tuning fork on the vertex of the skull and ask the patient where they hear the sound. This is called Weber's test.

- **Glossopharyngeal (Ninth Cranial) Nerve**

  - Test the primary tastes as above for the facial nerve.
  - Touch the posterior pharyngeal wall (with a cotton swab) to evoke the gag reflux.

- **Vagus (Tenth Cranial) Nerve**

  - Ask the patient to cough and to vocalise the vowel sounds: 'a', 'c', 'i', 'o', 'u'.
  - Observe the movements of the soft palate and uvula during the gag reflex.
  - Ask the patient to say a prolonged 'aah' and observe the movements of the soft palate and the uvula.

- **Spinal Accessory (Eleventh Cranial) Nerve**

Assess the bulk and power of sternocleidomastoid and trapezius muscles:

  - Ask the patient to shrug their shoulders against resistance.
  - Ask the patient to flex their neck, pressing the chin downwards against the examiner's hand.
  - Ask the patient to rotate their chin to each side in turn against resistance to test each sternocleidomastoid muscle.

- **Hypoglossal (Twelfth Cranial) Nerve**

Inspect the tongue for wasting and fasciculations

  - Ask the patient to protrude their tongue.
  - Ask the patient to press their tongue against each cheek while applying resistance externally.

Common Investigations in Nervous System Disorders

Some of the commonly employed investigations in neurological disorders include:

- Lumbar punctures for suspected infection subarachnoid haemorrhage and malignancy
- CT scans for subdural or extradural haematoma, intracranial tumours, oedema and hydrocephalus
- Angiography for vascular occlusions and vasculitis
- Electroencephalography for epilepsy and encephalopathy
- MRI for posterior fossa and spinal cord examination, and to detect demyelination,
- Myelography for visualising the spinal canal and cord
- Electromyography for distinguishing myopathies from neuropathies,
- Brain biopsies used for suspected tumour.

Neurological Diseases of Dental Interest

- **Headache**

Headache is not a disease, it is a symptom. The reader is referred to Chapter 4 for further details.

- **Migraine**

Migraine is a chronic neurological disorder characterised by recurrent moderate to severe headaches.

Cause: The underlying causes of migraines are unknown, however, they are believed to be related to a mix of environmental and genetic factors. In about two-thirds of cases, migraines run in families, and they rarely occur due to a single gene defect. Triggers may include cheese, wine and coffee.

Symptoms and signs: Typically the headache is unilateral and pulsating or throbbing in nature, lasting from 2 to 72 hours. Associated symptoms may include nausea (in 90% of cases), vomiting (in 30% of cases), photophobia (increased sensitivity to light), phonophobia (increased sensitivity to sound). The pain is generally aggravated by physical activity. Up to one-third of people with migraine headaches perceive an aura characterised by blurred vision, flashing lights, numbness, tingling sensation and funny smells. These are transient visual, sensory, or motor disturbances which signal that a headache will soon occur. Other symptoms may include: nasal stuffiness, diarrhoea, frequent urination, pallor, or sweating. Swelling or tenderness of the scalp and neck stiffness may occur.

Diagnosis - Investigations: The diagnosis of a migraine is based on signs and symptoms. Imaging tests, including x-rays or MRI, are occasionally performed to exclude other causes of headaches.

If medications are effective in reducing the frequency or severity of the migraine attacks by at least 50%, they are considered as preventive migraine medications. Topiramate,

propranolol, metoprolol, angiotensin-converting enzyme inhibitors, and amitriptyline are used with good results. Botox has also been found to be useful in sufferers of chronic migraines.

- **Stroke (Cerebrovascular accident)**

Definition/description: Stroke, also known as cerebrovascular accident (CVA) is characterised by a rapid loss of brain function due to disturbance in the blood supply usually resulting from ischaemic infarction or haemorrhage within the brain.

Cause: These include thrombosis, embolism, haemorrhage, vasculitis and hypoperfusion (general decrease in blood supply as in shock).

Risk factors: These include old age, hypertension, previous attack of stroke, transient ischaemic attack, diabetes, hyperlipidaemia, tobacco smoking, excessive alcohol, oral contraceptive pills and atrial fibrillation.

Symptoms and signs: Symptoms depend on the area of the brain involved. They include hemiplegia and weakness of the face, numbness, reduction in vibratory or sensory sensations, head ache, vomiting, initial flaccidity (hypotonicity) replaced by spasticity (hypertonicity). In most cases, involvement is unilateral. Depending on the part of the brain affected, the defect in the brain is usually manifested on the opposite side of the body.

If the brain stem is involved, signs include altered smell, taste, hearing, vision, drooping of the eyelid, weakness of ocular muscles, decreased reflexes (such as gag, swallowing, and pupil reactivity to light), decreased sensation and muscle weakness of the face, balance problems and nystagmus, altered breathing and heart rate, inability to turn the head to one side, inability to protrude the tongue and or move it from side to side.

If the cerebral cortex is involved, patients experience difficulty with verbal expression (aphasia), auditory comprehension, reading and writing (Broca's area involvement), altered voluntary movements, memory deficit, and disorganised thinking.

If the cerebellum is involved, an altered walking gait, movement coordination and vertigo are indicative.

Investigations: Blood pressure, urea and electrolytes, liver function tests, blood chemistry, CT scans, coagulation studies, ESR, EEG, ECG. MRI, ultrasound study of the carotids, and cerebral angiogram measurements are employed.

Management: Admission to a stroke unit of a hospital.

In acute cases, thrombolysis must be carried out. Measures include the use of aspirin, heparin or warfarin (for cardiac emboli). Carotid endarterectomy is indicated if carotid artery stenosis is the cause. Hypertension should be monitored and not treated acutely. A multidisciplinary rehabilitation approach is required in the management of these patients. Maintenance of hydration, frequent turning of the patient to avoid bed sores, and control risk factors need to be taken in to consideration.

Prognosis: 20% show improvements in the first month, 5-10% annually thereafter, and 40% make a complete recovery. Poor prognosis includes deviation of the eyes and head, drowsiness and hemiplegia.

Oral Manifestations and Dental Management Considerations

- Oral and facial manifestations include slurred speech, difficulty swallowing, unilateral paralysis of the oral and facial musculature, deviation of the tongue and loss of sensory stimuli of the oral tissues may occur after an episode of stroke.

Dental management considerations include:

- Identification of the stroke-prone individual. These include patients with hypertension, diabetes, coronary atherosclerosis, are heavy smokers and have had previous episodes of stroke or transient ischaemic attack (TIA).
- Consultation with the patient's physician.
- For patients who have had a stroke during the last six months, only emergency dental treatment is recommended.
- Patients on anticoagulant therapy are predisposed to bleeding problems.
- Mid-morning appointments are recommended.
- For procedures under local anaesthesia, the minimum amount of local anaesthetic with vasoconstrictor is to be used.
- Fixed prostheses are preferred over removable prosthetic appliances.
- Blood pressure should be monitored during treatment.

- **Transient Ischaemic Attack (TIA)**

Definition/description: Transient ischaemic attack refers to focal brain ischaemia, which produces sudden neurologic deficits that last less than an hour. Most TIAs last less than 5 minutes. Brain infarction is unlikely if neurological deficits resolve within an hour.

Cause includes emboli usually from carotid or vertebral arteries.

Symptoms and signs: TIAs and strokes cause the same symptoms, such as contralateral paralysis (opposite side of body from the affected brain hemisphere) or sudden weakness or numbness. A TIA may cause sudden dimming or loss of vision, aphasia, slurred speech (dysarthria) and mental confusion. Transient monocular blindness may occur when the ophthalmic artery is involved. Usually this lasts less than 5 minutes. Patients may have several TIAs daily, or 2 or 3 over several years.

Diagnosis/investigations: History offers some clue but neuroimaging such as CT (to exclude haemorrhage) and MRI (to rule out infarction) are recommended.

Management Arterial angioplasty and stenting is useful for those who have no neurologic deficits but are at a higher risk of stroke. Prevention of stroke with antiplatelet therapy should be considered in patients with TIA.

- **Epilepsy**

Definition/description: Epilepsy is a disorder characterised by a periodic disturbance in neurological function resulting in seizures due to abnormal excessive electrical discharge within the brain.

Cause: In the majority of cases, the cause is not known. In infants, causes include hypoxia, metabolic disorders and infections. In adolescents, trauma, alcohol, drugs, infections and tumours are causally associated with epilepsy. In the elderly, causes may be cerebrovascular, metabolic, tumours and infections.

Symptoms and signs: Changes in mood or behaviour are noticed during the prodromal period. This period may last for hours and is not a part of the seizure. Patients may also report a strange feeling in the gut. This sensation is called an aura.

Epileptic seizures: There are two types, partial and generalised.

Partial epileptic seizures may include motor, sensory, psychic and autonomic signs. Involuntary movements of body parts, olfactory and visual changes, hallucinations, fear, tachycardia and dizziness are common. Impaired consciousness lasting from a few seconds to two minutes, and repetitive movements of the face or limbs are the hallmarks of partial epileptic seizures.

Generalised seizures are divided in to several types. These include: Tonic-clonic seizures (Grand Mal seizures), status epilepticus, Petit-mal seizures, myoclonic seizures, atonic seizures, clonic seizures, and tonic seizures.

- ***Tonic-clonic seizures (grand mal type):*** Signs include an aura, consisting of auditory, gustatory, olfactory, or hallucinatory symptoms, slurred speech, frequent blinking and irritability, followed by the sudden loss of consciousness with an epileptic cry. This phase lasts for less than a minute and the individual may show signs of cyanosis and tachycardia. This is the tonic phase (of the tonic-clonic seizures). The clonic phase (of the tonic-clonic seizures) lasts for a few seconds to several minutes. Signs of the clonic phase include forceful jerking of the head, trunk and extremities, loss of bladder control, and biting the tongue. In the postictal phase, the individual slowly returns to consciousness, followed by headache, sleepiness and disorientation.
- ***Status epilepticus:*** A tonic-clonic seizure of repeated episodes, or an attack of seizure lasting more than five minutes without a recovery, is called status epilepticus. The possibility of airway obstruction and aspiration may cause hypoxemia and acidosis leading to death. This is a medical emergency.
- ***Petit Mal seizures:*** Signs include facial twitching and minor movements of the hands without generalised muscular activity.
- ***Myoclonic seizures:*** Signs include brief jerks of a finger, hand or foot lasting a few minutes.
- ***Atonic seizures (Drop seizures):*** Signs include the sudden loss of tone of muscles, resulting in hand dropping or the individual falling to the ground.
- ***Clonic seizures:*** Rhythmic jerking movements of the body with impaired consciousness are signs of this type of seizure.
- ***Tonic seizures:*** Signs are stiffening of the body or limbs with a risk of falling. Seizures last up to 20 seconds and are followed by a postictal phase.

Investigations: Blood chemistry, urea and electrolytes, EEG, CT and MRI scans of the brain are employed

Management: Anticonvulsants such as phenytoin, carbamazepine, valproate, gabapentin and vigabatrin. In some cases, surgery may become necessary.

Oral Manifestations and Dental Management Considerations

- Facial twitching is a feature of petit-mal seizures.
- Those patients taking phenytoin may present hyperplasia of the maxillary and mandibular anterior gingival tissues.
- Oral soft tissue injuries (ulcers of the tongue, for example) due to trauma received during seizures may be detected.
- Dental management considerations include:
- Identification of an epileptic patient by history taking is the first step.
- Once identified, seizure history, such as the frequency and type of seizures, and the patient's drug regime must be obtained
- Patients with well-controlled care for epilepsy pose no dental management problems.
- If epilepsy is poorly controlled, the patient's physician should be consulted before the commencement of dental treatment. Often modification of treatment may be necessary.
- Patients must be reminded of the adverse effects of anticonvulsant medications (drowsiness, dizziness and ataxia, for example).
- Patients on valporic acid or carbamezapine may present with bleeding tendencies. It is advisable to have bleeding and clotting time estimation done prior to the commencement of invasive treatment.
- Erythromycin should be avoided for those on carbamazepine, as it interferes with the metabolism of carbamazepine.
- Dentist should be prepared to manage seizures of the grand-mal type of epilepsy.
- If a seizure occurs during dental treatment, the patient must be turned onto their side to avoid aspiration. The surrounding area should be cleared as soon as the patient suffers an attack of epilepsy.
- The chair back should be in the supine position.
- The use of a padded tongue blade should be avoided in these patients.
- After the seizure, dental treatment should be discontinued and an arrangement for patient transport must be made.

- **Parkinsonism and Parkinson's disease**

Definition/Description: ***Parkinsonism*** is a clinical condition characterised by slow movement (bradykinesia) and speech, an expressionless, mask-like face, reduced movement (hypokinesia), rest tremor, rigidity and postural instability. *Parkinson's disease* is one of the causes of Parkinsonism, a result of dopamine depletion within the basal ganglion of unknown cause.

Cause: Degeneration of dopaminergic neurons in the substantia nigra (Parkinson's disease). Less common causes of this disorder include drugs, cerebral tumours, Wilson's disease, carbon monoxide poisoning, communicating hydrocephalus and head trauma.

Symptoms and signs: In Parkinson's disease, the gait is characterised by shuffling forwards with a flexed trunk (festinant gait). Limbs resist passive extension (lead-pipe rigidity or cog-wheel rigidity) during movement. The rest tremor is slow with a 'pill-rolling' movement which becomes worse during stress.

Investigations: Diagnosis is usually clinical through history and clinical (neurological) examination. PET and DAT scans may aid diagnosis.

Management: For mild cases, anticholinergic drugs (amantadine) and for advanced cases, L-dopa or dopamine agonists are prescribed. Speech therapy and occupational therapy are also important in advanced cases.

Prognosis: Improvement in symptoms can be achieved, but the progression of the disease cannot be stemmed. Lifespan in idiopathic Parkinson's disease is not greatly affected. Dementia appears in about 40% of cases.

Oral Manifestations and Dental Management Considerations

- Other than excessive salivation, there are no specific oral manifestations of Parkinson's disease**.**
- Dental management considerations include:
- The low-dose sedation of patients who have severe tremors.
- The amount of LA with epinephrine should be minimised.
- Clarithromycin, tetracycline, doxycycline and azole antifungals should be avoided for those on dopamine agonist drugs.

- **Multiple sclerosis (MS)**

Definition/Description: A chronic inflammatory demyelinating disorder with formation of plaques throughout the central nervous system. Peripheral nerves are not affected in MS.

Cause: Not known. Possible causes may include living in temperate zones and an autoimmune process.

Symptoms and signs: MS is common in women. Disturbances in visual function, such as painful eyeball movements, nystagmus (involuntary eye movement), diplopia (double vision), distortion of the central vision and vision loss (predominantly optic nerve involvement) are common in MS. Sensory symptoms include feeling numb, cold, pins and needles, swelling and tightness in the arms and legs. Motor weaknesses include paraplegia, difficulty in walking, vertigo and loss of balance. A relapsing-remitting course is typical.

Investigations: History and clinical features are suggestive. MRI is indicated for periventricular lesion detection. IgG in CSF is elevated. Investigations are not pathognomonic.

Management: There is no cure for MS. High doses of steroids (methylprednisolone), immunomodulators (cyclophosphamide, or azathioprine, for example) and interferon beta, are useful therapeutics.

Oral Manifestations and Dental Management Considerations

- MS Patients may experience facial pain, and the disorder can trigger trigeminal neuralgia or facial palsy.
- MS patients may complain of facial anaesthesia or lower lip numbness.
- Xerostomia and associated oral findings are common.
- Shorter and morning appointments are recommended.
- A semi-inclined chair position is recommended.
- A complete blood count is recommended before the start of invasive treatment because of the effects of immunosuppressive agents on the blood elements.
- Hepatotoxic drugs are to be avoided.
- Steroid supplements may be necessary prior to invasive dental treatment for those who are already on steroid therapy for MS (for longer than two weeks).
- The amount of anaesthetic agent should be minimised. In the presence of tricyclics antidepressants, local anaesthetics containing epinephrine are to be avoided.

- **Myasthenia gravis (MG)**

Definition/Description: An autoimmune disease causing the depletion of functioning muscle acetylcholine receptors in the neuromuscular junction, leading to muscle weakness.

Cause: Myasthenia gravis is an autoimmune disorder. It may be associated with thymic hyperplasia, hyperthyroidism, SLE, and rheumatoid arthritis.

Symptoms/signs: Muscle weakness of the neck, trunk, limbs and ocular muscles result in ptosis. Dysphasia (difficulty with speech), diplopia and dysarthria, and 'myasthenic snarl' on smiling, are other significant features of the disorder.

Investigations: These include clinical examination, detection of acetylcholine antibody receptors, positive Tensilon test (which shows improvement in muscle weakness), EMG and CT scans.

Management: Treatment of MG includes acetylcholinesterase inhibitors, steroids, and azathioprine. Plasma exchange (plasmapheresis) is indicated for severe cases.

Oral Manifestations and Dental Management Considerations

Oro-facial features include:

- Facial weakness, a sensation of stiffness of the mouth, an inability to whistle, myasthenic snarl, chewing difficulty, regurgitation of fluids through the nose, and choking.
- Patients of MG also experience inability to keep the head in balance

Dental management considerations include:

- Confirmation of the extent of the disease (entire body, and head and neck region in particular).
- Before the commencement of treatment, the gag reflex should be checked.

- Morning appointments are recommended.
- Active suction is necessary due to increased salivation.
- Muscle relaxants should be avoided.
- A minimal dose of local anaesthetic to be used.
- Diazepam (valium), tetracyclines and general anaesthesia are to be avoided.

- **Motor neuron disease (MND)**

Definition/Description: The motor neuron diseases (MND) are a group of degenerative neurological disorders that selectively affect motor neurons, the cells that control voluntary muscle activity including speaking, walking, breathing, swallowing and general movement of the body. Both upper motor neurons (UMN) and lower motor neurons (LMN) may be affected with no sensory abnormality.

Cause: Unknown.

Symptoms and signs include slurred speech, drooling saliva, dysphagia, weakness, breathlessness, limb pain, dysphasia, dysarthria, wasting of the tongue with back jaw jerk, and neck weakness.

Diagnosis/investigations: These include history and clinical examination, EMG, CSF analysis, syphilis serology, and cervical spine imaging.

Management protocol includes the delivery of analgesics, anticholinergics for reducing salivary production, and antidepressants. Good home care is essential.

Oral Manifestations and Dental Management Considerations

The oral aspects of MND are listed above.

Dental management considerations include:

- Consulting with the patient's physician to determine the extent of the disorder.
- Assessing the patient's ability to tolerate dental procedures.
- Early morning appointments are preferred.
- Active suction is required because of excessive saliva.
- Back jaw jerk is a feature of MND patients, so appropriate care should be taken to avoid accidental episodes with dental instrumentation.
- Maintaining good oral hygiene at home.

- **Bell's palsy**

Definition/Description: Bell's palsy is a form of facial paralysis resulting from a dysfunction of cranial nerve VII (the facial nerve). Patients with Bell's palsy experience inability to control facial muscles on the affected side.

Causes: These include idiopathic or viral infections (EBV or VZV) of the nerve, emotional and physical stress, exposure to cold conditions, brainstem tumours, multiple sclerosis, strokes, trauma to the parotid gland and parotid tumours.

Symptoms and signs: Unilateral sagging of the mouth, taste impairment, saliva dribbling and watery eyes, an inability to whistle and close the lips, or blow out the cheeks are all signs of Bell's palsy. The palpebral fissures are wide. Unilateral lacrimation (the production of crocodile tears) in the first month following a Bell's palsy episode occurs when the patient eats. Loss of taste is common.

Investigation: History and clinical examination offer important clues to diagnosis. Diagnosis is by the exclusion of other possibilities. CT scan and MRI are indicated if intracranial lesions have been suspected.

Management: Steroids, antiviral agents (less useful if used alone) and surgery in some cases are included in the treatment protocol.

Oral Manifestations and Dental Management Considerations

- Oral and facial manifestations of Bell's palsy are listed above.

Dental management considerations include:

- A history of the patient's recent illness (such as viral fevers) and the onset and duration of symptoms of facial palsy.
- A thorough clinical examination of the sensory and motor functions of the face.
- Long term corticosteroid therapy may cause adrenal suppression which must be taken into consideration. In children, great caution must be exercised with corticosteroid therapy. This may cause growth suppression, so medicating on alternate days is advised.
- Home oral care advice must be given to Bell's palsy patients, or if a child, to their parents/guardians.
- Consulting with a neurologist and ophthalmologist may become necessary if patient recovery is unduly delayed, and ophthalmic symptoms persist for a long time.

Neuralgias

Definition/Description: Neuralgia refers to a sharp, shocking pain that follows the path of a nerve and is due to irritation or damage to that nerve.

Under the general heading of neuralgias are trigeminal neuralgia (TN), atypical trigeminal neuralgia (ATN), occipital neuralgia, glossopharyngeal neuralgia, superior laryngeal neuralgia and postherpectic neuralgia (caused by shingles).

- **Trigeminal neuralgia (TN):**

Symptoms and signs: Oral manifestations

- Pain involves the excitation of one or more of the three branches (mandibular, maxillary or ocular) of the trigeminal nerve.
- In most cases, pain is precipitated by touching, eating or talking.

- Pain is of sudden onset, short duration, sharp or lightning-like, or stabbing in nature, and is unilateral.
- Repetitive episodes can occur.
- In between the attacks, the area involved generally does not show any signs of pathology.
- The pain does not cross to the contralateral side.

Treatment:

- The anticonvulsant carbamazepine (Tegretol) is the first line of treatment. Other medications used include baclofen, lamotrigine, oxcarbazepine, phenytoin, gabapentin, pregabalin, and sodium valproate.
- Uncontrolled trials have suggested that clonazepam and lidocaine may also be effective.
- Remissions occur spontaneously and may last for months to years.
- Surgical treatment (section of trigeminal root) is indicated if medical treatments fail.

- **Atypical trigeminal neuralgia (ATN)**

Symptoms: Oral manifestations

- ATN can display a wide range of symptoms.
- Pain can fluctuate in intensity from a mild aching to a crushing or burning sensation.
- ATN pain can be described as heavy, aching, and burning. Sufferers have a constant migraine-like headache and experience pain in all three trigeminal nerve branches.
- The areas affected include aching teeth, ear aches, feeling of fullness in sinuses, cheek pain, pain in the forehead and temples, jaw pain, pain around the eyes, and occasional electric shock-like stabs.
- Unlike typical neuralgia, this form can also cause pain in the back of the scalp and neck.
- Pain tends to worsen with talking, facial expressions, chewing, and on feeling certain sensations, such as a cool breeze. Vascular compression of the trigeminal nerve, infections of the teeth or sinuses, physical trauma, or past viral infections are all possible causes of ATN.

- **Occipital neuralgia**

Cause: Occipital neuralgia is caused by damage to the occipital nerves usually due to trauma, physical stress on the nerve, or repetitious neck contraction, flexion or extension.

Symptoms:

- An aching, burning and throbbing pain that typically starts at the base of the head and radiates to the scalp.
- Pain on one or both sides of the head.
- Pain behind the eyes.

- Sensitivity to light.
- Tender scalp.
- Pain when moving the neck.

Treatment: Antidepressants and nerve blocks are helpful.

- **Glossopharyngeal neuralgia**

This involves unilateral irritation of the ninth (IX) cranial nerve. Sensory vagal nerve afferents are also suspected in this pain syndrome.

Symptoms and Signs: Oral Manifestations

Cutting, stabbing and shooting pain, or sharp sensations in the throat.

Throat pain can last from minutes to hours. Ipsilateral ear sensations of "fullness" may occur prior to the pain episode in the throat.

Triggers include swallowing, talking, yawning, and coughing.

Activation of the dorsal motor nucleus of the vagus nerve (cranial nerve X) during a glossopharyngeal neuralgia episode may result in bradycardia and syncope.

- **Superior laryngeal neuralgia**

Symptoms: Oral manifestations

- Activation of the superior laryngeal nerve occurs via the general visceral afferent component of the vagus nerve.
- This is a rare pain syndrome associated with lowered pitch and pain on vocalisation, with lateral throat pain occurring within the submandibular region.
- Pain may also present under the ear. Pain episodes can last from minutes to days.

- **Postherpetic neuralgia (PHN)**

In PHN, nerve damage is caused by the reactivation of varicella-zoster virus. The damage causes nerves in the affected dermatomic area of the skin to send abnormal electrical signals to the brain. These signals may convey excruciating pain, and may persist or recur for months or years, or even for life.

Cause: With resolution of the herpes zoster (shingles) eruption, pain that continues for three months or more is defined as PHN. Elderly and immunocompromised patients are susceptible.

Symptoms and signs: Oral manifestations

- The pain level is variable, from discomfort to very severe, and may be described as burning, stabbing or gnawing.
- Area of previous HZ (the side of the face, for example) may show evidence of cutaneous scarring.
- Sensations may be altered over the involved areas, in the form of either hypersensitivity or decreased sensation.

Treatment. Possible options include:

- Antiviral agents, such as famciclovir, are given at the onset of attacks of herpes zoster to shorten the clinical course and to help prevent complications such as postherpetic neuralgia.
- Analgesics
    - Topical agents
        - Aspirin mixed into an appropriate solvent such as diethyl ether may reduce pain.
        - Gallium maltolate in a cream or ointment base has been reported to relieve refractory postherpetic neuralgia.
        - Lidocaine skin patches.
    - Systemic agents include
        - Paracetamol or non-steroidal anti-inflammatory drugs.
        - Opioids such as codeine, tramadol, morphine or fentanyl.
    - Antidepressants in smaller doses. Low dosages of tricyclic antidepressants, including amitriptyline.
    - Anticonvulsants such as phenytoin (Dilantin, Phenytek), carbamazepine (Carbatrol, Tegretol) for sharp, jabbing pain. Newer anticonvulsants include gabapentin (Neurontin) and lamotrigine (Lamictal).
    - Corticosteroids are often used but do not provide much relief.

Other Related Topics

Coma and Examination of the Comatose Patient

Definition/description: Coma is a state of unconsciousness.

Causes: These include extradural or subdural haemorrhage due to head injury, hypoglycemia, diabetic ketoacidosis, hypernatraemia, cardiac failure, respiratory failure, liver failure, renal failure (uremic coma), hypothyroidism, stroke, subarachnoid haemorrhage, meningitis encephalitis, cerebral malaria, alcohol intoxication, overdoses of opiates, carbon monoxide poisoning, brain tumours, epilepsy and brain abscesses.

Symptoms and signs: Prior to the onset of coma, severe headaches may occur. This may be caused by subarachnoid haemorrhage or meningitis. Progressive headaches, worse in the morning and associated with vomiting, may be due to increased intracranial pressure from a cerebral tumour. A history of diabetes, renal failure, respiratory failure, hepatic failure, or cardiac failure provide important clues to the cause or predisposing factors involved.

Examination of a Comatose Patient

- The body temperature of all comatose patients should be recorded to exclude hyperpyrexia or hypothermia.

- A detailed examination of the body in general and scalp in particular should be carried out for bleeding, haematomas or fractures of the skull.
- The presence of bilateral periorbital haematomas or CSF rhinorrhoea may indicate an anterior fossa fracture of the skull.
- In middle cranial fossa fractures, bruising over the mastoid region or CSF rhinorrhoea are present.
- In carbon monoxide poisoning, patients look bright red.
- Needle puncture wounds on the arms may indicate drug abuse.
- In meningococcal meningitis, the skin may show petechiae.
- The pulse should be examined for arrhythmias.
- JVP should be recorded for conditions such as tension pneumothorax.
- Auscultation of the thorax should be carried out for crepitations with pulmonary oedema from ventricular failure and bronchopneumonia.
- For trauma patients, neck stiffness is tested. This is also present in meningitis and subarachnoid haemorrhage.
- For trauma patients a cervical spine assessment is to be done before any manipulation is undertaken.
- Pupils are to be tested for size and light reflex. Pinpoint pupils are seen in opiate overdoses, small pupils in brainstem lesions, and dilated pupils in cocaine or amphetamine use. A unilateral fixed pupil occurs in patients with occulomotor nerve lesions which may be due to pressure from an intracranial tumour or haemorrhage.
- A limited neurological examination, including testing reflexes and tone, should be carried out. Unilateral increased tone and hyperflexia, and an up-turning plantar response is indicative of a contralateral upper motor neuron lesion such as a stroke, haemorrhage or tumour.
- A fundoscopy to identify retinal vein pulsation or frank papilloedema indicating increased intracranial pressure.
- General investigations include FBC, blood glucose, urinalysis for ketones, urea and creatinine for renal function, LFTs for liver function, a toxicology screen for poisoning and overdoses, ECG for cardiac arrhythmias or cardiac infarction, chest x-rays for pulmonary oedema, x-rays of the cervical spine in traumatic cases, and CT to detect intracranial tumours and haemorrhage.
- A lumbar puncture should be performed for suspected meningitis.
- EEG is to be recorded for suspected epilepsy and encephalitis.

Chapter 13

Haematological Disorders

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Abstract

This chapter deals with common haematological disorders of dental interest. The disease entities discussed include haemolytic anaemia, iron deficiency anaemia, aplastic anaemia, pernicious anaemia, sickle cell anaemia, thalassaemia, thrombocytopenia, haemophilia, Christmas disease, von Willebrand's disease, leukaemia, multiple myeloma, Hodgkin's lymphoma, non-Hodgkin's lymphoma, Burkitt's lymphoma and lymphadenopathy. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of haematological diseases are also described.

Introduction

Haematology includes the study of etiology, diagnosis, treatment, prognosis, and prevention of blood diseases that affect the production of blood and its components, such as blood cells, hemoglobin, blood proteins, and the mechanism of coagulation.

Common Symptoms in Haematological Disorders

Symptoms of diseases of the blood and its components include fatigue, pallor, bleeding tendencies, defective coagulation and haemorrhagic episodes, and symptoms associated with infections.

Investigations in Haematology

Investigations in haematology include a full blood count, blood film for morphology of red blood cells, white blood cells and platelets, ESR, haemoglobin studies, bone marrow studies, serum protein estimation, immunofluorescent studies and isotope studies.

Haematological Diseases of Dental Interest

- **Anaemias**

Definition/description: Anaemia refers to a decrease in the oxygen-carrying capacity of the blood, caused by decreased production of red blood cells, increased destruction of red blood cells, increased demand for iron, or formation of abnormal red blood cells.

Common symptoms and signs of anaemia: Symptoms include fatigue, breathlessness, palpitations, headache, tinnitus, anorexia, and bowel disturbances. Signs include pallor, retinal haemorrhages, and tachycardia, heart murmurs and cardiomegaly leading to heart failure in severe cases. In chronic iron deficiency, anaemic nail changes lead to spoon-shaped concave nails (koilonychia).

Classification of anaemia and haemoglobinopathies:

- Haemolytic anaemia (autoimmune and non-autoimmune)
- Iron-deficiency anaemia (microcytic anaemia)
- Aplastic anaemia (normocytic anaemia)
- Pernicious anaemia (macrocytic anaemia)
- Sickle cell anaemia
- Thalassaemia

- **Haemolytic anaemia**

Definition/description: Haemolytic anaemia refers to the excessive intravascular or extravascular (in the spleen) destruction of red blood cells. The normal survival rate of RBCs is about 120 days. In haemolytic anaemia, it is much shorter.

Causes include autoimmune causes, infections, splenomegaly, drugs, RBC membrane disorders (spherocytosis), enzymopathies (deficiency of glucose-6-phosphate dehydrogenase) and haemoglobinopathies (sickle cell disease and thalassaemia).

Drugs which trigger haemolysis in G-6-PD deficiency include acetylsalicylic acid, ascorbic acid, dapsone and vitamin K. Fava bean ingestion in the diet is also associated with this form of anaemia. Malaria is the most common cause of anaemia in the developing world.

Symptoms and signs (of G-6-PD deficiency associated haemolytic anaemia):

Symptoms include jaundice, palpitations, dyspnoea and dizziness. Signs include splenomegaly, cyanosis and Reynaud's phenomenon.

Investigations: A full blood count shows spherocytosis. A direct antiglobulin test (Coomb's test) demonstrates the antigen responsible for RBC destruction. A rise in bilirubin

and LDH, and urinary haemosiderin are other features of G-6-PD associated haemolytic anaemia.

Management: Identify the cause and administer the appropriate treatment. Steroids and splenectomy are useful. Raynaud's phenomenon can be avoided by keeping warm.

- **Iron deficiency anaemia (microcytic anaemia)**

Definition/Description: Iron deficiency anaemia is due to chronic blood loss. This is the most common form of anaemia.

Cause: An increased requirement for, or decrease in the intake of, iron can cause an iron deficiency. Chronic blood loss is the most common cause of iron deficiency anaemia.

Symptoms and signs: In the early stages of iron deficiency the condition may be asymptomatic. Fatigue and loss of stamina are common in severe cases. Patients may have an abnormal craving to eat clay, dirt, paint, ice, etc. This abnormal craving is called pica. Glossitis, cheilitis and abnormal nails showing concavity (spoon-shaped nails called koilonychia) are common in advanced stages. Postcricoid web in these patients may cause dysphagia. These patients are vulnerable to developing oesophageal carcinoma.

Investigations: Laboratory investigations include a FBC for the estimation of haemoglobin, haematocrit, mean corpuscular volume, and mean corpuscular haemoglobin concentrations. The FBC will show low values for all these in iron deficiency anaemia. In addition, microcytosis is present and the RBC distribution width is increased. Other investigations include serum iron (low), iron-binding capacity (increased) and serum ferritin (low) estimations.

Management: Management protocols include the identification and elimination of the cause of blood loss, and administration of oral or parenteral iron therapy. Iron salts such as ferrous sulphate, gluconate or fumarate are used. Therapy should continue for up to six months after the correction of haemoglobin levels in order to replenish iron stores in the tissues. The progress of treatments should be assessed by serial haemoglobin measurements until RBC levels reach normal values.

- **Aplastic anaemia (normocytic anaemia).**

Definition/description: A decrease in haematopoietic bone marrow leading to pancytopenia (involving all blood cells) results in aplastic anaemia.

Cause: These include idiopathic (60%), hereditary (Fanconi's anaemia), viral hepatitis, irradiation, insecticides and drugs (sulphonamides, NSAIDs, antithyroids, etc).

Symptoms and signs: Symptoms of anaemia (due to a deficiency of RBCs) include bleeding tendencies, purpura, haematuria, epistaxis, ecchymosis, and gingival bleeding (due to thrombocytopenia). Susceptibility to infections (due to leucopoenia) is common. Headache and dyspnoea occur in a majority of patients.

Investigations: A FBC and estimation of erythropoietin (raised).

Management: Identification and removal of the cause, control of infection, bone marrow transplantation, haemopoietic stem cell transplantation and use of steroids are all used based on the severity of the condition.

- **Pernicious anaemia (macrocytic anaemia)**

Definition/Description: Pernicious anaemia is characterised by the failure of intrinsic factor secretion in the stomach (due to an autoimmune process), which is responsible for the absorption of vitamin B_{12} (cobalamin).

Cause: An autoimmune disorder causes pernicious anaemia, resulting in permanent atrophy of the gastric mucosa. Total gastrotomy can also cause this form of anaemia. A higher incidence of pernicious anaemia occurs in individuals with blood group A.

Symptoms and signs: General symptoms and signs of anaemia include neurological symptoms such as paraesthesia of the fingers and toes, and dementia. Glossitis, periodic diarrhoea, weight loss, and mild jaundice due to haemolysis are also common.

Investigations: These include FBC (macrocytic), serum B_{12} levels (low), detection of autoantibodies (parietal cells), and a gastric biopsy showing atrophic gastritis.

Management: This includes treatment with hydroxycobalamin, iron, potassium supplements, immunosuppressive drugs and blood transfusions.

Haemoglobinopathies

- **Sickle cell anaemia**

Definition/Description: Sickle cell anaemia is an inherited disorder. Red blood cells become sickle-shaped when blood experiences lower oxygen tension (as in an unpressurised aircraft, or during GA administration), decreased pH, or dehydration. These changes result in erythrocytosis, increased RBC adhesion and blood viscosity, and increased vascular occlusion.

Cause: Sickle cell anaemia is inherited by an autosomal recessive means.

Symptoms and signs: It is common in equatorial Africa and among those Africans who have migrated from that region to other parts of the world. Symptoms and signs include general symptoms of anaemia and lethargy, growth retardation, delayed puberty, increased susceptibility to infection, leg ulceration, and infarcts in the spleen, lungs, kidneys, bowel, bones, and fingers. Often these features are precipitated by dehydration, excessive cooling, or infection.

Investigations: These include a FBC, blood film showing sickle-shaped cells (evident in sickle cell anaemia and not in sickle cell trait) and haemoglobin electrophoresis.

Management: Precipitating factors such as dehydration, infections and excessive cooling need to be avoided. Treatment includes folic acid supplements, hydration, a warm climate, antibiotics, analgesics, and blood transfusions (the frequency of which should be minimal).

- **Thalassaemias**

Definition/Description: Thalassaemias are inherited disorders in which synthesis of one of the globin chains of the haemoglobin is either reduced or absent, resulting in haemolysis and anaemia.

Normally, haemoglobin is composed of four protein chains, two α and two β globin chains. In thalassemia, patients have defects in either the α or β globin chain, causing the production of abnormal red blood cells.

Cause: The thalassaemias are classified according to which chain of the haemoglobin molecule is affected. In α thalassaemia (also known as thalassaemia major), production of the α globin chain is affected, while in β thalassaemia (also known as thalassaemia minor) production of the β globin chain is affected.

Deletion of one of the α loci is more common in people of African or Asian descent, making them more likely to develop α thalassaemia. β thalassaemias are common in Africans, Greeks and Italians.

Symptoms and signs: These include severe anaemia, failure to thrive and early death. Those who survive show a mongoloid appearance of the head and face due to bone marrow hyperplasia. Leg ulcerations and hepatosplenomegaly may also occur in these patients.

Investigations: Haemoglobin electrophoresis shows increased HbA in minor, and raised HbF in major thalassemia. Radiographs of the skull and phalanges show increased bone marrow cavities and a 'hair-on-end 'appearance of the skull vault.

Management: Blood transfusions and iron chelating agents are administered to avoid iron overloading. A splenectomy may be conducted if hypersplenism is present.

Oral Manifestations and Dental Management Considerations

- In all forms of anaemia, the oral mucosa shows pallor.
- Atrophic glossitis is a feature of megaloblastic anaemia. The dorsum of the tongue is smooth, bald and red. There may be accompanying erythematous candidosis.
- In iron deficiency anaemia, the dorsum of the tongue appears bald and red. Patients often complain of a burning sensation. In Plummer-Vinson syndrome, oral ulcerations, erythroplakic patches or squamous cell carcinomas may occur. Pharyngeal involvement is also common in these patients.
- Patients with haemolytic anaemia may show mucosal pallor and jaundice due to haemolysis. Dental radiographs may show increased radiolucency with lamellar striations due to hyperplasia of the bone marrow in response to increased haemolysis.
- In sickle cell anaemia, increased widening, and a decreased number of trabaculations and signs of osteoporosis of the jaw bones may be evident on radiographs. In these patients trabaculae between the teeth appear horizontal, giving a 'step ladder' appearance, and lamina dura may be more distinct and dense.
- Delayed eruption of teeth and dental hypoplasia may be seen in patients with sickle cell anaemia.
- Bimaxillary protrusion and alveolar enlargement, giving rise to chipmunk facies, is a feature of thalassaemia major.
- Vaso-occlusive events may precipitate ischaemic necrosis in the bone.

Dental Management Considerations

- When classic symptoms and signs of anaemia exist, the patient should be sent for haematological screening tests.
- If anaemia is caused by any underlying systemic diseases (gastric carcinoma or peptic ulcer, for example) they must be identified early. Patients should be referred to a physician or specialist for diagnosis and treatment.
- There is no increased risk of dental complications requiring treatment precautions in patients with anaemia. In severe anaemia, the tendency for syncope should be considered.
- In aplastic anaemia, bleeding tendencies occur. A platelet count should be ordered prior to the commencement of any surgical procedure.
- In patients with aplastic anaemia, significant neutropenia may be present. In these patients the risk of infection is high. After surgical procedures, antibiotic cover is recommended for these patients.
- For any inhalational GA procedure, oxygenation is important for patients with aplastic anaemia and sickle cell anaemia.
- Patients with glucose-6-phosphate dehydrogenase deficiency anaemia, dental infections and drugs containing phenacetin may accentuate the rate of haemolysis. These patients may also show drug sensitivity to sulphonamides, aspirin, and chloramphenicol.
- In sickle cell anaemia, long and complicated dental procedures should be avoided.
- In children with sickle cell anaemia, dental and periodontal tissues should be periodically checked for infections. Any foci of infection should be eliminated in order to avoid precipitation of a crisis attack.
- In sickle cell anaemia, local anaesthetic can be used for routine dental procedures, but epinephrine should be avoided in the anaesthetic agent.
- If necessary, sedation with diazepam can be used in patients with sickle cell anaemia.
- Prophylactic antibiotics for surgical procedures are recommended for patients with sickle cell anaemia.
- Liberal use of salicylates, acetaminophen (Tylenol) and codeine for pain control should be avoided in patients with sickle cell anaemia.
- Nitrous oxide-oxygen can be used with care: 50% oxygen with a high flow rate is recommended for use with thalassaemia patients.

Bleeding and Clotting Disorders

- **Thrombocytopenia**

Definition/Description: A bleeding disorder due to a circulating platelet (thrombocytes) count of below 50,000 per microlitre (a normal count is 150000 to 450000 per microlitre).

Idiopathic thrombocytopenic purpura is a severe form of thrombocytopenia, probably due to an IgG antibody attack which follows a viral infection.

Cause: These include various diseases and conditions leading to decreased marrow production, decreased platelet survival, increased platelet consumption, platelet sequestration and platelet dilution. In the presence of autoimmune disorders, HIV disease, bone marrow tumours, leukaemia and lymphomas, thrombocytopenia can occur.

Symptoms and signs: Skin and mucous membranes show purpuric spots and ecchymoses, epistaxis, gastrointestinal bleeding, haematuria, headache and dizziness.

Oral Manifestations and Dental Management Considerations

- In thrombocytopenia, oral mucous membranes my show purpuric spots. Often this is accompanied by purpuric spots on the skin.
- Spontaneous gingival bleeding is also a feature of a severe form of thrombocytopenia (as in idiopathic thrombocytopenia of autoimmune cause).

Dental management considerations

- Surgical procedures should not be undertaken unless the haematologist is involved with the diagnosis. For extractions, the platelet count should be at least 50,000/mm^3 before the commencement of surgery. Transfusion of platelets may be required in some cases.
- In children with idiopathic thrombocytopenia, prednisone (4 mg/kg/day for 1 week orally) is recommended. This will increase the platelet count within 48 hours.
- If the patient is on long term steroid therapy, the dose of steroids needs to be doubled prior to the commencement of an invasive procedure.

Investigations: These include a FBC, WBC, detection of antibodies to platelets, bone marrow studies, bleeding time, platelet count, platelet adhesion and aggregation studies, and coagulation screen (to rule out factor deficiency syndromes). A simple test is the estimation of bleeding time, which can be performed in the clinic.

Management: Steroids, splenectomy and immunosuppressive drugs are used in the treatment of thrombocytopenia.

- **The Haemophiliac disorders (haemophilia A, B and von Willebrandt's disease)**

The "haemophilias" are a group of genetic disorders resulting in deficiency of one of the coagulation pathway factors. This group consists of three conditions: haemophilia A, haemophilia B (Christmas disease) and Von Willebrand's disease.

Haemophilia A

Definition/Description: An inherited X–linked recessive disorder characterised by a deficiency of Factor VIII. This coagulation disorder affects males and is carried by females.

Cause: Genetic. The defective gene is located on the X chromosome (F8 gene). An affected male will not transmit the disorder to his sons, but all of his daughters will be carriers

of the trait because they inherit his X chromosome. A female carrier will transmit the condition to half of her sons and the carrier state to half of her daughters.

Symptoms and signs: Excessive bruising and haemarthroses are evident from very early in childhood. Swelling, pain and eventual deformity of the joints are common. Internal bleeding may occur. Spontaneous bleeding from oral soft tissues may occur in the severe form of the disorder. Excessive bleeding from trauma or surgery is common in these patients.

Investigations: Clinical history is suggestive. Screening tests include a prolonged partial thromboplastin test, (prolonged), bleeding time (normal), platelet count (normal) and specific tests for missing factors (Factor VIII assay).

Management includes the repeated administration of freeze dried or recombinant factor VIII concentrate, oral antifibrinolytics (tranexamic acid or desmopressin, for example), and plasmapheresis (A procedure whereby blood plasma components are removed, treated, and returned to circulation).

Haemophilia B (Christmas disease)

Factor IX is defective or deficient in haemophilia B. While less common than haemophilia A, clinical features are identical. Detection of defective or deficient Factor IX is diagnostic. Administration of purified factor IX products is required by these patients.

Von Willebrand's disease

This is an autosomal dominant inherited disorder characterised by defective platelet function and a deficiency or abnormality of factor VIII.

Symptoms and signs include mucocutaneous bleeding and haemarthrosis.

Investigations: Family history, bleeding time (normal), platelet count (normal), platelet function tests (poor aggregation adhesion), and a factor VIII deficiency test are required.

Management: Administration of factor VIII concentrate.

Oral Manifestations and Dental Management Considerations

- The level of deficiency of factor VIII is to be determined with the treating physician before invasive dental therapy.
- If multiple extractions are required, splints should be designed prior to surgical procedures taking place.
- Oral infections should be treated before surgical intervention.
- Prophylactic antibiotics prior to surgical procedures may be necessary for some patients.
- Postoperative antibiotic cover is indicated.
- Operative trauma to the tissues should be minimal.
- The majority of haemophiliac patients need to be hospitalised.
- Missing factor needs to be administered intravenously prior to the surgical procedure.
- There are no contraindications to the use of local anaesthetic.
- Aspirin or NSAIDs should be avoided.

Other Related Topics

Leukaemia

Definition/Description: A haematological malignancy characterised by the uncontrolled proliferation of (malignant) white blood cells derived from one of the haematopoietic precursor cells, resulting in replacement of the normal bone marrow.

Two major types of leukaemia exist: acute and chronic. Acute leukaemia is common in children. It is characterised by a rapid increase in the number of immature white blood cells. The chronic type is common in the elderly. Chronic leukaemia is characterised by the excessive and slow buildup of relatively mature, but still abnormal, white blood cells.

Acute and chronic leukaemias are further subdivided according to the type of white blood cells affected. This division includes two types, namely (acute and chronic) 1. lymphoblastic or lymphocytic leukaemias, and (acute and chronic) 2. myeloid or myelogenous leukaemias.

Causes: Causes of leukaemia largely remain unknown. Large doses of radiation, exposure to chemicals and infection with Epstein-Barr virus (EBV) and human lymphotropic virus (HTLV-1) are suspects. Electromagnetic fields have also been implicated as possible risk factors.

1. Acute Lymphoblastic leukaemia (ALL)

Definition/Description: ALL is common in children and in the elderly. It is a B-lymphocyte neoplasm. Malignant cells proliferate and infiltrate bone marrow as a result of which granulocytopenia, thrombocytopenia and anaemia result. Malignant cells also infiltrate viscera, skin and brain.

Cause: Implicated environmental causes include infections with Epstein-Barr virus (EBV) and human T-lymphotropic virus (HTLV) infections. Genetic factors may also play an important role in the causation. Philadelphia chromosome (a shortened chromosome) is present in 25% of adults and 5% of children with ALL. ALL is more common in Down's syndrome patients.

Symptoms and signs: These include flu–like symptoms, generalised lymphadenopathy, symptoms and signs of anaemia, bruising and bleeding tendencies (petechiae and ecchymoses), splenomegaly and hepatomegaly. CNS involvement may result in cranial nerve palsies.

Investigations include a FBC, peripheral blood smear, bone marrow studies (leukaemic blast cells), estimation of haemoglobin, platelets and haematocrit (these are depressed). Immunotyping and flow cytometry to assess cell maturation are also used.

Management: Management protocols include intensive combinations of chemotherapy, and bone marrow transplantation (BMT) for adult patients younger than 45 years of age and for children. For CNS relapse, prophylactic radiotherapy is given.

2. Chronic lymphocytic leukaemia (CLL)

Definition/Description: A chronic form of leukaemia involving mature clonal CD5 B-lymphocytes. This is the most common type of leukaemia in adults.

Cause: Unknown. Familial inheritance is a risk factor.

Symptoms and signs: Asymptomatic at presentation. When symptomatic, symptoms and signs include fatigue, anorexia and weight loss. As the disease advances, signs of anaemia, abdominal pain, thrombocytopenia, splenomegaly, lymphadenopathy and hepatomegaly are noted.

Investigations include peripheral blood smear and immunotyping.

Management: Chemotherapy. Anaemia and infections also need to be treated.

3. Acute myeloid (myelogenous) leukaemia (AML)

Definition/Description: Neoplasm of myeloid (immature) white blood cells resulting in uncontrolled proliferation in the bone marrow. These cells appear in the peripheral circulation.

Cause: Specific causes are unknown. Risk factors include radiation exposure, chemotherapy (therapy related), and exposure to chemicals (tobacco smoke and benzene are examples).

Symptoms and signs: These start with flu-like symptoms. Fatigue, easy bruising, bone pain, malaise, pallor, and dyspnoea on exertion are common. Other features include petechiae and ecchymoses in the skin and mucous membranes. Delayed healing, infections, and enlargement of tonsils, lymph nodes, spleen and gingival tissues are noted. CNS involvement due to infiltration of neoplastic white blood cells occurs in 35% of cases.

Investigations: These include a FBC, peripheral blood smear, bone marrow studies, cytochemical and immunophenotyping, and cytogenetic analysis. Presence of myeloblasts in the peripheral blood (or bone marrow) at a rate of 20% is diagnostic of AML.

Management: An aggressive combination of chemotherapy, allogenic bone marrow transplant, nutritional supplements, and the control of infections and complications of chemotherapy are included in the management protocol of AML.

4. Chronic myeloid (myelogenous) leukaemia (CML)

Definition/Description: This type of leukaemia involves the neoplasm of mature (differentiated) myeloid cell lines. CML is less aggressive than AML.

Cause: Unknown. Exposure to radiation is a risk factor. A shortened chromosome 22 (Philadelphia chromosome) is seen in 90% of cases.

Symptoms and signs: Many patients are asymptomatic at presentation. When symptomatic, symptoms and signs include lassitude, myalgia, arthralgia, epistaxis, weight loss, gout, sweating, recurrent infections and massive splenomegaly.

Investigations: Usually included are a WBC count (significantly raised with basophilia and eosinophilia), cytogenetic studies, (revealing Philadelphia chromosome), serum chemistry revealing elevated lactate dehydrogenase (LDH) and low levels of alkaline phosphatase. Bone marrow studies reveal a markedly hypercellular marrow.

Management: Interferon is used to control elevated WBCs. Allogenic bone marrow transplant is also used in the treatment protocol.

Oral Manifestations and Dental Management Considerations

- Patients with acute or chronic leukaemia may reveal ulceration of the mucosa, gingival enlargement (due to leukaemic infiltrate), petechiae and ecchymosis of the mucous membrane. Gingival enlargement is common in myelogenous leukaemia.
- Lymhadenopathy is common in leukaemic patients.
- Acute oral infections are common in leukaemic patients because of their increased susceptibility to infections. These infections must be promptly treated and their recurrence prevented.
- Aspirin or NSAIDs are to be avoided in these patients.
- Patients with chronic leukaemia should have their platelet status and bleeding time checked before the commencement of dental invasive procedures. If their platelet count is below 50,000 platelets/mm^3 and bleeding time is >6 minutes, surgery should be postponed until the platelet count and bleeding time are corrected.
- Any surgical procedure should be carried out in consultation with the oncologist.
- Topical or systemic antibiotics may be necessary before the commencement of procedures involving scaling and prophylaxis.

- **Multiple myeloma (MM)**

Definition/description: Multiple myeloma is a malignant disorder of abnormal immunoglobulin-secreting plasma cells. Disease is characterised by invasion and destruction of bone.

Cause: The cause of myeloma is unknown. Suggested factors include chromosomal and genetic factors, radiation and exposure to chemicals.

Symptoms and signs: Weight loss, fatigue, persistent bone pain (in the back and thorax in particular, and also at night or at rest), recurring bacterial infections, signs of renal failure, pathologic bone fractures, spinal cord compression and paraplegia are salient features of MM. Signs of anaemia are persistent and are often the reason for medical consultation. Peripheral neuropathy, abnormal bleeding, carpal tunnel syndrome, polyuria and polydipsia are common.

Oral Manifestations and Dental Management Considerations

Oral manifestations of MM include enlargement of the tongue due to amyloid deposition. Oral symptoms may include paraesthesia and pain of the jaw bones. Radiographs of the jaw bones or the skull show characteristic “punched out” radiolucencies. These may be single or multiple.

Dental management considerations include maintenance of good oral hygiene before and during chemotherapy. Due to bleeding tendencies and susceptibility to infections, invasive treatment needs to be postponed until the patient is medically stable. Those on bisphosphonate therapy should be advised of the adverse effects (such as osteonecrosis) of the medication.

Diagnosis and investigations: Symptoms of bone pain accompanied by symptoms and signs of anaemia are early manifestations of the disease. Laboratory findings show elevated blood and urine protein levels, hypercalcaemia, renal insufficiency and anaemia. Serum protein electrophoresis detecting M-protein, x-rays of bones showing osteolytic defects, bone marrow examination detecting abnormal light immunoglobulin chains, and detection of light chain immunoglobin proteins (called Bence-Jones proteins) in urine are diagnostic.

Management: Conventional chemotherapy with oral melphalan, alkylating agents, corticosteroids, and thalidomide are the therapeutic agents used. Autologous peripheral blood stem cell transplantation is also used in MM management. Bisphosphonate therapy is required to control osteolysis. Prognosis of MM is generally poor.

Lymphoma

Introduction

Lymphomas are a heterogeneous group of neoplasms arising from the reticuloendothelial and lymphatic systems. Major types are Hodgkin's lymphoma and non-Hodgkin's lymphoma.

- **Hodgkin's lymphoma**

Definition/description: Hodgkin's lymphoma is a localised or disseminated malignant proliferation of cells of the lymphoreticular system. This malignancy primarily involves the lymph nodes, spleen, liver and bone marrow.

Cause: The cause of Hodgkin's lymphoma is not known but genetic susceptibility and association with environmental factors have been identified as playing a role in its causation. Environmental factors include a history of treatment with phenytoin, radiation therapy, chemotherapy, Epstein-Barr virus infections, HIV disease, herpes virus type 6 (HHV-6) and tuberculosis.

Symptoms and signs: These include painless cervical lymphadenopathy, fever, night sweats, weight loss, pruritus, splenomegaly and hepatomegaly. A finding that pain may occur in the diseased regions immediately after drinking alcohol offers a diagnostic clue. Cachexia is common in advanced cases.

Diagnosis and investigations: Often Hodgkin's lymphoma is detected on routine chest x-rays showing a mediastinal adenopathy. Painless lymphadenopathy is one of the major signs. X-rays can be followed by torso CT or PET scans. The lymph node biopsy that follows shows large binucleated cells, called Reid-Sternberg cells, mixed with numerous histiocytes, lymphocytes, plasma cells, eosinophils and monocytes.

Blood tests include a complete blood count showing polymorphonuclear leukocytosis, and raised ESR, alkaline phosphatase and serum heptoglobin levels.

Management: Chemotherapy plus radiation therapy are recommended for patients with Hodgkin's lymphoma. Complications of chemotherapy may pose an increased risk of the development of leukaemia. With combination therapy, the risk of developing breast, GI and lung cancers increases. After therapy, survival without a relapse of the disease is considered a cure.

- **Non-Hodgkin's lymphoma**

Definition/description: Non-Hodgkin's lymphomas (NHL) are a heterogeneous group of disorders involving malignant proliferation of lymphoid cells in lymphoreticular sites. Sites of involvement include lymph nodes, bone marrow, the spleen, liver and gastrointestinal tract. NHL is more common than Hodgkin's lymphoma. NHL originates from B-lymphocytes in about 85% of cases. The remainder arise from T-cells or natural killer cells.

Cause: The cause is unknown. A viral association has been suggested, including human T-cell leukaemia–lymphoma virus, Epstein-Barr virus, and human immunodeficiency virus. Risk factors include HIV/AIDS, rheumatoid arthritis, Sjögren's syndrome, *H. pylori* infections and exposure to certain chemicals.

Symptoms and signs: Peripheral asymptomatic lymphadenopathy is a common finding of NHL. Lymph nodes are rubbery, discrete and in the later stages, matted. Multiple areas of involvement may occur. Extra nodal sites which include the GI tract may simulate GIT carcinomas. Malabsorption and neurological symptoms are common in HIV patients with NHL. In patients with abdominal or thoracic involvement, due to lymphatic obstruction, ascites and pleural effusion may be present. In some patients, skin and bones may be involved in the early stages.

Weight loss, fever and night sweats are indicative of the disseminated form of NHL. Splenomegaly and hepatomegaly also occur in NHL. Congestion and oedema of the face and neck is a feature of NHL. These effects are due to pressure on the superior vena cava.

Diagnosis/investigations: Painless lymphadenopthy or the presence of mediastinal adenopathy seen on routine chest x-rays is suggestive of NHL. CT and PET scans, and lymph node or bone marrow biopsies are confirmatory tests. Histological evidence points to the destruction of the normal architecture of the lymph node including invasion of the capsule by neoplastic cells.

Management: Treatment includes radiation and/or chemotherapy with stem cell transplantation.

- **Burkitt's lymphoma**

Definition/description: Burkitt's lymphoma (BL) is a B-cell lymphoma occurring primarily in children in endemic (African) and sporadic (non-African) forms. In the endemic form, jaw bone involvement predominates whereas in the sporadic form, abdominal disease is common. The liver, spleen and kidneys may also be involved in non-African form of BL.

Cause: In the endemic form, Epstein-Barr virus has been shown to be etiologically associated with the disease. A genetic predisposition has been reported in these patients.

Diagnosis and investigations: Tests include a biopsy of the lymph node or any other suspected site. Histology reveals cells with a high mitotic rate and monoclonal proliferation of B-cells, giving a characteristic "starry-sky" pattern of benign macrophages which have engulfed malignant lymphocytes. CT and PET scans are other useful tools.

Management: CODOX-M/IVAC (a combination of several medications including cyclophosphomide, vincristine, doxorubicin, methotrexate, ifosfamide, etoposide, cytarbine) has been reported to be a highly successful regimen for BL both in children and adults. A cure rate of 90% has been reported with this therapy.

Hodgkin's lymphoma (HL): There are no specific oral manifestations in Hodgkin's lymphoma.

Non-Hodgkin's lymphoma (NHL): Oral manifestations of non-Hodgkin's lymphoma include Waldeyer's tonsillar ring, and it can also affect the oropharynx and soft palate. Salivary glands and mandible may also be involved in NHL. In NHL involvement of the gingiva, palate, alveolar ridge, buccal sulcus and floor of the mouth have also been reported. In NHL petechiae, fungal infections, viral infections, mucosal ulcers and oral paresthesia are common.

Patients receiving radiation therapy for head and neck lymphoma exhibit mucositis, xerostomia and susceptibility to fungal infections.

Burkitt's lymphoma (BL): Jaw lesions show extensive osteolytic changes. The earliest sign is loosening of the teeth. Teeth float in the tumour mass**.** Expansion of the jaw and protrusion of the tumour mass into the mouth is rapid. Bilateral or involvement of all four quadrants of the jaws by the tumour may occur. Early radiographic signs show a loss of lamina dura and enlargement of the crypts of developing teeth in children. Focal areas of radiolucency and the displacement of teeth are also common findings on the extraoral radiographic views. Large radiolucent defects are seen in advanced cases.

- **Lymphadenopathy**

Lymphadenopathy refers to lymph node enlargement. This is a common presenting condition. Lymphadenopathy may be localised or generalised. Cervical lymphadenopathy confined to the neck region is commonly encountered in dental and medical practices. A careful history and thorough clinical examination offer useful information on the nature and causes of lympahdenopathy. Often patients are unaware of the presence of enlarged lymph nodes. Lymphadenopathy may be detected incidentally on clinical examination. Lymph nodes larger than 1 cm in diameter should be considered abnormal and investigated further. It must be remembered that not all neck lumps are examples of lympahdenopathy. Lymphadenopathy can be a symptom or a sign, and not a diagnosis.

In adults, cervical lymph nodes are not normally palpable. In healthy children up to the age of 12 year of age, cervical lymph nodes may be palpable.

Causes of lymphadenopathy are varied. They may be inflammatory (lymphadenitis), degenerative or neoplastic. Nodes which heal by scarring may also remain palpable. These should not be considered as abnormal.

Minor lymph node enlargements are common in children with upper respiratory infections. Inguinal lymph nodes in many healthy adults may also be palpable.

The clinician should check the lymph nodes carefully in patients with:

- A history of weight loss for no obvious reasons
- Unexplained fever
- Unexplained anaemia
- Bruising tendencies
- Jaundice
- An abnormal mass

- Finger clubbing
- Suspected breast cancer
- Suspected oral or pharyngeal cancer
- A history of HIV disease
- Tuberculosis
- Sarcoidosis

Causes of lymphadenopathy are listed as follows:

- **Due to nonspecific or secondary infections involving oro-pharyngeal tissues**
  - Pericoronitis
  - Dento-alveolar abscess
  - Tonsillitis
  - Pharyngitis
- **Due to viral infections:**
  - Infectious mononucleosis
  - Rubella
  - HIV
  - Cytomegalovirus infections
  - Hepatitis A
  - Measles
  - Herpetic gingivostomatitis
- **Due to bacterial infections:**
  - Acute necrotising ulcerative gingivitis
  - Actinomycosis
  - Tuberculosis
  - Syphilis
  - Cat scratch disease
- **Due to fungal infections:**
  - Histoplasmosis
- **Due to protozoal diseases:**
  - Malaria
  - Toxoplasmosis
- **Due to neoplastic diseases:**
  - Acute lymphoblastic leukaemia
  - Chronic lymphatic leukaemia
  - Hodgkin’s lymphoma
  - Non-Hodgkin’s lymphoma
  - Myeloproliferative disorders
  - Metastasis from oro-pharyngeal cancers
- **Due to immune disorders:**
  - Systemic lupus erythematosus
  - Rheumatoid arthritis
- **Due to other causes:**
  - Sarcoidosis

- Drugs (phenytoin)
- Histiocytosis X

Evaluation of Lymphadenopathy

History: Local infections are usually symptomatic such as a dental abscess with cervical lymphadenopathy. A new swelling of a few days duration following symptoms of upper respiratory infections or pharyngitis suggests benign reactive lymphadenopathy. Pain in the lymph node suggests inflammation. A painless mass may be due to a tumour or cyst. A history of fever, lethargy and malaise suggest infections. A history of alcohol and tobacco consumption, ill-fitting dentures or appliances, and trauma from sharp cusps of teeth should be obtained in order to determine the cause of cervical lymphadenitis.

Examination includes the following:

- The scalp, face, ear, nasal cavities, oral cavity, nasopharynx, oropharynx, hypopharynx and larynx should be closely inspected.
- The neck (for any mass), base of the tongue, salivary glands and thyroid should be palpated.
- A hard, non-tender and fixed lymph node suggests malignancy.
- Generalised lymphadenopathy and splenomegaly suggests glandular fever (infectious mononucleosis) or a lymphoreticular malignancy (leukaemia or lymphoma).
- Nodes that are enlarged, firm, and matted or rubbery in consistency suggest leukaemia.
- Tuberculous lymphadenitis (scrofula) is characterised by painless lymph node enlargement in the neck that does not show signs of inflammation (cold abscess).
- Finger clubbing in a patient with lymphadenopathy suggests respiratory disorders (complicated TB, for example).
- Often, checking for lymphadenopathy of other sites (axillary, inguinal, popliteal and epitrochlear nodes, for example) and for hepatomegaly and splenomegaly becomes necessary.

Diagnosis/investigations include the following:

- Every patient with palpable lymphadenopathy without an obvious cause should have a complete blood count (CBC), erythrocyte sedimentation rate (ESR) and a chest x-ray.
- Other investigations include swabs or smears for local infections, a Mantoux test for TB, clotting screen for blood dyscrasias, blood film for leukaemia, liver function tests (LFT) for malignant infiltration of the liver, a chest x-ray of the hilar glands in sarcoidosis and TB, an antibody screen for SLE, and a VDRL for syphilis.
- If examination reveals an oral or pharyngeal lesion that fails to resolve within two to three weeks (non-healing ulcer, for example), testing should include a chest x-ray, CT scan, MRI, and a needle and/or incisional biopsy.

Chapter 14

Disorders of the Immune System

S. R. Prabhu

Abstract

This chapter deals with common immunological disorders of dental interest. The disease entities discussed include hypersensitivity reactions, anaphylaxis, allergic contact dermatitis/allergic contact mucositis, angioneurotic oedema, urticaria, Sjögren's syndrome, systemic lupus erythematosus (SLE), giant cell arteritis, Wagener's granulomatosis, Reiter's syndrome, Behcet's disease and HIV disease. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of these immunological diseases are also described.

Introduction

Diseases of the immune system can be grouped as hypersensitivity reactions, autoimmune diseases and immunodeficiency diseases. Some of these disorders are common and of importance to practicing dentists.

Common Symptoms of Immune Disorders

There are a wide range of symptoms associated with diseases of the immune system. Symptoms can vary depending on the type of immune response involved. Some of these include allergic manifestations such as hives, rashes, vesicles, patches, ulcers, blood blisters, anaphylactic reactions, swelling of the lips, breathlessness, and wheezing. Symptoms of autoimmune disorders may include fever, cough, haemoptysis, haematuria, diarrhoea, neurological symptoms, joint pain, a dry mouth and dry eyes.

Common Investigations in Immune Disorders

Investigations employed in diseases of the immune system include patch testing, immunofluorescence techniques, radioimmunoassay (RIA), polymerase chain reactions (PCR), enzyme immunoassay (EIA), immunoelectroblot assay, detection of autoantibodies, and biopsy for histopathology.

Common Immune Disorders of Dental Interest

1. Hypersensitivity reactions

Hypersensitivity reactions are characterised by hyper function of the immune system resulting in allergic manifestations of varying severity. Systemic manifestation resulting in anaphylaxis is a life threatening condition whereas those of the skin and mucous membranes, known as contact dermatitis (contact mucositis), are less dangerous. Transplantation rejection is also an important aspect of the immune response to grafts.

- **Anaphylaxis**

Definition/Description: A severe, life threatening allergic (type 1 hypersensitivity) response to agents such as food, medications and insect stings.

Causes include the following:

- Food stuffs, including shell fish, eggs, peanuts, soy, wheat and milk
- Medications such as penicillins, aspirin, NSAIDS, tetracycline, local anaesthetics and vaccines
- Venom from insect bites from bees and wasps
- Other causes include x-ray contrast medium (iodine content), latex, food additives, food colours, physical exercise and extremes of weather

Symptoms and signs including oral manifestations: Symptoms occur within minutes; between 5 and 20 minutes if the allergen is injected, and within 2 hours if ingested. Signs include any of the following:

- Skin hives, itchiness, or swelling of the lips, conjunctiva, throat or tongue
- A runny nose, shortness of breath, wheeze, or stridor, cough, hoarseness of voice or painful swallowing
- Diarrhoea, vomiting, abdominal pain, low blood pressure, loss of bladder control, headache, confusion and fear, dysrhythmias and cardiac arrest.

Investigations: Clinical features are suggestive of an anaphylactic attack. Blood tests for histamine (or tryptase) released from mast cells during an attack can be useful in determining an anaphylactic reaction from insect bites. Skin patch testing for skin reactions is useful for certain food products, venoms and for penicillins.

Management: An anaphylactic attack is a medical emergency. Resuscitation, oxygen, intravenous fluids, and administration of epinephrine are important measures. Corticosteroids and anthistamines may also be used as adjuncts. Hospitalisation of the patient is essential in anaphylactic attack.

Prevention of allergies by avoiding known triggers is the key. Immunotherapy in desensitising patients to allergies from insect bites is also recommended.

- **Allergic contact dermatitis (ACD) and allergic contact mucositis**

Definition/Description: Allergic contact dermatitis (allergic contact mucositis when mucosa is involved) is an inflammatory condition of the skin which requires sensitisation to an antigen and is termed a Type IV delayed hypersensitivity reaction involving a cell-mediated allergic response. (This is to be differentiated from contact dermatitis which results from direct skin contact with an external agent without involving immunologic mechanisms). Immunologic events in allergic contact dermatitis require interaction among antigen, antigen presenting cells (Langerhans' cells) in the skin, and lymphocytes.

Cause: The majority of contact allergens are of plant origin: poison ivy, poison oak, the shells of cashew nuts, and resin from the Japanese lacquer tree, the skin of mangoes, primroses and chrysanthemums. Other allergens include nickel, gold, chromium, neomycin, cosmetic products, insecticides, soaps, household cleaners, hair dye, photographic developers, shampoos and conditioners.

Symptoms and signs: Itching, redness and vesiculation of the skin at the site of exposure within 12 hours to 2 days after contact with the allergen (in contact dermatitis, lesions appear soon after exposure to an irritant). Depending on the type of allergen involved, the lesions can ooze, drain and crust, or become scaled, raw or thickened. Those who develop allergic contact dermatitis to a known allergen will continue to present skin manifestations on subsequent contacts throughout life.

Investigations: History and physical examination. Skin patch testing is useful in identifying the contact allergen involved.

Management: Skin creams containing corticosteroids for mild reactions, and systemic steroids in severe cases, are effective. Avoidance of known allergens is the key to a prevention strategy.

Oral and Maxillofacial Manifestations and Dental Management Considerations

- Allergic contact dermatitis involving mucosa can be termed as allergic mucositis
- Allergic mucositis can manifest as lichenoid patches of the mucosa, or as swelling of the tongue or lips due to inflammatory oedema.
- Allergies to lip stick, tooth paste, food items, chewing gums containing artificial cinnamon, and spices have been reported to cause allergic contact mucositis
- When allergic manifestations occur during dental procedures, such treatment should be immediately terminated
- Steroids are useful in treating allergic manifestations

- Diagnosis of allergic mucositis can be done with patch testing.

- **Angioedema (angioneurotic oedema)**

Definition/description: Angioedema (formerly known as angioneurotic oedema) is characterised by the rapid swelling of the dermis subcutaneous tissue, mucosa and submucosal tissues. Angioedema is classified as either acquired or hereditary. **Acquired angioedema** can be immunologic, non-immunologic or idiopathic. It is usually caused by allergy and may occur together with other allergic symptoms and urticaria. It can also occur as a side effect to certain medications, particularly ACE inhibitors (non-allergic type). **Hereditary angioedema** (HAE), on the other hand, is caused by a genetic mutation inherited in an autosomal dominant form.

Cause: Acquired angioedema is caused by an allergic type 1 reaction which may be induced by foods (nuts, for example) and drugs (such as antibiotics or aspirin). Other causes may also be responsible for the acquired type of angioedema. Drug induced non-allergic angioedema can result from angiotensin converting enzyme (ACE) inhibitors. The hereditary type has a genetic background.

Symptoms and Signs Including Oral Manifestations

- Clinical features of **allergic angioedema** include acute and pronounced labial and periorbital swelling. Swelling can also extend to the tongue, pharynx and neck, causing fatal respiratory obstruction.
- Symptoms of **drug induced non-allergic angioedema** include swelling of the lips and this may extend to the tongue.
- Symptoms of **hereditary angioedema** include swelling of the lips, face, mouth, tongue, neck region, extremities and the gastrointestinal tract. Precipitating events for the hereditary type include a blunt injury and sometimes, emotional stress.

Diagnosis/investigations: History and clinical examination are suggestive. Symptoms are life threatening and there is often no time for detailed investigations. Patch testing may be of use.

Management: Angioedema is a medical emergency. It should be treated immediately. When a threat to life exists, an intramuscular adrenaline injection and the systemic use of corticosteroids and antihistamines are necessary. Mild reactions respond to the use of antihistamines (chlorpheniramine, for example).

For the hereditary type of angioedema, CI-INH replacement, fresh plasma, plasminogen inhibitors (tranexamic acid, for example) or androgenic steroids are effective.

- **Urticaria**

Definition/description: Urticaria is a skin disorder characterised by migratory erythematous, pruritic plaques. Two types of urticaria occur: acute and chronic forms.

Cause: Acute urticaria is caused by the release of histamines representing a type 1 hypersensitivity response to several topical or systemic substances. Aspirin, NSAIDs, ACE inhibitors, sun light, cold and friction also can cause acute urticaria by triggering the release of histamine. Most cases of chronic urticaria are idiopathic.

Symptoms and signs: Raised areas surrounded by a red base (weals) and hives are the hallmarks of urticaria. Lesions are itchy. Weals that persist for more than six weeks are considered to be examples of chronic urticaria.

D**iagnosis/investigations:** History and clinical examination offer important clues to diagnosis. No special investigations are generally required. Patch testing for allergens may be useful.

Management: Any identified cause should be eliminated. Implicated food or drugs should be avoided. Antihistamines are the mainstay of treatment. In severe attacks, systemic corticosteroids are effective.

2. Autoimmune disorders

Autoimmune disorders are characterised by the failure of the immune system to recognise 'self' from 'non-self' and cause clinical manifestations by forming autoantibodies against one's own tissue antigens. Some autoimmune diseases are organ specific whilst others involve multiple body systems.

- **Sjögren's syndrome**

Definition/Description: Sjögren's syndrome is a chronic autoimmune disorder in which the acinar tissue of exocrine glands (salivary and lacrimal glands in particular) is replaced and destroyed by lymphocytic infiltrate, causing dry eyes and a dry mouth. This is called **primary Sjögren's syndrome or SS-1**.

A triad of dry mouth, dry eyes and a connective tissue disorder (rheumatoid arthritis, for example) is called **secondary Sjögren's syndrome**. Involvement of other exocrine glands may cause nasal dryness, tracheitis, pancreatitis and vaginal dryness.

Cause: An autoimmune process. Genetic predisposition may exist

Symptoms and signs: In primary Sjögren's syndrome, symptoms include dryness of all mucosal sites, predominantly the eyes (keratoconjunctivitis sicca) and mouth (xerostomia). **Oral manifestations** include disturbances in taste sensation, a fissured tongue, candidal infections and extensive dental decay due to xerostomia. A risk of developing lymphoma of the parotid glands exists at a later stage of the primary disease.

In secondary Sjogren's syndrome, in addition to the dryness of the mucosal surfaces, joints (rheumatoid arthritis), kidneys (interstitial nephritis), blood vessels (vasculitis), lungs (bronchitis), liver, pancreas, thyroid glands, peripheral nervous system (carpal tunnel syndrome and peripheral neuropathy) may be involved.

Investigations include:

- Shirmer's test to measure the production of tears
- Measurement of non-stimulated whole saliva flow
- Slit lamp examination of the eyes to test dryness on the surface of the eyes
- A lip biopsy to assess lymphocytic infiltration of the minor salivary glands

- Ultrasound of the salivary glands
- Radiographs of the parotid glands using contrast medium (snow storm appearance in SS positive cases)
- Serum antibodies, especially antinuclear antibodies (ANA) SS-A or SSB. Hypergammaglobulinaemia as a result of rheumatoid factor (RF) may be present.

Management: There is no cure. Symptomatic and supportive treatments are recommended. Artificial tears for dry eyes and artificial saliva for xerostomia, sialogogues (pilocarpine) to stimulate salivary flow, corticosteroids (prednisolone) or immunosuppressants (methotrexate) for complications, antirheumatic agents, NSAIDS for musculoskeletal symptoms, and frequent sips of water to keep the mouth hydrated, are effective.

- **Systemic lupus erythematosus (SLE)**

Definition/description: An autoimmune disorder characterised by the presence of non-organ specific antibodies, SLE predominantly affects females.

Cause: Unknown. Sometimes a variety of drugs may precipitate the condition.

Symptoms and signs including oral manifestations: SLE is a multisystem disorder involving the skin, musculoskeletal system, renal system, nervous system, cardiovascular system and respiratory system. Symptoms include joint pain (arthritis), myalgia, photosensitive skin rash on the face (malar 'butterfly rash'), Raynaud's phenomena, vasculitis, purpura, oral white patches or ulcers, glomerulonephritis, peripheral neuropathy, cranial nerve palsies, seizures, hemiparesis, pancreatitis, abdominal pain, haemolytic anaemia, jaundice, abnormal liver function tests, splenomegaly and lymphadenopathy. General symptoms include fever, malaise and lethargy.

Investigations include a full blood count for anaemia, leukopenia and thrombocytopenia, INR (prolonged) , ESR (elevated), albumin (low), fibrinogen (raised), urea and creatinine (raised) in renal involvement, antinuclear antibodies (ANA) positive in 95% of cases, rheumatoid factor (positive in 25-50% of cases), skin or renal biopsy, and the presence of LE cells is confirmatory.

Management: Steroids and an immunosuppressant (azathioprine) help mild to moderately affected patients. For severe cases involving the kidneys and CNS, chemotherapeutic agents (cyclophosphamide) and steroids are necessary.

- **Giant cell arteritis (Temporal arteritis)**

Definition/description: Also known as temporal arteritis, giant cell arteritis (GCA) is an autoimmune disorder involving the large arteries of the head and neck (the temporal artery in particular) in the elderly, and associated with polymyalgia rheumatica.

Cause: Not known. Cellular and humoral immunological systems are implicated.

Symptoms and signs including oral manifestations: A disease of elderly Caucasian females in the majority of cases. Features include scalp tenderness, headaches, pulseless temporal arteries, ulcers on the scalp, visual disturbances including blindness in advanced cases, and thickened, tender temporal arteries. If basilar artery occlusion occurs GCA is fatal.

Rarely, lingual and facial arteries may be involved. In these patients, paraesthesia of the tongue and pain at the level of the gums are reported.

Investigation: ESR is elevated. C-reactive protein is elevated in the active disease.

Management: Systemic corticosteroids are effective. Advanced cases may need immunosuppressants such as methotrexate or azathioprine.

- **Wagener's granulomatosis**

Definition/Description: Wagener's granulomatosis is an autoimmune disorder causing granulomatous vasculitis of small arteries. The current terminology for this disorder is granulomatosis with polyangitis.

Cause: Unknown. There is a familial predisposition in persons with previous viral or bacterial infections.

Symptoms and signs:

- General symptoms first include malaise, fever, arthralgia, and rhinitis Nasal and oral manifestations include bloody nasal discharge, a depressed nasal bridge, ulcers on the palate and pharynx (painless or painful), strawberry gingivitis, underlying bone destruction with loosening of the teeth, non-specific ulcerations throughout oral mucosa.
- Pulmonary symptoms and signs include cough with haemoptysis, chest pain, dyspnoea and pleural effusion
- Abnormal chest x-rays may show nodules, infiltrates or cavities
- Kidney involvement shows urinary sediment with microhaematuria or red cell casts
- Nervous system involvement results in peripheral neuritis
- Eye involvement results in uveitis, scleritis and retinal vasculitis
- Skin manifestations show purpura and skin rash
- Ear involvement results in hearing loss

Investigations: FBC, (WBC increased, Hb low), C-reactive protein (raised), ESR(raised), antineutrophil cytoplasmic antibody (cANCA positive), urine positive for blood and protein, chest x-ray (nodules), biopsy of nasal mucosa, lungs or kidneys for vasculitis.

Management: Immunosuppressants with corticosteroids are effective. Plasma exchange or intravenous gammaglobulin may also be necessary in some advanced cases. The 5 year survival rate is 87% for those on treatment.

- **Reiter's syndrome (Reiter's arthritis)**

Definition/Description: An autoimmune disorder characterised by a triad of conjunctivitis, urethritis and arthritis.

Cause: Unknown. Reiter's syndrome may develop in response to an infection in another part of the body (cross-reactivity).

Symptoms and signs: An inflammatory arthritis of large joints, inflammation of the eyes in the form of conjunctivitis or uveitis, and urethritis in men or cervicitis in women, mucocutaneous lesions, as well as psoriasis-like skin lesions such as circinate balanitis, and

keratoderma blennorrhagica. Enthesitis (inflammation of the sites where tendons or ligaments insert into the bone) can involve the Achilles tendon resulting in heel pain. Not all affected persons have all of the manifestations.

Investigations: Clinical presentation (triad of conjunctivitis, urethritis and arthritis)

Management: The condition is self-limiting. If it persists, steroids, immunosuppressants, steroid injections in the joint, steroid eye drops and antibiotics are effective.

- **Behcet's disease**

Definition/Description: An autoimmune multisystem disorder, Behcet's disease is characterised by arthritis, iritis, and recurrent oral and genital ulceration.

Cause: Unknown. Association with an infective trigger may exist (cross –reactivity).

Symptoms and signs including oral manifestations include mouth ulcers, dysuria, epididymitis, erythema nodosum, anterior uveitis, keratitis, conjunctivitis, seizures, transient ischaemic attack (TIA), arthritis, arrhythmias and encephalitis. An exaggerated skin response occurring after minor trauma resulting in blisters is often seen (this response is called pathergy).

Investigations: Clinical presentation with arthritis, iritis and oro-genital ulcerations. A FBC (Hb low), ESR (raised), CRP (raised), IgA (elevated) will confirm the diagnosis.

Management: Topical and systemic steroids and immunosuppressants (azathioprine) are administered, while thalidomides are effective in advanced cases.

Immunodeficiency Diseases

Definition/Description: Immunodeficiency disorders fall into two categories: primary and secondary. Primary immunodeficiency disorders are of genetic origin, whereas secondary immunodeficiency disorders are acquired. Individuals with immunodeficiencies have an increased susceptibility to infections.

Primary (Genetic) Immunodeficiency Diseases

Primary immunodeficiency diseases are generally fatal, often resulting in death at an early age. They are either T-cell or B-cell defects. Examples of primary defects include congenital thymic dysplasia, severe combined immunodeficiency, and immunodeficiency with thrombocytopenia and eczema.

Oral involvement in primary immunodeficiency disorders may include periodontal disease, oral ulcerations and recurrent herpes infections.

Secondary (acquired) immunodeficiency diseases

- **HIV disease (for full discussion, see Chapter 5)**

Definition/Description: Immunodeficiency is due to a progressive reduction in CD4 lymphocytes from the circulation caused by human immunodeficiency virus (HIV) infection. Bacterial, fungal and viral infections, protozoal infestations and malignancies result from an immune deficiency.

Cause: Human immunodeficiency virus.

Symptoms and signs including oral lesions: Depending on the stage of the HIV disease, the following symptoms are encountered: malaise, fatigue, fever, weight loss, and diarrhoea. Signs include lymphadenopathy, wasting, oral candidosis, oral hairy leukoplakia, oral herpes zoster infections, immune thrombocytopenia, anal herpes infections and splenomegaly. Kaposi sarcoma and lymphomas are seen in advanced stages of the infection leading to acquired immunodeficiency syndrome (AIDS). A comprehensive list of oral manifestations is provided in Chapter 5.

Investigations: Detection of HIV antibodies in serum by an enzyme linked immunosorbent assay (ELISA) followed by a Western Blot test is confirmatory. HIV antigens can be measured in blood using a polymerase chain reaction (PCR) test. ESR, FBC (anaemia and thrombocytopenia), CD4 count (low), and a viral load count.

Two symptoms, two signs and two positive tests are required to make a diagnosis of an AIDS related complex.

Management: Three groups of antiretroviral drugs are available:

1. Nucleoside reverse transcriptase inhibitors (NRTIs) - zidovudin,
2. Non-nucleoside reverse transcriptase inhibitors (NNRTIs) - nevirapine and
3. Protease inhibitors (PIs) - indinavir.

Combinations of NRTI and PI are more effective; this therapy is called highly active antiretroviral therapy (HAART). Symptomatic and other treatment modalities for infections and neoplasms are part of the management regime. Avoidance of risk factors is important.

Chapter 15

Musculoskeletal and Joint Disorders

S. R. Prabhu

Abstract

This chapter deals with common musculoskeletal and joint disorders of dental interest. The disease entities discussed include muscular dystrophies, polymyositis, dermatomyositis, rheumatoid arthritis, osteoarthritis, osteoporosis, Paget's disease of the bone, and fibrous dysplasia. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of musculoskeletal and joint disorders are also described.

Introduction

Musculoskeletal and joint disorders are common. These can have developmental, inflammatory, immunological, infective, degenerative or neoplastic origins. Often dental patients with musculoskeletal disorders pose special problems in the management of dental diseases. A few conditions involving the muscles, bones and joints are discussed in this chapter.

Common Symptoms of the Musculoskeletal and Joint Disease

Depending on the bones and joints involved and the type of underlying pathology, symptoms include pain, rigidity, stiffness or impaired movement of joints or limbs, deformity, and susceptibility to fracturing.

Examination of the Locomotor System

Examination:

- Observe any abnormality of the posture or gait
- Inspect joints and muscles
- Check active joint movements
- Inspect the dorsum of the hands and wrists. Ask the patient to make a clenched fist, show the palms, touch the little finger with the thumb, and put the wrists through a full range of movements.
- **Ask the patient:**
  - To bend and straighten their elbows
  - To raise an arm above their head and then touch the back of their neck
  - To flex their neck and try to touch the tip of each shoulder with their ears
  - To attempt to touch their toes with the knees held straight
  - To extend by leaning backwards and lateral extension by sliding a hand as far as possible down the lateral side of the thigh, and to rotate by turning their head and shoulders to the right and left
  - To move the joints in all directions.

Palpate for any swelling, coarse crepitus and effusion, and note any tenderness.

Investigations in Musculoskeletal and Joint Disease

Investigations in musculoskeletal and joint disease include x-rays, CT scans, arthroscopy, immunological studies, biopsy, biochemical studies, and electromyography.

Diseases of Muscle, Bone and Joints of Dental Interest

- **Muscular dystrophies**

Definition/Description: Muscular dystrophies are a group of inherited, progressive muscle disorders caused due to defects in one or more genes responsible for normal muscle function. Muscular dystrophy symptoms become evident in children between the ages of 2 and 3 years. This condition is called Duchenne's muscular dystrophy (DMD). When symptoms become evident in later life (around 15 years) the condition is called Becker's muscular dystrophy (BMD).

Cause includes genetic defects, which are X–linked recessive disorders.

Symptoms and signs: In both types, progressive proximal muscles, particularly of the lower limbs, become weak. Patients have difficulty walking, characterised by wading gait and toe walking. Frequent falls are common. The majority of patients are confined to a wheel chair. Respiratory involvement is often fatal.

Investigations: Clinical findings and family history are suggestive. Electromyography is abnormal. Creatinine kinase (CK) levels are elevated. Diagnosis is confirmed by the detection of an abnormal expression of dystrophin (a cytoplasmic protein in the muscle fibres) using immunostaining.

Management: Management includes moderate exercise, the use of steroids, ventilator support for respiratory complications, and elective tracheostomy. This disorder is fatal.

Oral and Maxillofacial Manifestations and Dental Management Considerations

- Oral and maxillofacial manifestations include hypertrophy of the masseter and muscles of the lips and tongue
- Cross bite and open bite are common. Flattening of the palate becomes evident in later stages of the disease
- Delayed eruption can occur
- Oral health is poor in those patients with inflammatory periodontal disease

Dental management:

- Allow for shorter appointments.
- Progressive muscular dystrophy may produce abnormal reactions to several drugs. Dentist should consult the patient's physician before invasive procedures are undertaken.
- Steroids may cause adrenal suppression and any invasive dental procedure needs to be carried out with steroid supplements prior to its commencement.
- DMD and BMD predispose patients to developing significant complications when an inhalational anaesthetic is administered.
- As the muscle weakness and wasting associated with progressive muscular dystrophy makes it uncomfortable for the patient, the dental chair must be appropriately positioned. Some patients may experience respiratory symptoms.
- Administration of general anaesthesia should be carefully monitored because of the possible development of malignant hyperthermia and rhabdomyolysis.
- Oral hygiene care should receive a high priority. Patients and care givers should be advised on proper tooth brushing techniques and preventive of dental decay measures.

- **Polymyositis and dermatomyositis**

Definition/Description: The polymyositis (PM) and dermatomyositis (DM) are a heterogeneous group of acquired autoimmune skeletal muscle diseases called idiopathic inflammatory myopathies (IIM). These are characterised by the triad of chronic inflammation, fibrosis and the loss of muscle fibres.

Cause: These are autoimmune diseases.

Symptoms and signs: These include muscle ischaemia and necrosis, fever, weight loss, arthritis, arthralgia, subcutaneous calcifications, cardiac symptoms such as myocarditis, and pulmonary symptoms due to weakness of the thoracic muscles. Connective tissue diseases such as lupus erythematosus (LE), Sjögren's syndrome (SS), vasculitis and sarcoidosis may also be present in these patients.

Investigations: Clinical history, electromyography, blood tests for creatinine kinase (CK) and aldolase (both elevated), and muscle biopsy.

Management includes physical therapy to tone up the muscles, and steroids. Since steroids, if used on a long term basis, cause osteoporosis, bisphosphonates are also recommended. If steroids are not effective, an immunosuppressant such as Azathioprine is used.

Oral Manifestations and Dental Management Considerations

- Presence of telangiectasia of the oral mucosa and perioral tissues
- Higher prevalence of dental caries and dental plaque accumulation
- Hyposalivation and oral infections are common
- In rare cases calcinosis and generalised oral mucosal oedema may occur

Dental management considerations:

- Shorter appointments
- Dental caries preventive measures
- Treatment of oral infections
- Pre-operative steroid supplements to avoid adrenal crisis during invasive dental treatment
- Those on long term bisphosphonates (for osteoporosis) may present antiresorptive-induced necrosis of the jaw bones.

- **Rheumatoid arthritis**

Definition/Description: Rheumatoid arthritis is a multisystem immunologically mediated disorder characterised by inflammatory changes involving mainly the synovial joints such as hands, wrists, ankles and knees, and the presence of circulating antibodies to IgG (rheumatoid factor).

Cause: Unknown. The majority of patients are genetically predisposed individuals.

Symptoms and signs: These include symmetrical joint pain, stiffness, redness, swelling of joints of the hands, wrists and ankles mostly in the morning, 'spindled' appearance of the fingers and 'broadening' of the forefoot may appear at some stage of the disease. As disease progresses, shoulders, elbows, knees, cervical spine and temporomandibular joints may be involved. Hips are usually not involved.

General symptoms include fever, malaise, night sweats and weight loss. Joint mobility and stability are impaired and subluxation and ankylosis may occur. Deformities include ulnar deviation of fingers, loss of finger function, 'Z' deformity of the thumb, 'swan necking' of fingers, clawing of toes with painful sensations (walking on pebbles), and subcutaneous

nodules (rheumatoid nodules). Some patients may present signs and symptoms of Sjögren's syndrome or amylodosis.

Investigations: These include x-rays (of joints showing erosions, periarticular osteoporosis, ankylosis or subluxation), ESR (elevated), FBC, C-reactive protein (high), serum albumin (low) and globulin (high), fibrinogen (high), urinalysis for protein, arthroscopy, synovial biopsy and RA factor titre (may be negative in up to 30% of cases).

Management: NSAIDs (aspirin or indomethacin, for example), intra-articular corticosteroid injections, and immunosuppressants such as cyclophosphamide, azathioprine or methotrexate are used in management of the condition.

Prognosis: After 10 years, 25% of patients will enjoy complete remission, 40% moderate impairment, 25% severe impairment, and 10% will be crippled by the disease.

Oral and Maxillofacial Manifestations and Dental Management Considerations

- There are no specific oral mucosal manifestations. However, if secondary Sjögren's syndrome is present in these patients, xerostomia and associated oral findings may occur. Saliva substitutes or sialogogues may be necessary depending on the severity of xerostomia.
- Some patients may present temporomandibular joint symptoms. These include narrowed joint spaces, flattened condyles, osteoporotic changes and erosions which may be noted on x-rays.
- Long standing RA patients may show signs of advanced periodontal disease.
- A detailed history of the joint symptoms, their onset and duration should be recorded.
- Examination of joints of the hand and temporomandibular joint should be carried out.
- Oral hygiene must be maintained.
- Physical comfort in the dental chair should be provided, allowing the patient to rest periodically and to shift positions. The use of pillows on the dental chair may be necessary.
- Schedule shorter appointments.
- RA patients on corticosteroids and immunosuppressant medications are prone to oral infections. Infections should be aggressively treated with appropriate antibiotics. Pain medication also becomes a major part of management.
- If invasive dental treatment is required, patients on long term steroid therapy need to receive supplementary (double dose) steroids prior to the commencement of dental procedures. This is to avoid adrenal crisis. This needs to be carried out in consultation with the patient’s physician.
- Some RA patients may be on bisphosphonates for osteoporosis. The dental practitioner should be aware of the bisphosphonate associated jaw necrosis and its management.
- In order to facilitate home care, the use of floss holders, electric tooth brushes, and modification to traditional tooth brushes may be required.

- **Osteoarthritis**

Definition: Osteoarthritis, also known as degenerative joint disease, is the most common form of inflammatory joint diseases involving often-used joints such as hips, knees, feet, spine, hands and temporomandibular joint.

Cause: The exact cause of osteoarthritis is not known. Long term wear and tear of joints is associated with its cause. Other factors associated with osteoarthritis include trauma to the joints, metabolic disorders, pre-existing structural defects of the joints, and obesity.

Symptoms and signs include stiffness or pain in the joint(s) in the morning lasting 15-20 minutes, and without any signs of redness or swelling. Joint noises (crepitus) on movement of the joint(s) and appearance of **Heberden's** nodes (gelatinous cysts or bony outgrowths on the dorsal aspects of the distal interphalangeal joints) may also occur. If nodes appear on the proximal interphalangeal joints, they are called **Bouchard's nodes**.

Investigations: These include x-rays of the joint (revealing erosions, osteophytes, joint-space narrowing, etc), FBC (normal), ESR (normal), rheumatoid factor (negative), synovial fluid aspiration/microscopy, and serum uric acid (to rule out gout).

Management includes NSAIDS and treatment of any underlying condition. Intra-articular corticosteroids and joint replacement may occasionally be necessary.

Oral Manifestations and Dental Management Considerations

- Osteoarthritis (OA) is a common form of arthritis of the temporomandibular joint. With osteoarthritis, the temporomandibular joint can be the first joint to present symptoms, whereas in rheumatoid arthritis it is the last joint to be affected.
- The patients, who develop OA of the TMJ, present with a variety of symptoms including pain on opening, limited movement to the opposite side, a coarse grinding noise on function, a history of clicking, and deviation on opening to the affected side.
- A significant percentage of patients with TMJ osteoarthritis have history of grinding and clenching of teeth.
- Management options for TMJ osteoarthritis include reassurance, occlusal appliances, physical therapy, pain medication, and in some cases surgical intervention.

- **Osteoporosis**

Definition: Osteoporosis refers to loss of bone mass per unit volume.

Cause: Causes of and predisposing factors for osteoporosis include advancing age, androgen/oestrogen deficiency as in postmenopausal women, thyrotoxicosis, Cushing's syndrome, low calcium intake, long term steroid use, inflammatory arthritis, chronic renal disease and bone marrow replacement as seen in lymphomas and leukaemia treatments.

Symptoms and signs: Typical symptoms and signs of osteoporosis include bone pain, back ache, kyphosis, crush vertebral fractures, and fractures with minimal trauma, (particularly of the neck of the femur and distal radius).

Investigations: These include radiology (reduced bone density, fractures), DEXA scan or bone mineral density (BMD) test of the wrist and hip.

Management: These include calcium and Vitamin D supplements, bisphosphonates and oral calcium, and hormonal replacement therapy (HRT) for postmenopausal women.

Oral Manifestations and Dental Management Considerations

- Bisphosphonate associated osteonecrosis of the jaw (BONJ) may occur in those osteoporotic patients who are on bisphosphonate therapy for 4four or more years.
- Both mandible and maxilla may show necrosis. The mandible is more commonly involved.
- Predisposing factors for BONJ include dento-alveolar surgery including extractions, periodontal disease, dentures, presence of diabetes, and smoking.
- Prior to the start of bisphosphonate therapy, the physician should refer the patient to a dentist for clearance of the foci of oral infection.
- Mandibular or maxillary bone exposure for more than eight weeks in a patient on bisphosphonates can be regarded as an example of BONJ.
- The exposed bone area is painful. Swelling, halitosis and sinus track with pus may be present in the area of BONJ.
- Patients must be informed of the need for regular dental visits for oral hygiene instructions.
- Home oral care is important.

- **Paget's disease of the bone**

Definition/Description: Also known as osteitis deformans, Paget's disease of bone is a disorder characterised by excessive bone resorption followed by disorderly and excessive new bone formation, leading to softening and painful enlargement of the bone(s) involved.

Cause: Unknown. Viral association (persisting measles or respiratory viral infection) with the disorder has been suggested in recent years.

Symptoms and signs: Common bones involved are the skull, vertebrae, pelvis and long bones. Symptoms include bone pain especially at night, tenderness, deafness, nerve entrapment, pathological fractures, impaired vision and rarely, development of osteosarcoma. Enlargement of the maxilla (leontiasis ossea) occurs in long standing cases of Paget's disease.

Investigations: These include a FBC (normal), ESR (normal), alkaline phosphatase (elevated levels indicate increased osteoblastic activity), urinary hydroxyproline (elevated levels indicate increased osteoclastic activity) and acid phosphatase (normal). Radiography reveals osteolytic lesions, stress fractures, areas of osteosclerosis and osteoporosis circumscripta in the skull.

Management: Asymptomatic patients do not need any treatment. Symptomatic patients are treated with bisphosphonates, calcitonin, physiotherapy and occasionally, surgery.

- Progressive enlargement of the skull (requiring larger hats) due to thickening of the outer table of the vault.
- Enlargement of the jaws with wide alveolar ridges and a flattened, hard palate. Teeth are displaced.
- Denture wearers often complain of denture problems.
- Radiographic changes include irregular areas of radiolucency and radiopacity giving rise to a "cotton wool" appearance. Lamina dura is absent on radiographs. Hypercementosis is common and pagetoid bone may cause ankylosis of teeth.
- Cranial nerve symptoms and signs occur due to the narrowing of skull foramina. These include facial pain and facial paralysis.
- Osteosarcoma and osteomyelitis of the jaw have been reported in patients with Paget's disease.
- Dental management considerations:
- Those on bisphophonates need to be monitored for BONJ, and strict oral hygiene protocols need to be implemented
- Some cases of Paget's disease of the jaw bone need surgical correction for cosmetic reasons.

- **Fibrous dysplasia: (Monostotic and polyostotic (Albright's syndrome) fibrous dysplasia).**

Definition/description: Fibrous dysplasia is characterised by the replacement of an area of one bone (monostotic) or multiple bones (polyostotic) by fibrous tissue and causes localised swelling(s).

Cause: Unknown.

Symptoms and signs: Monostotic fibrous dysplasia commonly involves the jaws (the maxilla in particular) predominantly in females. Painless, slow enlargement of the maxilla ceases to progress after adolescence.

In the polyostotic type (Albright syndrome), enlargement can be seen in multiple bones associated with skin pigmentation called "cafe au lait" patches. Preferred sites include the dorsal aspect of trunk and limbs. Females affected present with precocious puberty in Albright's syndrome.

Investigations: Radiographs of the affected region show a ground glass appearance with no defined margins. In blood chemistry analysis, alkaline phosphatase levels are elevated. Urinary hydroxyproline levels are also increased.

Management: Fibrous dysplasia is a self-limiting disorder. Patients can be managed with calcitonin. Testolactone treatment is recommended for those females with precocious puberty in Albright's syndrome

Oral Manifestations and Dental Management Considerations

- Jaw lesions of monostotic fibrous dysplasia start in childhood.
- Bony, painless, slow growing, swelling of the maxilla results in malocclusion.
- Radiographs of the affected areas resemble orange–peel in appearance.
- In polyostotic fibrous dysplasia (Albright's syndrome), jaw involvement, skin pigmentation and precocious puberty are common
- Monostotic lesions are self-limiting.
- Surgical shaving of the enlarged part of the bone may be necessary for cosmetic reasons.
- Lesions should not be subjected to radiation.
- Bisphosphonates are used to treat symptomatic cases.

Chapter 16

Diseases of the Skin

S. R. Prabhu

Abstract

This chapter deals with skin disorders of dental interest. The clinical entities discussed include acne vulgaris, perioral dermatitis, atopic dermatitis, contact dermatitis, seborrheic dermatitis, actinic keratosis, skin cancer, melanoma, bullous pemphigoid, pemphigus vulgaris, dermatitis herpetiformis, epidermolysis bullosa acquisita, linear immunoglobulin A disease, herpes simplex virus infections, chicken pox, shingles, candidiasis, dermatophytoses, furuncles and carbuncles, molluscum contagiosum, warts, vitiligo, albinism, moles, lichen planus, psoriasis, erythema multiforme and lupus erythematosus. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of the skin disorders are described.

Introduction

Skin is the most accessible organ of the body. Diseases of the skin are common. A sizable number of skin diseases manifest in oral mucosal lesions. Dental practitioners need to have an adequate knowledge of the range of skin diseases, some of which may present oral manifestations.

Common Symptoms of Skin Diseases

Common symptoms of skin diseases include pruritus (itching), rash, hives and weals, white, red or pigmented patches, blisters, scales, scabs, ulcers, scars, haemorrhagic lesions and those with spots that can have nodular, ring like, linear, coin shaped or reticulated appearances. Pain may be an associated feature in some of these conditions.

Examination of the Skin

History and examination of the skin lesions are adequate in diagnosing a large number of skin disorders. Visual inspection is an important tool in the diagnosis of skin diseases. The characteristic appearances and morphology of skin lesions provide important clues to diagnosis.

Some important aspects of the examination process include:

- **Number of skin lesions:** are skin lesions single or multiple and which of the body parts are involved? Psoriasis, for example, involves the scalp, extensor surfaces of the elbows and knees, and the umbilical and gluteal areas. Lichen planus, on the other hand, has an affinity for skin on the wrists, forearms, genitals and lower legs. Lupus erythematosus lesions are confined to the face.
- **Colour of skin lesions** provides important information:
  - Red lesions may be developmental (vascular), inflammatory or infectious.
  - Yellow colour is due to jaundice in most cases.
  - Violet coloured skin lesions may be a sign of vasculitis.
  - Purple lesions may be examples of haemangioma or Kaposi's sarcoma.
  - Black lesions are melanotic including nevi and melanomas.

Some useful signs:

- Clinical signs such as epidermal separation which occurs on the application of gentle pressure on the skin around the lesion may be a sign of autoimmune bullous disease (**Nikolsky's sign**),
- The appearance of pin-point bleeding after the removal of scale from plaques, as in psoriasis (**Auspitz's sign**)
- Development of lesions within an area of trauma (scratching, for example) as in psoriasis and lichen planus (**Koebner's phenomena**) are important clinical signs that provide useful diagnostic clues.

Investigations in Dermatology

Investigations in dermatology include:

Scraping: Skin scraping is helpful in the diagnosis of fungal infections and scabies. For fungal infections, scraped material from the lesional border is placed on a microscope slide, and a drop of 10-20% potassium hydroxide (KOH) is added before microscopically examining the material. Hyphae or budding yeast are seen in tinea or **candidiasis**. For **scabies**, the material is placed directly on the slide with mineral oil under a coverslip. Mites or eggs can be detected microscopically confirming the diagnosis.

Diascopy: This test involves a microscope slide which is pressed against a lesion to see if it blanches (as in haemangioma).

Use of Wood's light. Wood's light (black light) is used to distinguish hypopigmented lesions from depigmented lesions. Depigmentation in vitiligo fluoresces ivory-white where as hypopigmented lesions do not fluoresce.

Tzanck testing: This involves a scraping of cellular material using a #15 scalpel blade from the base and sides of a vesicle, and staining with Giemsa or Wright's stain. When examined microscopically, multinucleated giant cells are seen in a blister-forming viral disease (herpes simplex or varicella–zoster infections, for example).

Biopsy: This is a confirmatory test for a wide range of skin lesions. Methods include incisional (for large lesions), excisional (for small lesions) and punch biopsy (for deep dermal tissues) techniques.

Skin Diseases of Dental Interest

- **Acne vulgaris**

Acne vulgaris, commonly known as acne, refers to the formation of comedones (uninfected sebaceous plugs impacted within follicles), papules, pustules, cysts and nodules as a result of obstruction and inflammation of the hair follicles and the corresponding sebaceous glands (pilosabaceous units).

Acne is common in adolescents. Clinical appearance is diagnostic. These often remit in the 20s but may continue into the 40s, particularly in women. Treatment includes topical and systemic agents which reduce the secretion of sebum and inflammation. Topical antibacterial agents (Benzoil peroxide gel, for example), topical comedolytic and exfoliant agents (tretinoin cream, for example), oral antibiotics (tetracycline, for example) and oral retinoids (isotretinoin, for example) are effective.

- **Perioral dermatitis**

Perioral dermatitis is an erythematous papulopustular facial (perioral) eruption which resembles acne. The exact cause is not known. Topical applications of steroids, and exposure to fluorides in water or tooth paste are sometimes associated with this disorder. Though this condition is not due to infections, treatment with systemic tetracyclines seems to be effective.

- **Atopic dermatitis (AD)**

Atopic dermatitis refers to an immune-mediated (IgE mediated in 70% of cases) inflammatory condition of the skin characterised by pruritus. Skin lesions range from mild erythema to lichenification. Environmental triggers include food (milk, soy, eggs, wheat, etc) airborn allergens (dust mites, dander etc), and *Staphylococcus aureus* colonization in the skin. Genetic predisposition may also be implicated. History and examination provide important clues to diagnosis. Treatment involves the use of moisturisers, topical steroids and the avoidance of triggers.

- **Contact dermatitis(CD)**

Contact dermatitis refers to acute inflammation of the skin caused by irritants or allergens and is characterised by pruritus, pain with erythematous or blistering lesions usually found on hands or other exposed surfaces. The majority of CD cases are examples of irritant contact dermatitis (ICD) in those who have had contact with acids, alkalies, etc. Allergic contact dermatitis cases are examples of type IV hypersensitivity responses to allergens such as chemicals used in shoe industry, cosmetics, fragrances, dyes, ingredients in topical drugs, latex, metal compounds and plants. Irritant contact dermatitis is predominantly painful whereas allergic contact dermatitis is predominantly pruritic. Diagnosis is by history and clinical examination. Patch testing is useful in allergic contact dermatitis (ACD). Treatment involves symptomatic treatment and avoiding contact with triggers. Topical application of steroids (oral steroids for severe cases of ACD) and antihistamines are effective for ACD cases. Dressings of the oozing lesions are useful in promoting healing.

- **Seborrheic dermatitis (SD)**

Seborrheic dermatitis (SD) refers to inflammation of the skin with a high density of sebaceous glands on the face, scalp and upper extremities. The cause of SD is unknown. A normal skin organism called *Pityrosporum ovale* is believed to be associated with the condition. SD is common in patients with HIV disease. Dandruff, yellow, greasy scaling along the hair line, and pruritus are common in SD. SD may precede the onset of psoriasis. Clinical diagnosis is adequate. Treatment involves the use of tar shampoos and topical corticosteroids.

- **Actinic keratosis**

Actinic keratosis is a premalignant keratotic lesion which occurs as a consequence of chronic sun exposure. Individuals with blonde or red hair are susceptible. Skin lesions are scaly or crusted and red, pink or grey in colour.

- **Skin cancer**

People with fair and light skin are susceptible to developing squamous cell and basal cell carcinomas due to chronic sun exposure. Skin cancer incidence is highest in outdoor workers.

Squamous cell carcinoma is a malignant neoplasm of the epidermal keratinocytes which infiltrate the dermis. Local spread and distance spread are common. Diagnosis is by biopsy. Treatment involves surgical dissection, cryosurgery, or occasionally radiotherapy.

Basal cell carcinoma is a slow-growing, superficial, malignant neoplasm which is derived from the epidermal basal cells. Local growth is destructive but distant metastasis is uncommon. Diagnosis is by biopsy. Treatment involves surgery, cryosurgery or occasionally, radiation therapy.

- **Melanoma**

Melanoma (malignant melanoma) arises from melanocytes in pigmented areas such as the skin, mouth, eyes and CNS. About 50% of melanomas develop from pigmented moles and the rest arise from melanocytes in normal skin. Though rare, melanomas can occur on the mucosa of the oral and genital regions and conjunctiva. Four types of melanomas exist. These are lentigo maligna melanoma, superficial spreading melanoma, nodular melanoma and acral-lentiginous melanoma.

Melanomas may vary in size, shape and colour. They spread rapidly causing death if not diagnosed and treated early. Distant metastasis occurs via lymphatics and blood vessels. Local metastasis is characterised by the presence of nearby satellite papules or nodules with or without pigmentatation. Treatment is by surgical excision.

- **Bullous Pemphigoid**

Bullous pemphigoid is an autoimmune disorder of the skin producing chronic bullous lesions, particularly in the elderly. Tense bullae appear on the normally appearing reddened skin. Pruritus is common. Nikolsky's sign is negative in these patients. Up to 30% of patients develop oral lesions. A skin biopsy of the lesion, and serum antibody titres are diagnostic. Corticosteroids are effective. Prognosis is generally good.

- **Pemphigus vulgaris**

Pemphigus vulgaris is an autoimmune disorder of the skin characterised by intraepidermal bullae and extensive erosions on healthy looking skin and mucous membranes. In this disease autoantibodies are directed against epidermal desmosomes causing intraepidermal split and blistering. Nikolsky's sign is positive in pemphigus vulgaris. Lesions occur first in the mouth, followed by skin lesions. Diagnosis is by biopsy of the edge of the fresh lesion and also from the nearby normal skin. Light microscopy and direct immunofluorescent methods are diagnostic. Treatment includes systemic use of corticosteroids, and sometimes with immunosuppressants. Maintenance therapy is essential. Prognosis is variable.

- **Dermatitis herpetiformis**

Dermatitis herpetiformis is an autoimmune chronic skin disease characterised by clusters of intensely pruritic vesicles and urticarial-like lesions. This disorder is common in patients with coeliac disease. Lesions tend to occur on the extensor surfaces of the skin on elbows, knees, sacrum, occiput and buttocks. Diagnosis is by skin biopsy. A direct immunofluorescent test showing IgA deposition in the dermal papillary tips is confirmatory. Treatment involves the use of dapsone and sulphapyridine.

- **Epidermolysis Bullosa Acquisita**

Epidermolysis bullosa aquisita is an autoimmune disorder characterised by the spontaneous appearance of blisters on the skin, and occasionally involving mucous membranes of the mouth, eyes, larynx, oesophagus and genitals. Diagnosis is by skin biopsy. Treatment involves the use of steroids or cyclosporins.

- **Linear immunoglobulin A disease**

Linear IgA disease is an autoimmune disorder characterised by vesicular or bullous lesions of the skin with intense pruritus or burning. Diagnosis is by skin biopsy. Treatment involves the use of dapsone.

- **Herpes simplex virus infections**

Herpes simplex viruses are known to cause recurrent infections of the skin and mucous membranes. Mucocutaneous infections cause clusters of small painful vesicles on an erythematous base. HSV-1 causes oral lesions, whereas HSV-2 causes genital lesions. Lesions may also occur on the anal area in individuals who engage in receptive rectal intercourse. HSV infections are common in immunocompromised individuals. HSV-1 causes acute herpetic gingivostomatitis and (recurrent) herpes labialis (see Chapter 5). In health care workers, HSV infection of the distal phalanx can occur. This is called herpetic whitlow. Diagnosis is often clinical. In severe infections serology is helpful. Treatment for isolated infections (which often go unnoticed) is not required. Severe infections are treated with acyclovir or famciclovir and analgesics for pain and fever.

- **Chickenpox**

Chickenpox is an acute, systemic, and usually childhood, infection caused by the varicella-zoster virus (VZV). This belongs to the *Herpesviridiae* family (HHV type 3). Usually chickenpox starts with mild constitutional symptoms, followed by skin lesions appearing in crops. These include macules, papules, vesicles, and crusting lesions. Diagnosis is clinical. Generally the infection is self-limiting. Symptomatic treatment for pruritus, pain and fever may be necessary. The severe form (common in adults) requires antiviral treatment with acyclovir or famciclovir.

- **Shingles (Herpes zoster)**

Herpes zoster (shingles) results when varicella-zoster virus reactivates from its latent state in a posterior dorsal root ganglion. Pain along the affected nerve is the initial symptom. This is soon followed by the appearance of vesicular eruptions. Diagnosis is clinical. Treatment includes antiviral agents, and corticosteroids in the severe form of the disease. Postherpetic neuralgia may occur in elderly patients at a later stage.

- **Candidiasis**

Infection by Candida species (commonly *C. albicans*) can result in candidiasis. Skin infection can occur anywhere. Skin folds are the most commonly involved sites.

Infection can also involve mucocutaneous sites. Vaginal involvement (candidal vaginitis) is common in pregnant females, with a burning and itching sensation. Diagnosis is clinical. Skin scrapings examined microscopically in a wet mount with potassium hydroxide is diagnostic. Treatment involves antifungal agents and drying agents.

- **Dermatophytoses**

Dermatophytoses are fungal infections of keratin in the skin and nails.

Nail infection is called tinea unguium.

Tinea barbae is a dermatophyte infection of the beard area caused by *Trichphyton mentagrophytes* or *T. verrucosum.*

Tinea capitis (scalp ringworm caused by *Trichphyton tonsurans*) is a dermatophyte infection of the scalp.

Tinea corporis (caused by *Trichphyton mentagrophytes)* is a dermatophyte infection of the face, trunk and the extremities (Body ringworm).

Tinea cruris (caused by *Trichphyton mentagrophytes*) is a dermatophyte infection of the groin.

Tinea pedis **(**caused by *Trichphyton mentagrophytes)* is a dermatophyte infection of the feet. Diagnosis is by clinical and KOH wet mount. Dermatophytes need to be treated with antifungal agents.

- **Furuncles and carbuncles**

Furuncles (boils) are tender nodules caused by staphylococcal infection of the skin. When clusters of furuncles are connected subcutaneously causing deeper suppuration, they are called carbuncles. Diagnosis is by clinical appearance. Treatment involves the use of warm compresses and oral antibiotics.

- **Molluscum contagiosum**

Molluscum contagiosum is characterised by clusters of smooth, waxy or pearly umbilicated papules of 1-5 mm in diameter caused by the molluscum contagiosum virus. Lesions occur frequently on the face, trunk, pubis, penis and vulva. In HIV and immunocompromised patients, lesions may grow larger. Diagnosis is based on the clinical appearance. Treatment involves trichloroacetic acid application or the use of electrotherapy, cryosurgery, or laser surgery.

- **Warts (Verruca vulgaris)**

Skin warts are common, benign epidermal lesions associated with human papilloma virus (HPV) infection. They are asymptomatic and appear as flat, verrucous or cauliflower-like

lesions on the skin. Palmar and plantar warts, periungual warts, flat warts, filiform warts and genital warts are common. Diagnosis is based on clinical appearance. Treatment involves the use of an irritant such as trichloroacetic acid, salicylic acid or podophyllum resin.

- **Vitiligo**

Vitiligo is the loss of skin melanocytes that cause areas of skin depigmentation of varying sizes. The cause of vitiligo is unknown. Autoimmunity is associated in about 30 % of cases. Diagnosis is clinical. Topical corticosteroids, psoralens plus ultraviolet A are used in the treatment of vitiligo.

- **Albinism (oculocutaneous albinism)**

Albinism is an inherited defect in melanin formation resulting in generalised hypopigmentation of the skin, hair and eyes. Ocular involvement often produces strabismus or nystagmus. No treatment is required. Protection from sunlight is necessary in order to avoid skin cancers.

- **Moles (nevi)**

Moles, or nevi, are pigmented papules or nodules on the skin. These are common. They may present as small or large, flesh-coloured, yellow-brown, brown or dark brown lesions. These lesions can be flat or raised, smooth, hairy or warty. Although these lesions have a potential to become malignant, the majority of moles are benign at the time of presentation. Colour changes in the moles, with irregular borders, or bleeding or ulcerative features may suggest malignant change. These suspect lesions should be biopsied and histologically examined. Prophylactic removal is not indicated for moles of benign nature.

- **Lichen planus (LP)**

Lichen planus is a chronic recurrent inflammatory condition characterised by small, flat, polygonal, violaceous papules and scales. Oral reticular, erosive or bullous lesions are also common (in 50% of skin LP) in lichen planus. Skin lesions are itchy and symmetrically distributed on the flexor surfaces of wrists, legs and lower back. Causes of LP are uncertain. Immunologic association has been suggested. Diagnosis is by history and clinical appearance of the lesions. Biopsy is confirmatory. Erosive oral lichen planus may carry malignant potential.

- **Psoriasis**

Psoriasis is an inflammatory disease characterised by well circumscribed erythematous papules and plaques covered with silvery scales. The cause is unknown. Triggers of the disorder include trauma, infection and certain drugs. Mild itching may be present. Arthritis is a feature of the severe form of the disease. Diagnosis is based on the clinical appearance and distribution of the skin lesions. Treatment involves the use of emollients, vitamin D

analogues, retinoids, tar, anthralin, corticosteroids, and phototherapy. Methotrexate and immunosuppressants are used for severe cases.

- **Erythema multiforme**

Erythema multiforme is an inflammatory reaction characterised by "target" or "iris" lesions on the skin. Onset is sudden with erythematous macules, papules, weals, vesicles and/or bullae on the distal extremities including the palms and soles. The face is also frequently involved. The classic lesion is annular with a violaceous centre and pink halo separated by a pale ring. This lesion is called an iris or target lesion. Lesions occur as a reaction to a drug or an infectious agent such as herpes simplex virus. Lesions spontaneously resolve but are recurrent.

Diagnosis is by history and clinical appearance. Rarely, biopsy may be necessary. Lesions resolve and treatment is not required for the majority of cases. Topical and systemic corticosteroids are effective for severe symptoms. Antiviral therapy is recommended if recurrences are frequent.

Chapter 17

Diseases of the Eye, Ear, Nose and Throat

S. R. Prabhu

Abstract

This chapter deals with diseases and disorders of the eye, ear, nose and throat. Clinical entities discussed include blepharitis, conjunctivitis, cicatricial pemphigoid, trachoma, allergic conjunctivitis, keratoconjunctivitis sicca, glaucoma, cataracts, diabetic retinopathy, hypertensive retinopathy, retinal detachment, age-related macular degeneration, otitis externa, acute otitis media, chronic otitis media, nasal polyps, rhinitis, sinusitis, acute pharyngitis, acute laryngitis and globus pharyngeus. Each disease entity starts with the definition or description followed by its causes, symptoms, signs, investigations employed and principles of management. Where relevant, the oral and dental aspects of eye, ear, nose and throat disorders are described.

Introduction

Basic knowledge of the diseases of the eye, ear, nose and throat are of importance to dental practitioners because the dental practitioner is often faced with diagnostic confusion in determining the origin and nature of symptoms originating in the face.

Common symptoms of Eye Disorders

Vision loss: Symptoms of eye disorders include acute or gradual vision loss. Acute vision loss may be due to central retinal artery or vein occlusion, optic neuropathy, vitreous haemorrhage, retinal detachment and neo-vascular age-related macular degeneration. Gradual vision loss is due to cataracts, glaucoma, and age-related macular degeneration.

Blurred vision is one of the most common eye complaints. This is characterised by general dimming or distortion of vision.

Double vision (diplopia) is also common in patients with cataracts, corneal scarring and dislocated lens and retinal detachment.

Short-sightedness Also called myopia, short-sightedness is a common eye condition that causes distant objects to appear blurred, while close objects can be seen clearly.

Long-sightedness (hypermetropia): A hypermetropic person may have blurred vision when looking at objects close to them, and clearer vision when looking at objects in the distance.

Tunnel vision: Tunnel vision (also known as **Kalnienk vision**) is the loss of peripheral vision while retaining central vision, resulting in a constricted circular tunnel-like field of vision.

Eye lid swelling, eye pain, red eye and tearing are other commonly encountered symptoms. Often these have inflammatory or infectious cause.

Squint (strabismus) is characterised by a lack of alignment of the eyes.

Stigmatism (sometimes called astigmatism) happens when the cornea of the eye is misshapen and unable to focus sharply on a single object or point in space.

Exophthalmos (protrusion of the eye balls) is a sign of Graves' disease.

Investigations in Ophthalmology

Diagnostic aids used for eye disorders include physical examination and special tests. Physical examination of the eye includes tests for visual acuity, eyelid and conjunctival examination, corneal examination, ophthalmoscopy, slit lamp examination, visual field testing and colour vision testing. Special tests used in ophthalmology include tonometry (measuring intraocular pressure), fluorescein angiography (to investigate perfusion and neovascularisation), electroretinography (to determine retinal function), ultrasonography (to determine retinal tumours and retinal detachments) and CT scans and MRI (to determine ocular trauma, tumours and intraocular foreign bodies).

Eye Disorders of Dental Interest

- **Blepharitis**

Definition: Blepharitis is acute or chronic inflammation of the eyelid margins.

Cause: In the majority of cases, this is caused by staphylococci.

Symptoms include burning or itching of the eyelids, with redness and swelling.

Diagnosis is by history and clinical examination. This can manifest as ulcerative and non-ulcerative forms.

Treatment: For ulcerative blepharitis treatment includes topical antibiotics or systemic antivirals. Topical steroids are effective for non-ulcerative blepharitis. The sudden onset of localised swellings of the eyelid is called chalazion.

- **Conjunctivitis**

Definition: Inflammation of the conjunctiva results in conjunctivitis.
Causes of conjunctivitis include infection, allergy or irritation.
Symptoms include discomfort, ocular discharge, and itching.
Diagnosis is by history and clinical examination.
Treatment depends on the cause and may include topical antibiotics, antihistamines and corticosteroids.

Viral conjunctivitis is a highly contagious acute infection of the conjunctiva. This is usually caused by an adenovirus. Infection is self-limited. Topical steroids are required in severe cases.

- **Cicatricial pemphigoid**

Definition: Cicatricial pemphigoid is a bilateral, chronic, progressive, scarring and shrinkage of the conjunctiva with opacification of the cornea.
Cause: This is an autoimmune disorder.
Treatment: Systemic corticosteroids are used to treat this condition. Sometimes the condition results in blindness.

- **Trachoma**

Definition: Trachoma is a chronic conjunctivitis caused by *Chlamydia trachomatis*, which is the leading cause of preventable blindness worldwide.
Symptoms include eyelid oedema, photophobia and lacrimation. In later stages, corneal scarring may result.
Treatment: Topical or systemic antibiotics are used to treat this condition.

- **Allergic conjunctivitis**

Definition: Allergic conjunctivitis is caused by airborne allergens.
Symptoms include itching, lacrimation, and discharge. This is also called hay fever conjunctivitis.
Treatment includes the use of topical antihistamines.

- **Keratoconjunctavitis sicca (Dry eyes)**

Definition: Keratoconjunctivitis sicca is a chronic, bilateral desiccation of the conjunctiva and cornea due to an inadequate tear film.
Symptoms: Itching, burning, photophobia and irritation are common symptoms. **Treatment** includes topical tear supplements.

- **Glaucoma**

Definition: The glaucomas are a group of eye disorders characterised by progressive optic nerve damage.

Cause: Increased intraocular pressure (normally, 11-21 mm Hg). This causes increased pressure on the nerve or diminution of the blood flow

Symptoms: Glaucoma can permanently damage vision in the affected eye and cause blindness if not treated early.

Treatment includes laser surgery.

- **Cataracts**

Definition: Cataracts are a degenerative opacity of the lens characterised by gradual vision blurring.

Diagnosis is by ophthalmoscopy and slit lamp examination.

Treatment is by surgical removal and replacement of an intraocular lens (either plastic or silicone).

- **Diabetic retinopathy**

Definition: Diabetic retinopathy includes microaneurysms, haemorrhages, exudates and macular oedema occurring with diabetes of several years duration.

Diagnosis is by fundoscopy.

Treatment includes controlling diabetes and laser coagulation of threatening lesions.

- **Hypertensive retinopathy**

Definition: Hypertensive retinopathy is retinal vascular damage due to high blood pressure.

Cause: Hypertension.

Diagnosis: Fundoscopy reveals arteriolar constriction, vascular wall changes, flame shaped haemorrhages, cotton-wool spots, yellow hard exudates and papilloedema.

Management: Treatment is directed at controlling hypertension.

- **Retinal detachment**

Definition: Rental detachment is the separation of the neural retinal layer from the underlying retinal pigment epithelium layer.

Symptoms: Decreased peripheral or central vision results. Flashing lights and floaters are also reported by patients.

Diagnosis is by indirect fundoscopy and ultrasonography.

Treatment: Systemic corticosteroids, laser surgery, and cryotherapy are used to treat retinal detachment.

- **Age-related macular degeneration**

Definition: Age-related macular degeneration (AMD) is atrophy or degeneration of the macula.

Symptoms: This is a common cause of deteriorating central vision in the elderly. Two forms exist: Atrophic AMD (the dry form) and exudative AMD (the wet form). Atrophic AMD causes vision loss but rarely leads to blindness. The exudative form, if untreated, results in vision deterioration leading to blindness.

Diagnosis: Fundoscopic examination is diagnostic. The exudative form can be detected on an Amsler grid which, when used by patients with wet AMD, shows a central dark patch and wavy or distorted lines.

Treatment for atrophic AMD is generally not recommended. For the exudative form of AMD, laser treatment and injectable anti-vascular endothelial growth factor are used. Magnifying glasses are helpful in those who have lost central vision.

ENT Diseases of Dental Interest

Common symptoms of ENT diseases

Symptoms of ENT diseases include hearing problems, vertigo, dizziness, loss of balance, earache, ringing in the ears, pus discharge from the ear, loss of smell, stuffy nose, runny nose, nose bleeds, pain and discharge from the maxillary sinus, throat pain due to infections, voice disorders, difficulty in swallowing and neuralgic pain.

Investigations in ENT practice

These include Rinne and Webber tests, barium meal, video fluoroscopy, oesophageoscopy, imaging tests such as x-rays, CT and MRI, fine needle aspiration (FNA) biopsy and incisional/excisional biopsy.

- **Otitis Externa**

Definition/description: Inflammation of the skin of the external auditory canal.

Cause: Infection from *Pseudomonas aeruginosa, Escherichia coli, and Staphylococcus aureus* and fungi.

Symptoms and signs: discharge from the ear, discomfort and hearing impairment. Rarely, cervical lymphadenopathy may be present.

Investigations: History and clinical examinations are adequate in diagnosing the condition. Culture studies are required only for resistant cases.

Management: Aural toilet and topical antibiotics, and steroid drops are useful. Systemic antibiotics are used only if infection spreads to other tissues.

- **Acute otitis media.**

Definition/description: Acute otitis media is a bacterial infection of the middle ear and mastoid air cell system. It is common in children.

Cause: Bacteria cause this condition, namely *Haemophilus influenza, Streptococcus pneumonia, Streptococcus pyogenes and Staphylococcus aureus.*

Symptoms and signs include earache (otalgia), fever, discharge, ear drum inflammation and occasionally, ear drum perforation.

Management includes the use of symptomatic treatment with analgesics or antipyretics. Antibiotics are administered if signs of spreading infection become evident. In such cases, facial palsy is one complication in rare circumstances.

- **Chronic otitis media**

Definition/description: Chronic otitis media is a permanent abnormality of the tympanic membrane due to scarring (tympanosclerosis) or perforations. Two types are known: chronic otitis media and chronic mucosal otitis media. This is a common condition in those with lower socioeconomic status.

Cause: A consequence of acute otitis media in childhood. In some cases, patients with oro-facial clefts are vulnerable.

Symptoms and signs: intermittent discharge and hearing loss are common.

Diagnosis includes history and clinical examination.

Management: Aural toilet and topical medication with antibiotics and hydrocortisone are effective.

- **Nasal polyps**

Definition/Description: Nasal polyps are benign masses arising from the mucous membranes of the nose and paranasal sinuses. They are overgrowths of the mucosa that frequently accompany allergic rhinitis. Nasal polyps are freely movable and generally painless and non-tender.

Cause: Patients with a history of allergic rhinitis are at a higher risk of developing nasal polyps. Nasal polyps may also be found in asthmatics and in children with cystic fibrosis.

Symptoms and signs: Frequent symptoms include nasal congestion, sinusitis, loss of smell (anosmia), and secondary infection leading to headache.

Diagnosis: History and clinical examination of the nose offers useful diagnostic information. Imaging for sinus may be necessary for those with polyps arising from paranasal sinuses.

Management: Polyps may be removed by surgery. They tend to recur in about 70% of cases. Individuals with asthma and nasal polyps should avoid aspirin because of its potential to cause severe shortness of breath.

- **Rhinitis**

Definition/description: Rhinitis essentially means inflammation of the nasal mucous membrane caused by allergens (allergic rhinitis, or hay fever) or by viruses (non-allergic rhinitis).

Symptoms and signs: Sneezing and nasal itching, stuffy nose, coughing, headache, fatigue and malaise, watery, reddened or itchy eyes, and puffiness around the eyes are common.

Diagnosis: Clinical examination.

Management includes avoidance of allergens, use of antihistamines and topical corticosteroids for allergic rhinitis. Viral rhinitis is self-limiting.

- **Sinusitis**

Definition/description: Sinusitis is inflammation of the paranasal sinuses.

Causes include infection by bacteria (such as *H. influenzae, Strep. pneumoniae, Strep. aureus* and *Moraxella catarrhalis),* allergy, or systemic autoimmune disorders.

Symptoms and signs include headache, facial pain and nasal obstruction. Pain may worsen when the affected person bends over or when lying down. Thick nasal discharge (usually green in colour) may contain pus (purulent) and/or blood. Halitosis, postnasal drip and cough are present. Fullness in the ear, headache or toothache is often present. In rare cases, infection of the eye socket may result in the loss of sight and is accompanied by fever and severe illness.

Investigations: Clinical. Sinus tap, lavage and culture are common investigative methods. CT scans may be used to determine the extent of chronic disease.

Management: Usually self-limiting. Antibiotics are used to speed the recovery. Topical decongestants are useful in relieving a stuffy nose. Complications may include orbital cellulitis, cavernous sinus thrombosis, meningitis and brain abscesses.

- **Acute pharyngitis (sore throat)**

Definition/description: Acute pharyngitis is a generalised (acute) inflammation of the whole pharynx (including tonsils).

Cause: Causes include viral (40-60%), bacterial (5-30%), fungal (in immunocompromised patients), and non-infectious such as allergy, gastroesophageal reflux disease, post –nasal drip, chemical injury, smoking and trauma during endotracheal intubation.

Glandular fever (infectious mononucleosis) caused by EBV also presents with sore throat in the initial stages of the disease. Streptococcal sore throat may present tonsillar exudates.

Symptoms and signs: The majority of patients develop only the sore throat. Other features include fever, headache, malaise, anorexia and loss of appetite. Palatal haemorrhagic spots may be seen in some cases. Anterior cervical lymph node enlargement occurs. Generalised lymphadenopathy is seen in cases with glandular fever.

Investigations: Clinical. Throat swabs in resistant cases are useful. Conduct a monospot test if glandular fever is suspected.

Management: Analgesics or antipyretics are used for pain relief and fever. Antibiotics in severe cases (penicillin-based antibiotics are not to be used for those with glandular fever).Tonsillectomy is recommended for those patients with large tonsils (causing sleep apnoea) and for those with tonsillitis for more than two years.

Complications: These include rheumatic fever, glomerulonephritis, (due to beta-haemolytic streptococcal pharyngitis) and parapharyngeal abscesses.

- **Acute Laryngitis**

Definition/description: This refers to an acute inflammation of the vocal cords.

Cause: Viral or bacterial infection in most cases. Dust, allergies or gastroesophageal reflux disease (GORD) may also cause acute laryngitis.

Symptoms and signs: Sore throat, husky voice, impaired breathing or strider is common.

Investigation: Clinical.

Management: Rest the voice and symptomatic care. Antibiotics are used only when a bacterial cause is established.

- **Globus pharyngeus**

Definition/description: Globus pharyngeus refers to a sensation that something is stuck in the throat.

Cause: GORD, psychogenic or oesophageal dysmotility.

Symptoms and signs: Choking sensation or a ball-like lump in the throat that is generally relieved by eating.

Investigation: Throat examination includes clinical and nasendoscopy. Examination of the neck for lymphadenopathy is also essential to rule out lumps in the neck. Barium studies (for GORD or those with oesophageal dysmotility) are useful in eliminating other causes.

Management: Reassurance that there is no underlying cause is an important step. Treatment for GORD and antidepressants in some cases may be necessary.

Chapter 18

Psychiatric and Related Disorders

S. R. Prabhu

Abstract

This chapter deals with psychiatric disorders of dental interest. Topics discussed include anxiety neurosis, depression, hysteria, obsessive neurosis, post-traumatic stress disorder, delirium, dementia, schizophrenia, bipolar disorder, dysthymia, substance-induced disorders, anorexia nervosa, bulimia nervosa, autistic spectrum disorder, attention deficit hyperactivity disorder, Down syndrome and cerebral palsy. Where relevant, dental management aspects are also discussed

Introduction

Psychiatric disorders, also referred to as mental disorders or mental illnesses, are a group of psychological disorders potentially reflected in the behaviour of an individual. A psychotic disorder is generally determined by a combination of how an individual thinks, feels, acts and perceives, and his or her ability to relate to others. Psychiatric disorder can be broadly grouped as anxiety, mood, psychotic and eating disorders.

Neurodevelopmental disorders, on the other hand, are characterised by impairments of the growth and development of the brain. Neurodevelopmental disorders of interest include autism, autism spectrum disorder (ASD), attention deficit hyperactivity disorder (ADHD), Down syndrome (DS) and cerebral palsy (CP). Some patients with psychiatric and neurodevelopmental disorders present oral manifestations. Special dental management considerations are also necessary for these patients.

Common Symptoms of Psychiatric Disorders

Symptoms (and signs) of psychiatric disorders include:

- Appearance and behaviour (self-neglect, depression, mania, tics, compulsions, etc),
- Speech (dysarthria, stammering, etc),
- Emotion (mood changes)
- Thought content (obsessions, phobias, etc), abnormal beliefs and interpretation of events (delusion, abnormal experiences, or hallucinations).
- Cognitive disorders (distractibility, amnesia, learning disabilities, etc).

Investigations in Psychiatric Disorders

These include interviews (mental status examination), physical examination and in some cases, neuroimaging tests.

Psychiatric Disorders of Dental Interest

- **Anxiety neurosis**

Definition/description: Anxiety neurosis is characterised by increased autonomic activity which results in the release of adrenaline. This leads to restlessness, dry mouth, palpitations, sweating, headaches and diarrhoea. When certain objects provoke anxiety the condition is called phobia. Fear of closed spaces (claustrophobia) and fear of spiders (arachnophobia) are common.

- **Depression**

Definition/description: Depression is characterised by sleep disturbances resulting in early morning waking, sadness of mood, loss of appetite, and loss of weight, loss of interest in daily activities, atypical facial pain, depersonalisation and suicidal thoughts. Depression is generally reactive to adverse life events such as bereavement, retirement, separation, divorce, etc.

- **Hysteria**

Definition/description: Hysteria is a subconscious effort used by the individual to resolve anxiety. Hyperventilation is a feature of hysteria. In rare instances, this may cause changes in the acid-base balance resulting in tetany and collapse.

- **Obsessive neurosis**

Definition/description: Obsessive neurosis leads to repetitive actions or compulsions and obsessional thoughts. Examples include constant hand washing, returning home to check that lights or gas burners have been switched off or doors locked.

- **Post-traumatic stress disorders**

Definition/description: Post-traumatic stress disorders (PTSDs) occur after dangerous and life threatening experiences such as car or plane crashes, or battle ground experiences. Irritability, loss of concentration and recurrent nightmares are common features of PTSDs.

- **Delirium**

Definition/description: Delirium is characterised by a clouding of consciousness leading to the disorientation of time and place. Delirium can occur in alcohol withdrawal, chest infections that cause brain hypoxia, drug overdose and stroke.

- **Dementia**

Definition/description: Dementia is characterised by an irreversible decline in mental capacity, short term memory loss and slow laboured thinking. This is common in senile dementia as in Alzheimer's disease (due to neuronal atrophy) or after stroke.

- **Schizophrenia**

Definition/description: Schizophrenia involves disorders of thought, emotion and volition (making a conscious choice or decision). Delusions and hallucinations are characteristic features of schizophrenia. Patients may become catatonic (motionless and speechless) as well.

- **Bipolar disorder**

Definition/description: Bipolar disorder is a mood disorder often referred to as manic depression and is characterised by alternating periods of mania and depression.

- **Dysthymia**

Definition/description: Dysthymia is a mood disorder characterised by a person reporting a low mood on a daily basis over a span of two years.

- **Substance-induced mood disorders**

Definition/description: Substance-induced mood disorders are caused by psychoactive drugs or other chemical agents giving rise to mood disorders. Alcoholism and chronic use of benzodiazepine (valium) are included in this category.

- **Anorexia nervosa**

Definition/description: Anorexia nervosa is an eating disorder characterised by deliberate weight loss induced by the individual (mostly adolescent girls) by self-induced vomiting, self-induced purging, use of appetite suppressants or diuretics, and excessive exercise. This leads to malnutrition and secondary endocrine and metabolic disturbances.

- **Bulimia nervosa**

Definition/description: Bulimia nervosa is an eating disorder characterised by repeated bouts of overeating to control the body weight. The effects of repeated vomiting in these patients may give rise to tetany and electrolyte disturbances leading to cardiac problems. Dental effects include tooth erosion.

Other Related Disorders of Dental Interest

- **Autistic Spectrum Disorder (ASD)**

Definition/description: ASD includes typical autism, Asperger's syndrome and atypical autism.

Cause: Causes of ASD are multifactorial. Genetic and others environmental causes have been associated with ASD. First degree relatives and identical twins are affected supporting a genetic basis. Suggested environmental factors include impaired methylation and gene mutation involving metabolism of vitamin D, maternal infection, maternally derived antibodies and paternal age at time of conception.

Symptoms and signs: Boys are more likely to have an ASD than girls (M:F 4:1). The condition is characterised by a lack of social interaction, a lack of verbal or non-verbal communication, repetitive and stereotyped behaviours, and resistance to change.

Diagnosis/investigations: Diagnosis of ASD is based on the behaviour of the individual. Often a multidisciplinary team approach is required to study the behavioural patterns.

Management: There is no specific treatment for ASD. These individuals are put on medications to control hyperactivity, repetitive behaviours and aggression. Antidepressants and anti-seizure medications are commonly used.

- **Attention Deficit Hyperactivity Disorder (ADHD)**

Definition/description: Attention deficit hyperactivity disorder (ADHD) is characterised by an inability to regulate attention, sometimes with displays of hyperactivity and impulsivity.

Cause: the exact cause of ADHD is unknown. A strong genetic association has been suggested.

Symptoms and signs: Boys are more likely to have an ADHD than girls. There is inability to regulate attention. ADHD children show hyperactive and impulsive behaviour.

Diagnosis: Diagnosis involves classifying children into three types: predominantly inattentive, predominantly hyperactive-impulsive, or a combination of the two. Inadequately treated adults may take up illicit drug use.

Management: This consists of behavioural therapy and pharmacotherapy with tricyclic antidepressants and CNS stimulants.

- **Down syndrome**

Definition/description: Down syndrome (DS) is a congenital chromosomal abnormality characterised by systemic anomalies, intellectual disabilities and a recognisable craniofacial appearance.

Cause: Down syndrome is also known as trisomy 21, and is caused by a mutation resulting in an extra copy of chromosome 21.

Symptoms and signs: Live births are commonly associated with mothers who are around 35 years or older. Systemic anomalies are common in DS individuals. These include cardiac anomalies, immune defects, hypotonicity, atlantoaxial instability, intellectual disability, and increased risk for leukaemia and Alzheimer's disease. Oral manifestations are common.

Diagnosis: Diagnosis of DS can be determined prenatally by foetal ultrasound or by detecting abnormal levels on maternal serum screening for fetoprotein or (high) chorionic gonadotropin

Management: The underlying disorder cannot be treated. Congenital cardiac anomalies are surgically corrected

- **Cerebral palsy (CP)**

Definition/description: Cerebral palsy refers to a group of non-progressive syndromes characterised by impaired voluntary movement (spasticity). This is a common neuromuscular disorder.

Cause of CP is multifactorial. Prematurity, in utero disorders, neonatal encephalopathy, and kernicterus (bilirubin-induced brain dysfunction) are said to be contributory factors.

Symptoms and signs: The motor disorders of CP are often accompanied by disturbances of sensation, cognition, communication, perception and /or behavior. CP can be classified based on which part of the body is affected: 1. Quadriplegic CP, in which all four extremities, trunk and oromotor musculature are involved 2. Hemiplegic CP, with one side of the body involved, 3. Diplegic CP involving just the legs and 4. Monoplegic CP involving just one limb.

Diagnosis/investigations: Clinical symptoms are suggestive. A cranial MRI is done to detect abnormalities of the brain.

Management: Depending on the severity CP patients are managed with antispasmodic and anti-seizure medications. Surgical intervention to correct contractures of the foot, ankle, hand wrist and knee may be necessary for some patients.

Oral Manifestations and Dental Management Considerations in Psychiatric and Neurodevelopmental Disorders

- Patients with psychiatric disorders may show poor oral hygiene, xerostomia (due to medication) and higher rate or risk of dental caries and periodontal disease.
- Bruxism, oro-facial pain and temporomandibular joint symptoms are often reported in post-traumatic stress disorder (PTDS) patients.
- Some patients may be uncooperative and may not disclose facts about the medications they are on. Consultation with the physician is necessary in these circumstances.
- Wearing of removable prosthesis is problematic in patients with schizophrenia due to uncontrolled jaw movement.
- Anorexia nervosa and bulimia patients need to be advised of home oral hygiene care.
- Dental erosion on the palatal surfaces of the maxillary anterior teeth is associated with bulimia nervosa. Fluoride regimen should be regularly used to prevent caries.
- Trauma to the soft palate due to self-induced vomiting is often reported in patients with bulimia nervosa.
- Parotid gland enlargement may be a feature of eating disorders.
- Patients with schizophrenia are put on clozapine (an antipsychotic drug). Long term use of this medication is known to cause agranulocytosis, as a result of which patients may show oral ulceration and higher susceptibility to infections.
- Patients on antipsychotic drugs often complain of dry mouth, and taste disturbances. Stomatitis and glossitis are common in these patients.
- Patients with psychotic disorders often become irritated, agitated and uncooperative with dental procedures. Conscious sedation, iatrosedation or the use of anxiolytic medications may be necessary in these patients.
- Oral manifestations of Down syndrome include:
    - Mid-face hypoplasia
    - High palatal vault
    - Macroglossia
    - Fissured tongue
    - Large thick lips with hypotonia
    - Halitosis due to chronic mouth breathing
    - Microdontia, hypodontia, supernumerary teeth, delayed eruption and maxillary impactions due to maxillary under development.
- In cerebral palsy (CP) malocclusion, hypersalivation, bruxism, and extensive calculus deposits have been reported.

Chapter 19

Nutritional Disorders

S. R. Prabhu

Abstract

Advanced nutritional deficiencies have profound impacts on oral health. In this chapter, some important nutritional diseases and their possible oral manifestations are briefly discussed. The topics discussed include protein-calorie malnutrition, vitamin deficiencies, obesity, metabolic syndrome and a balanced, healthy diet.

Introduction

Nutritional deficiencies have a profound effect on oral health. A reasonable understanding of the common nutritional deficiencies and their effect on oral health is necessary for the practicing dentist.

Common Symptoms and Signs of Nutritional Deficiencies

These include:

- wasting of the body in calorie deficiency,
- skin rashes in vitamin deficiencies,
- oedema of extremities, wasting of muscles, thinning of and hair loss in protein deficiencies,
- nails exhibiting spooning in iron deficiency,
- mouth ulcers in riboflavin, niacin and iron deficiency,
- bleeding gums in vitamin C deficiency,
- bone deformities in calcium and vitamin D deficiencies.

Common Investigations in Nutritional Deficiencies

Investigations into nutritional deficiencies include serum albumin estimation in PEM, measurement of iron, vitamins and other specific elements depending on the suspected underlying disorder.

Nutritional Disorders of Dental Interest

- **Protein-energy malnutrition (PEM)**

Introduction: The World Health Organization (WHO) defines malnutrition as "the cellular imbalance between the supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions". [5] Protein-energy malnutrition (PEM) or protein calorie malnutrition (PCM) is an energy deficit due to chronic deficiency of all macronutrients (protein, fat and carbohydrates) which also includes deficiencies of micronutrients (minerals and vitamins).

PEM applies to a group of related disorders that include **marasmus, kwashiorkor** and an intermediate state called **marasmus-kwashiorkor**. The term marasmus in Greek means withering or wasting, whereas kwashiokor in the Ga language of Ghana means "the sickness of the weaning". Marasmus involves inadequate intake of protein and calories and is characterised by emaciation. Kwashiorkor refers to inadequate protein intake with reasonable caloric (energy) intake. Oedema is present in kwashiorkor but not in marasmus. PEM is common in the developing world. WHO figures in the year 2000 indicated that 181.9 million children in developing countries were affected by malnutrition. [6]

PEM may be primary and secondary. Primary PEM is caused by inadequate nutrient intake whereas secondary PEM results from disorders or drugs that interfere with nutrient use.

Marasmus (also known as the dry form of PEM) is characterised by the depletion of fat and muscle. There is considerable weight loss in these patients. This is the most common form of PEM in the developing world.

Low calorie intake or an inability to absorb calories is the key factor in the development of kwashiorkor.

Kwashiorkor is also known as the wet form of PEM. It is common in developing countries. It affects children who are being weaned. Premature abandonment of breastfeeding (when a younger sibling is born displacing the older child) is the most common cause of kwashiorkor. A diet insufficient in protein (cassava, banana, and sweet potatoes, for example) rather than energy is also associated with kwashiorkor. Kwashiorkor may also result from an acute illness or infection. In kwashiorkor, cell membranes leak and extravasation of the intravascular fluid and protein results in peripheral oedema.

Marasmic-Kwashiorkor is characterised by the features of both marasmus and kwashiorkor. Starvation due to voluntary fasting (anorexia nervosa, for example) or natural disasters (famine, for example) also causes PEM.

PEM also occurs in patients with wasting disorders (cancer, renal failure or AIDS, for example), gastrointestinal disorders (enteritis or enteropathy, for example) or conditions that increase metabolic demand (infections, hyperthyroidism, cystic fibrosis, congenital heart

disease or Addison's disease, for example). These are common factors contributing to malnutrition in the developed countries. Elderly patients are at a risk of PEM because of inadequate nutrition. Patients with gastric cancer and those on renal dialysis are also at a higher risk of malnutrition.

Symptoms and signs: Wasting of fat is common in all three forms of PEM. In marasmus, the child appears emaciated with a marked loss of subcutaneous fat and muscle wasting. The skin is xerotic, wrinkled, and loose. Buccal fat is lost, giving rise to the typical 'monkey facies'. Brittle hair or alopecia, and fissuring nails may be present.

Kwashiorkor is characterised by failure to thrive, oedema, moon facies, swollen abdomen (potbelly), and a fatty liver. The skin is dark, dry and splits open when stretched. This is common on the pressure areas. Depigmentation of hair causes it to be reddish-yellow to white. Curly hair becomes straight, lacklustre, sparse and brittle. Atrophy of papillae on the dorsum of the tongue and angular cheilitis, and xerophthalmia are common. Vitamin C deficiency can cause petechiae, gingival bleeding and splinter haemorrhages. Niacin deficiency may cause dementia, diarrhoea and dermatitis (pellagra) in advanced cases. Dermatitis in the sun-exposed areas of the neck and back (Casal's necklace), face, and dorsum of the hands is also seen.

Diagnosis/investigations: History and physical examination usually confirm the diagnosis. Laboratory tests are necessary to detect the causes of secondary PEM.

Prevention and management: A balanced diet is necessary to treat the milder forms of PEM. Multivitamins are also added. Severe PEM is treated in the hospital setting with a controlled diet. Fluid and electrolyte imbalances need to be corrected and infections should be treated promptly. Macronutrients should be given orally or, if necessary, through a feeding tube. Parenteral nutrition is required if malabsorption is severe. Underlying disorders must be identified and treated.

Complications of treatment must be avoided. When present they need to be addressed. These include fluid overload, hyperglycaemia, electrolyte deficits and cardiac arrhythmias.

- **Vitamin deficiencies**

Vitamins are classified as water soluble (vitamin B group and C) or fat soluble (vitamin A, D, E and K).

The B vitamins include biotin, folate, niacin, pantothenic acid, riboflavin, thiamine pyridoxine and B_{12}.

Biotin

Biotin acts as a co-enzyme which assists in the metabolism of carbohydrates and fats. Principal sources of biotin include liver, kidney, egg yolk, yeast, cauliflower, nuts and legumes. Deficiency of biotin results in dermatitis, glossitis and metabolic acidosis.

- **Folate (folic acid)**

Folates are necessary for RBC maturation and the synthesis of purines and pyrimidines. They are required for the development of the foetal nervous system. Folate is available in leafy vegetables, fruits, enriched cereals and breads, and meats. Its bioavailability is greater

when it is added to enriched grain foods. Prolonged cooking destroys folate. Folate deficiency is common. This occurs as a result of inadequate intake or malabsorption. Folate deficiency causes megaloblastic anaemia and neural tube birth defects.

- **Niacin (nicotinic acid, niacinamide)**

Niacin is vital in cell metabolism. Principal sources include red meat, fish, poultry, milk, legumes and enriched bread and cereals. A deficiency of niacin results in pellagra. This is characterised by dermatitis, glossitis, gastrointestinal and central nervous system dysfunction.

- **Riboflavin (vitamin B_2)**

Riboflavin is necessary for many aspects of carbohydrate and protein metabolism, and the integrity of mucous membranes. Principal sources of riboflavin include milk, cheese, meat, liver, eggs and enriched bread and cereals. Deficiency in riboflavin results in cheilosis, angular stomatitis and corneal vascularisation.

- **Thiamin (vitamin B_1)**

Thiamin is required for carbohydrate, fat, amino acid, glucose and alcohol metabolism. Principal sources of B_1 include pork, liver, whole grains and nuts. Thiamine is also necessary for myocardial function, and central and peripheral nerve cell function. A deficiency of thiamine includes beriberi, characterised by peripheral neuropathy, and heart failure.

- **Vitamin B_6 group (pyridoxine, pyridoxal and pyridoxamine)**

The vitamin B_6 group is necessary for many aspects of nitrogen metabolism, nucleic acid biosynthesis, linoleic acid, and lipid and carbohydrate metabolism. Principal sources include liver, whole grain, fish and legumes. Deficiencies result in seizures, anaemias, neuropathies and seborrhoeic dermatitis.

- **Vitamin B_{12} (cobalamins)**

Vitamin B_{12} is necessary for RBC maturation, neural functioning, DNA synthesis, and repair. Principal sources include beef, pork, fish, poultry, eggs and fortified cereals. A deficiency results in megaloblastic anaemia, neurologic deficits such as paraesthesia, and ataxia.

- **Vitamin C (ascorbic acid)**

Vitamin C is required for collagen formation, hormone and amino acid formation, and wound healing. Principal sources include citrus fruits, tomatoes, potatoes, broccoli, strawberries and capsicums (sweet peppers). A deficiency in vitamin C results in scurvy, characterised by haemorrhages, loose teeth, gingivitis and bone defects.

- **Vitamin A (retinol)**

Vitamin A is necessary for the formation of rhodopsin (a photoreceptor pigment in the retina), epithelial integrity and lysosome stability. Principal sources of this vitamin include fish liver oils, liver, egg yolks, butter and vitamin A fortified dairy products. Deficiency includes night blindness, xerophthalmia and keratomalacia.

- **Vitamin D (cholecalciferol, ergocalciferol)**

Vitamin D is necessary for calcium and phosphorus absorption and for resorption, mineralisation and maturation of bones. Sources include ultraviolet radiation of the skin, fortified milk, fish liver oils, butter and eggs. Deficiencies include rickets in children and osteomalacia in adults.

- **Vitamin K**

Vitamin K is necessary for the formation of prothrombin, other coagulation factors and bone proteins. Sources include green leafy vegetables, pork, liver, vegetable oils and soy beans. Vitamin K deficiency results in bleeding due to prothrombin decrease and other clotting factor deficiencies.

- **Vitamin E (Alpha tocopherol)**

Vitamin E is necessary as an intracellular antioxidant, and as a scavenger of free radicals in biologic membranes. Deficiency of vitamin E results in RBC haemolysis, neurologic deficits and cretinuria.

- **Obesity**

Definition: Obesity refers to severe excess body fat.

Cause: Almost all cases of obesity result from over-eating and inadequate exercise. A genetic predisposition has also been associated with obesity.

Complications of obesity include diabetes mellitus, cardiovascular diseases, cholilithiasis, cancers, fatty liver and cirrhosis, osteoarthritis, obstructive sleep apnoea and psychological disorders. Skin infections (fungal in particular) are common in the thick folds of the skin.

Diagnosis of obesity is assessed on body mass index, blood pressure, blood glucose and lipid levels.

Management includes dietary and behavioural modifications and sometimes, drugs and surgery. Prevention includes healthy eating, exercise and behavioural changes.

- **Metabolic syndrome**

Metabolic syndrome is characterised by excessive abdominal fat causing at least two of the following: insulin resistance, dyslipidaemia, and hypertension. Metabolic syndrome is very common. Complications, diagnosis and management are similar to those for obesity.

- **Balanced/healthy diet**

Eating a balanced or healthy diet means including a wide variety of foods and drinks from all of the food groups. It also means eating certain things in moderation, namely saturated fats, refined sugar, salt and alcohol. The goal is to take in nutrients that are needed for health at the recommended levels.

A healthy diet

The following information is useful to all health professionals and the community at large. Dentists have a responsibility to advise their patients by offering the following suggestions for a healthy diet.

The Nutrition Source at the Harvard School of Public Health makes the following 10 recommendations for a healthy diet.

- Choose good carbohydrates: whole grains (the less processed the better), vegetables, fruits and beans. Avoid white bread, white rice and the like, as well as pastries, sugared sodas, and other highly-processed foods.
- Pay attention to the protein package: good choices include fish, poultry, nuts, and beans. Try to avoid red meat.
- Choose foods containing healthy fats. Plant oils, nuts, and fish are the best choices. Limit consumption of saturated fats, and avoid foods with transfats.
- Choose a fiber-filled diet which includes whole grains, vegetables, and fruits.
- Eat more vegetables and fruits—the more colourful and varied, the better.
- Calcium is important, but milk is not its best source. Good sources of calcium are collards, bok choy, fortified soy milk, baked beans, and supplements which contain calcium and vitamin D.
- Water is the best source of liquid. Avoid sugary drinks, and limit intake of juices and milk. Coffee, tea, artificially-sweetened drinks, 100-percent fruit juices, low-fat milk and alcohol can fit into a healthy diet but are best consumed in moderation. Sports drinks are recommended only for people who exercise more than an hour at a stretch to replace substances lost in sweat.
- Limit salt intake. Choose more fresh foods, instead of processed ones.
- Moderate alcohol drinking has health benefits, but is not recommended for everyone.
- Daily multivitamin and extra vitamin D intake has potential health benefits.

Other than nutrition, the guide also recommends frequent physical activity (exercise) and maintaining a healthy body weight. [7].

Chapter 20

The Female Patient

S. R. Prabhu

Abstract

This chapter deals with gynaecological issues of the female patient, particularly the disorders of menstruation, menopause and pregnancy. Breast cancer and cervical cancer are also discussed briefly. Where relevant, dental management issues are also briefly discussed in this chapter.

Introduction

Females attending dental clinics may complain of symptoms associated with the reproductive system. Although the majority of gynaecological symptoms have very little impact on oral health, the dental practitioner is expected to be familiar with the various gynaecological terms used as well as the salient clinical features of some of the more common gynaecological disorders. In some instances (pregnancy, for example), the dental practitioner is expected to know the radiation hazards associated with x-rays during pregnancy, and also how to position the patient on the dental chair appropriately in order to avoid foetal asphyxia. Some aspects have been discussed briefly in this chapter.

Common Gynaecological Symptoms

Common gynaecological symptoms include pelvic pain, vaginal bleeding, vaginal discharge, irregular menstrual periods, painful menstruation, excessive blood loss during menstruation, or failure to menstruate during the normal reproductive years.

Common Gynaecological Investigations

Diagnostic methods in gynaecology include a thorough history, clinical examination and laboratory tests.

Tests include detection of human chorionic gonadotropin (hCG) in the urine sample for pregnancy, Pap smear test for suspected cervical dysplastic lesions, microscopic examination of vaginal secretions for microbial infections of the vagina, and culture or molecular studies for specific organisms (of STDs). Imaging of suspicious masses by ultrasound has become a common method in the clinic. Other tests include CT scans, MRI and laparoscopic examinations.

Disorders of menstruation

Amenorrhoea refers to failure to menstruate. This occurs during pregnancy, prior to puberty and after menopause. In all three of these situations amenorrhoea is physiological. Pathological amenorrhoea occurs if failure to menstruate lasts for at least six months during the normal reproductive years in the absence of pregnancy.

Diagnosis is by clinical examination, pregnancy testing and measurement of hormone (oestrogen) levels. Treatment is undertaken to identify and correct underlying conditions, such as endocrine dysfunction.

Oligomenorhoea refers to infrequent menstruation, particularly when the duration of the menstrual cycle exceeds the norm for the individual concerned by two weeks.

Menorrhagia refers to excessive blood loss during menstruation. Causes of menorrhagia include fibroids, endometriosis, carcinoma and bleeding disorders such as von-Willebrand's disease. These individuals are prone to develop iron deficiency anaemia. Diagnosis includes physical examination and tests such as pregnancy testing, FBC, and Hb estimation. Other tests include estimations of thyroid stimulating hormone (TSH) and serum progesterone levels, transvaginal ultrasonography, and uterine biopsy. Treatment includes detection and elimination of the underlying causes. In dysfunctional uterine bleeding without the structural abnormalities, oral contraceptives are effective.

Dysmenorrhoea refers to painful menstruation. Two types exist: primary and secondary dysmenorrhoea. **Primary dysmenorrhoea** is spasmodic and starts soon after the onset of menstruation (menarche) during puberty and lasting through the ages of 15-25 years. Colic pain in the suprapubic region radiating to the lower back, groin and thighs is the major symptom.

Secondary dysmenorrhoea refers to pain which arises as a result of an underlying condition. It usually begins in older women in the third or fourth decade. Pain in the lower back and pelvis starts several days before menstruation. Endometriosis and pelvic inflammatory disease may cause secondary dysmenorrhoea. Diagnosis is by ultrasonography. Pain is treated with NSAIDs. Underlying disorders causing dysmenorrhoea should be identified and treated.

Disorders of Menopause

Menopause is physiologic cessation of menses due to decreasing ovarian function. Menopausal manifestations occur after the cessation of menstrual cycles between 40 and 58 years of age (a mean age of 51).

Symptoms of menopause include hot flushes, sweating, insomnia, depression, atypical facial pain and dysaesthesia. Menopausal women are prone to developing osteoporosis. A diagnosis of menopause is likely if an absence of menses for one year is reported. Pregnancy testing may be necessary in some cases. Hormone replacement therapy (HRT) for women during menopause is common. Current evidence, however, points to an increased risk for breast cancers among those who are on long term HRT.

Menopausal Oral Symptoms

- During menopause, women often complain of oral symptoms. These include mucosal burning sensations, altered taste, and a dry mouth. Disorders such as Sjögren's syndrome and burning mouth syndrome need to be investigated in these patients.
- Women taking HRT have an increased risk of dry sockets after tooth extractions.

Management of oral symptoms depends upon the underlying causes. Symptomatic treatment is adequate if no underlying disorder is identified.

- **Pregnancy**

Full term pregnancy is considered to be 40 weeks from the first day of the woman’s last menses. Pregnancy is divided in to trimesters, each lasting approximately 3 months.

Common Symptoms during Pregnancy

First trimester (0-12 weeks): Missed menstrual periods, swelling and tender breasts, fatigue, nausea and vomiting (especially worse in the morning), hunger, food cravings and frequent urination are common in the first 12 weeks (first trimester) of pregnancy.

Second trimester (12-28 weeks): Symptoms during the second trimester include external signs of pregnancy, darkening of the skin, darkening and enlargement of the nipples, increased appetite and increased heart rate.

Third trimester (29-40 weeks): Common symptoms during the third trimester include lower backache, abdominal enlargement and discomfort, constipation, haemorrhoids, swollen ankles and frequent urination.

Diagnosis and Investigations in Pregnancy

These include a history, a physical examination, pregnancy tests and ultrasonography. Conventional x-rays are to be avoided because of their hazardous effects on the foetus during early pregnancy. If x-rays are unavoidable, a uterine shield with a lead apron should be provided during exposure. Prenatal testing during pregnancy to detect foetal defects includes the estimation of alpha-fetoprotein (AFP). AFP is produced by the foetal liver. Abnormally low levels of AFP may indicate Down syndrome, whereas abnormally high levels indicate neural tube abnormalities of the foetus. Amniocentesis is used prenatally to diagnose chromosomal defects and infectious diseases of the foetus. Other tests during pregnancy include those used for sexually transmitted diseases, HIV disease and gestational diabetes.

Pregnancy Related Disorders

- **Pre-eclampsia**

Definition/Description: Pre-eclampsia is a medical condition characterised by high blood pressure (of more than 30 mm of Hg systolic, or 15 mm of Hg diastolic from measurements early in pregnancy), and significant amounts of protein in the urine (of more than 3 g per day) in a pregnant woman. If left untreated, it can develop into eclampsia, which is a life-threatening condition characterised by the occurrence of seizures during pregnancy.

Cause/risk factors: These include a first pregnancy under the age of 17 years, or over the age of 35. A family history of hypertension, poor diet during pregnancy, obesity, smoking and diabetes are also considered to be important risk factors.

Symptoms and signs include oedema of the hands, feet and face (pitting oedema). Breathing and kidney problems are common, and seizures and stroke in rare cases. Babies will have growth problems. In most cases, high blood pressure resolves after delivery.

Investigations: Clinical examination. Predictive tests include blood test for quantification of placental growth factor (PGF) late in the first, or during the second trimester. Urine tests are indicated for specific cells called podocytes. All pregnant women with pre-eclampsia are positive for these cells.

Management: The only known treatments for eclampsia or advancing pre-eclampsia are abortion, or delivery by labour induction or Caesarean section. Hypertension can sometimes be controlled with anti-hypertensive medication.

Oral Manifestations and Dental Management Considerations

- During pregnancy, the risk of dental caries may increase because many pregnant women often have a craving for cariogenic food. Attention to oral hygiene also suffers during pregnancy which may contribute to the increased risk of caries and periodontal disease.
- Gingivitis among pregnant women (pregnancy gingivitis) is a common feature. Pregnancy gingivitis occurs in a significant percentage of pregnant women as

generalised or localised gingival inflammation. The gingival response to bacterial plaque is exaggerated during pregnancy due to hormonal imbalances. Gingival tissue (usually in the anterior part of the mouth) is swollen, red and bleeds easily. The inflammatory process begins around the second month of pregnancy and peaks in the middle of the third trimester. Pregnancy gingivitis usually disappears after childbirth.

- The so called 'pyogenic tumour" of the gingival tissues (pregnancy epulis) occurs in a smaller percentage of pregnant women. This is an example of pyogenic granuloma occurring in pregnancy.
- Some studies suggest that periodontal disease in pregnant women can induce preterm birth (PTB) or low birth weight (LBW) babies. This hypothesis has been seriously challenged by recent research studies.
- Good oral hygiene during pregnancy is essential.

Dental Management Considerations

- Increased treatment disruptions during dental procedures are common among pregnant women because of the increased frequency of urination, caused by increased foetal pressure on the bladder.
- During the second and third trimesters pregnant woman may have difficulty sitting on the dental chair in a reclining or semi-supine position. In this position, compression on the superior vena cava by the gravid uterus can cause hypotension, decreased cardiac output and loss of consciousness. To relieve this pressure during the second trimester it is advisable to use a support (a pillow, for example) under the right hip and buttocks to create a 15 degree elevation. This can prevent a hypotensive event. In the third trimester, the pregnant woman should be turned onto her left side (left lateral decubitus position) to prevent compression on the superior vena cava by the gravid uterus.
- Elective dental treatment is best provided during the second trimester and early part of the third trimester of pregnancy. Emergency treatment can be provided at any time of the pregnancy after consulting with the patient's obstetrician.
- Dental x-rays are contraindicated during pregnancy, particularly during the first trimester when foetal organogenesis is taking place. However, if an emergency demands the need for x-rays, precautions to protect the mother and the foetus should be taken by using protective abdominal and thyroid shielding. Lower radiation-emitting digital radiographs can be used in these situations.
- Drugs known to have no adverse effects on the developing foetus can be used during pregnancy.
- Drugs that are known to have adverse effects and potential risks to the foetus include aspirin, bupivacaine, mepivacaine, procaine, and codeine. In cases of emergency these may be used in consultation with the obstetrician. Drugs to be avoided include tetracyclines, thalidomide and benzodiazepines. Fluconazole as an antifungal agent is also not recommended during pregnancy.
- General anaesthesia during the third trimester is contraindicated.

Breastfeeding Mothers

- Local anaesthesia administered to the lactating mother is safe.
- Acetaminophen, codeine, tylenol and oxycodone are safe for use by the lactating mother.
- Penicillins, cephalosporins and clindamycins are safe, but preferably should be taken after breastfeeding. Tetracyclines are contraindicated during the lactation period.

Women on Contraceptives

- Should a dentist need to prescribe antibiotics (for oral infections) to a woman taking oral contraceptives, the patient must be advised of the potential risk for the antibiotic to reduce the effectiveness of such oral (hormonal) contraceptive.

- **Gestational diabetes (gestational diabetes mellitus)**

Definition/description: Diabetes that develops during pregnancy, in women without previously diagnosed diabetes, is called gestational diabetes. Often this is referred to as type III diabetes and usually occurs in the third trimester.

Symptoms and signs: Thirst, frequent urination, weight loss and weakness are signs. Gestational diabetes can lead to pre-eclampsia and foetal abnormalities.

Investigations include blood and urine tests for blood sugar.

Management includes regular blood glucose monitoring, a diet low in sugar and fat, exercise, and insulin injections if required.

Oral Manifestations and Dental Management Considerations

- There are no specific oral manifestations in patients with gestational diabetes.
- Susceptibility to oral infections (such as thrush), and periodontal inflammation may increase.
- Burning mouth syndrome and altered taste may also be reported by these patients.

Management considerations

- Oral hygiene instructions,
- Caries prevention protocol,
- Management of xerostomia, and
- Appropriate antifungal treatment (fluconazole is to be avoided).

- **Breast cancer**

Definition/Description: Breast cancer is the most common cancer occurring in women. Ductal and lobular forms of breast cancers occur either as invasive or *in situ* types.

Cause/risk factors: Genetic factors play a major role in the causation of breast cancer (mutations in BRCA-1 and BRCA-2). A first degree relative (mother, sister, or daughter) with breast cancer, prior exposure to high levels of radiation or prior treatment for Hodgkin's disease pose higher risks for breast cancers.

Symptoms and signs: Most breast cancers are discovered as lumps by patients or during routine physical examination or mammography. Initial symptoms may include pain, swelling or thickening in the breast.

A lump distinctly different from the rest of the breast tissue is a clinical sign that may suggest breast cancer. Fixation of the lump to the underlying tissues (chest wall), or to the overlying skin, is a sign of an advanced stage of breast cancer. Matted or fixed axillary nodes and supraclavicular or infraclavicular lymphadenopathy suggest tumour spread.

Diagnosis/investigations: These include mammograms (bilateral), needle biopsy or incisional biopsy, stereotactic biopsy (needle biopsy during mammography), and ultrasound-guided biopsy. Diagnosis is confirmed by biopsy.

Management: the primary treatment for most patients is surgery, often with radiation. Chemotherapy and hormone therapy are also used.

Oral Manifestations and Dental Management Considerations

- In advanced cases of breast cancer, metastasis to the lungs, bone and liver may occur. Jaw bone metastasis is also a possibility.
- Those receiving chemotherapy may present oral mucositis.
- Intravenous bisphophonates are used in patients with breast cancer metastasis to the bone. These patients are prone to develop antiresorptive agent induced osteonecrosis of the jaws (ARONJ).These patients need antibiotic and conservative surgical procedures. Patients should be advised of oral hygiene home care procedures.

- **Cervical cancer**

Definition/description: Cervical cancer is usually a carcinoma caused by the human papillomavirus (HPV) infection or an adenocarcinoma.

Symptoms: Early cancers are asymptomatic, or there is irregular vaginal bleeding between menses. Late stage cancer usually manifests in postcoital vaginal bleeding, foul smelling vaginal discharge and/or pelvic pain. Back pain, and leg swelling due to lymphatic or venous obstruction may be present in more widespread cancers. The majority of cervical cancers develop from cervical intraepithelial neoplasia (CIN).

Diagnosis is by cervical screening using the Papanicolaou (Pap) test and biopsy. Clinical staging is carried out based on cervical biopsy results. CT or MRI are also useful in detecting metastasis.

Management: Management protocols include radical surgical hysterectomy, pelvic radiation and chemotherapy.

Chapter 21

Dental Management of Patients Taking Medications for Systemic Disorders: An Overview

S. R. Prabhu

Abstract

This chapter deals with the dental management of patients taking medications for systemic conditions. Topics include dental management of those who are on anticoagulant and antiplatelet therapy, systemic corticosteroid therapy, chemotherapy, radiation therapy, immunosuppressant drugs and also those on medications for diabetes, asthma, cardiovascular diseases, seizures and HIV disease. Dental aspects of those who are known to have alcohol and substance abuse are also discussed in this chapter.

Introduction

Many patients attending dental practices are on multiple medications for systemic disorders. Since these medications may pose potential problems in dental management, it is important that a detailed medical and drug history should be obtained by the dentist before the commencement of dental procedures. Patients must be encouraged to bring a list of the medications they take. This will help practicing dentists to record the name, strength and dose of the medications. This chapter deals with some of the common medications used by the medically compromised patient and the dental management issues associated with such use.

- **Dental Management of Patients Taking Anticoagulant and Antiplatelet Therapy**

Definition: Anticoagulants are medications which prevent or reduce the clotting of blood. Anticoagulants and antiplatelet medications prevent deep vein thrombosis, pulmonary embolism, myocardial infarction and stroke.

Drugs: Drugs used in anticoagulant therapy include warfarin, heparin, aspirin, clopidogrel, dipyridamole and glycoprotein IIb/IIIA inhibitors. New oral anticoagulants include dabigatran, apixaban, and rivaroxaban.

Dental implications: Patients taking anticoagulants have an increased risk of bleeding during oral surgery if the drug is not discontinued. On the other hand, if the drug is stopped, they have an increased risk of developing intravascular thrombi and emboli**.** Since intravascular events can be potentially serious, it is recommended that the anticoagulant therapy should be continued without altering the dose, and that any intraoral bleeding should be managed using appropriate local haemostatic measures.

Patients taking aspirin have a normal INR and prolonged bleeding time. Extraction wounds usually do not result in extensive bleeding. There is no indication that the dose of aspirin for dento-alveolar surgery be altered. Patients should be informed of the possibility of slightly excessive bleeding or bruising. Haemostasis should be achieved in the dental practice by local haemostatic means.

Patients taking warfarin should be allowed to continue taking the medication as prescribed by the physician for their medical condition, however, the following steps must be taken prior to the commencement of oral surgical procedures:

- **A Detailed history** of the medical condition and drug history must be recorded.
- **INR estimation must be made 24 hours before surgery**: if INR is below 2.2, continue with the procedure. If INR is between 2.2 and 4, use tranexamic acid mouth wash and continue with the procedure. If INR is greater than 4, refer the patient to the physician.

The effect of warfarin is altered by antibiotics and NSAIDs. The use of NSAIDs should be avoided.

- **Dental management of patients taking systemic corticosteroid therapy**

Corticosteroids are used in inflammatory conditions, adrenal insufficiency, immunological disorders, and for asthma. Doses greater than 5 to 10 mg of corticosteroids (prednisone or prednisolone, for example) per day for more than a few weeks may be sufficient to cause adrenal suppression. The adverse effects of adrenal suppression resulting from long term steroid use may cause diabetes, peptic ulceration, osteoporosis, skin atrophy, higher risk of infections, and immunosuppression.

In patients on long term steroids, stress caused prior or after surgery may result in adrenal crisis (Addisonian crisis). This is characterised by circulatory collapse, dehydration, hypoglycaemia, and hypotension. Adrenal crisis is a medical emergency. If not promptly treated, the condition can be fatal. In order to prevent an adrenal crisis, the patient should be asked to take double the normal dose prior to the commencement of surgery. Consult the patient's physician before carrying out surgical procedures on these patients.

- For non-surgical dental procedures, steroid supplementation is not required for those currently taking steroids.
- For surgical dental /oral procedures, patients currently on steroids should double the normal dose of steroids on the day of procedure (for LA procedures).

- For procedures under GA, the anaesthetist's guidelines need to be followed. Patients may need a higher dose of hydrocortisone on the morning of the procedure.
- Patients on steroids are susceptible to oral candidal infections.
- Steroids also interfere with healing and may cause delayed healing.

- **Dental management of patients taking medication for diabetes mellitus**

Most diabetic patients attending dental clinics have a well-controlled medication, diet and exercise regimen. They monitor glucose levels regularly. Occasionally however, instability of diabetic control may occur due to factors such as oral infections.

It is important that diabetic patients maintain their calorie intake, medication and activity level even if their oral symptoms limit their ability to eat.

Mid-morning or early afternoon appointments should be scheduled, and prolonged appointments avoided. Prior to treatment, ensure that the patient has eaten at least 30 minutes before the start of the procedure.

A sweet drink should not be given unless it becomes obvious that the patient is showing signs of hypoglycaemia.

Blood glucose levels can be checked using a glucometer (which the patient should be advised to bring with them) before and during dental procedures.

- **Dental management of patients taking medication for asthma**

Most importantly dental practitioners should avoid triggering an asthmatic attack during treatment of a patient with a history of asthma. This can happen if the practitioner gives the patient aspirin or NSAIDs prior to or during the procedure. Paracetamol is the drug of choice for these patients.

If patients are on systemic steroids, the dose should be increased before the treatment commences in order to avoid an adrenal crisis.

Patients regularly using steroid inhalers should be asked to bring them to the dental appointment.

Inhaled steroids often cause oral candidosis. Patients must be advised to use spacers and rinse their mouth with water (and spit out) after inhalation.

- **Dental management of patients taking medication for cardiovascular disorders**

Hypertensive patients may show an increased risk of post-operative bleeding. The risk of angina or myocardial infarction also increases in these patients.

If the BP is up to 160/100 mmHg, the patient can be considered for routine dental treatment. If the reading is above this limit, referral to the physician for investigation and control of hypertension is necessary before elective surgery. The use of intravenous sedation with midazolam is recommended in uncontrolled hypertension. Blood pressure needs to be recorded throughout the surgical procedure and also post-operatively before the patient is discharged.

Angina. Common medications used by patients with a history of angina include glyceryl nitrate spray or tablets, antiplatelet therapy (aspirin, for example) and beta-blockers. These patients have a higher risk of an angina attack or myocardial infarction.

It is advisable to ask patients to use GTN before the surgical procedure. There is no need to alter or stop antiplatelet medication. Oxygen should be made available to address any emergency situation.

Myocardial infarction: For patients with recent (under 3 months) MI, no elective surgical treatment should be considered**.** General anaesthetic should be avoided for those who have had MI in the preceding six months. These patients have an over 50 % increased risk of a repeat MI.

Other cardiac problems: For those with cardiac defects or valve replacements, a history of previous endocarditis, or hypertrophic cardiomyopathy, current evidence indicates that antibiotic cover prior to surgical procedures is not required. Good oral hygiene should be maintained, and patients should be informed of the (unlikely) possibility of infective endocarditis including the signs and symptoms.

- **Dental management of patients taking immunosuppressants**

Immunosuppressant therapy patients: In general, deliberately induced immunosuppression is performed to prevent the body from rejecting an organ transplant, treating graft-versus-host disease, after a bone marrow transplant, or for the treatment of auto-immune diseases such as rheumatoid arthritis, vasculitis or Crohn's disease. This is done using immunosuppressant drugs. Immunosuppressant drugs include azathioprine, cyclosporine, and monoclonal antibodies including basiliximab and munomorab, and corticosteroids.

Patients on immunosuppressant therapy have an increased risk of infections and cancers. A thorough history and detailed oral examination is recommended prior to any dental procedures being undertaken.

Annual immunisation for influenza, pneumococcal vaccination at baseline and one time revaccination after five years is recommended for these patients.

- **Dental management of the solid organ transplant patient**

In the developed countries, solid organ transplants are becoming common. Solid organ replacements include those of the heart, intestine, kidneys, liver, lungs and pancreas. Organ transplant patients need specialised oral care. These patients are at a greater risk of systemic as well as oral infections. When planning dental treatment this aspect must be taken into consideration and the patient’s specialist must be consulted before commencement of dental treatment. Two important aspects which need to be considered are the management of the patient’s oral health before an organ transplant, and oral health management afterwards.

Before Organ Transplantation

- Obtain the patient’s medical and dental history.
- Perform a non-invasive initial oral examination (without periodontal probing).

- Consult the patient's physician or specialist regarding the patient's current status of health and that of their immune system, the degree of organ dysfunction, a decision on the timing of treatment, the need for antibiotic prophylaxis, any precautions to prevent excessive bleeding, appropriate medication and dosage, and whether patient is in a position to tolerate dental treatment.
- Non-restorable teeth should be extracted.
- Procedures should not be carried out on kidney transplant patients on their days of renal dialysis.
- Educate patients on the importance of oral hygiene maintenance.

After Organ Transplantation

- Dental treatment should be given only after patient's transplant has stabilised, and elective treatment should be postponed for at least three months following the organ transplant.
- Check the patient's BP every time before commencement of treatment. If BP exceeds the baseline reading, their physician should be contacted.
- Check with the physician if patient needs antibiotic prophylaxis.
- Those on corticosteroids may face adrenal crisis, so consult the physician about increasing the dose prior to invasive treatment.
- Prescribe antimicrobial mouth rinse and saliva substitutes where appropriate.
- Alcohol and smoking should be discouraged.

Specific issues

- For heart transplant patients, use LA without a vasoconstrictor.
- For lung transplant patients, avoid narcotic analgesics,
- For liver transplant patients, use drugs that are not metabolised by the liver.
- For kidney transplant patients, use drugs with caution (considering renal elimination of the drug).
- For pancreas transplant patients, evaluate blood sugar levels.

Oral Manifestations in Transplant Patients

As a result of long term immunosuppressive therapy the following oral manifestations can occur in transplant patients:

- Susceptibility to infections such as candidosis, cytomegalovirus infections (common in the first month after the transplant), herpes virus infections, herpes zoster and HHV-8 infections and Epstein-Barr virus (EBV) infections (hairy leukoplakia). There is also an increased risk of malignant or potentially malignant lesions.
- Gingival hyperplasia in those who are on long term cyclosporine.

- **Dental management of patients receiving chemotherapy and radiation therapy for cancer**

- All dental care must be completed before cancer therapy is initiated. If this is not possible, then prioritise the treatment procedures.
- Stabilise or eliminate any existing and potential sources of infection in the mouth.
- Communicate with the oncology team regarding the oral health status and the timing of treatment.
- Educate the patient regarding the importance of optimal oral care.
- The risk of bleeding due to thrombocytopenia is high.
- Anaemia is common.
- Patient's blood counts: if absolute neutrophil count is below the normal range, antibiotic cover is recommended
- If the platelet count is below the normal range, platelet transfusions are recommended.
- When mucositis occurs, the following measures are recommended:
  - Good oral hygiene
  - Analgesics
  - Non-medicated oral rinses
  - The use of mucosal coating agents(kaopectate, for example)
  - The use of palifermin (keratinocyte growth factor-1) for prevention of mucositis
  - In patients receiving radiotherapy, chlorhexidine mouth wash is no longer recommended (for the prevention of mucositis)
  - For patients on radiotherapy, extractions in a hospital setting should be performed up to three weeks prior to the start of radiation therapy
  - Before surgery, antibiotic prophylaxis is recommended for some patients
  - Post-operative antibiotics need to be given until the surgical wound is healed
  - The patient must be advised not to wear dentures until healing is complete
  - Osteoradionecrosis is a complication of radiation therapy
  - A topical anaesthesia may be used for pain

- **Dental management of patients taking medication for seizures**

- If a patient's epilepsy is poorly controlled, surgical procedures are best performed in a hospital setting.
- Frequency of seizures must be assessed.
- History on what triggers seizures must be sought.
- Check if the patient has taken medication for the day. It is important to note that a person with stable epilepsy who has not taken his/her medication in the last 12-48 hours is potentially at risk of a seizure.
- Avoid stressful procedures.
- Treatment under intravenous sedation is recommended.
- Consider the use of a mouth prop (some do not recommend this) in order to avoid injury to the operators fingers if an acute generalised attack occurs.
- Protect the patient's airway.
- Some patients develop generalised gingival hyperplasia (dilantin hyperplasia).

- **Dental management of patients taking medication for osteoporosis**

  - Patients with osteoporosis are on anti-resorptive agents such as bisphosphonates (Fosamax) intravenous annually, and RANKIL inhibitors (6-monthly) to treat osteoporosis. These drugs are also used for metastatic malignancies.
  - Patients have a risk of developing anti-resorptive agent induced osteonecrosis of the jaws (ARONJ).
  - Prior to starting anti-resorptive therapy, a thorough dental examination is required.
  - Antibiotic prophylaxis may be required for some patients requiring surgery.
  - Post-operative antibiotic cover is recommended.
  - Oral infections must be eliminated.

- **Dental management of patients taking HIV medication**

  - HIV patients with a CD4 count below 200: a full blood count is required prior to the start of surgical treatment. If significant leucopoenia is present, antibiotic prophylaxis is recommended.
  - If viral load is less than 50 viral RNA copies/mm blood, infectivity is low. The patient is suitable for treatment.
  - Oro-facial infections should be treated vigorously.
  - Steroid cover is to be made available for those on corticosteroids in order to avoid adrenal crisis.
  - If the bleeding tendency is high due to thrombocytopenia, platelet transfusion may be necessary if the platelet count is below 200 cells/mm of blood. If above 200 cells/mm of blood, invasive dental treatment can be given.
  - A liver profile, full blood count and coagulation screen should be sought before surgical procedures.
  - If exposure occurs, post-exposure prophylaxis (PEP) should be considered for up to 4 weeks afterwards.
  - Consultation with an HIV expert is recommended.

- **Dental management of patients taking medication for psychiatric disorders**

  - Drugs commonly used in dentistry rarely have any interactions with medications used in psychiatric diseases.
  - Local anaesthetics with vasoconstrictors are acceptable for patients on tricyclics, antidepressants and selective serotonin uptake inhibitors (SSRIs).
  - For those on monoamine oxidase inhibitors (MOIs), local anaesthetics with vasoconstrictors should be avoided as they may interact adversely.
  - Those on lithium can have serious underlying psychiatric disorders but may look normal. Consultation with their psychiatrist is useful.
  - Dental practitioners should identify drug-dependant patients.

- **Dental management of patients with alcohol and substance abuse (addiction disorders)**

Addiction forming substances include alcohol, methamphetamine, cocaine, marijuana, tobacco and opiate drugs such as morphine, heroin, meperidine, hydromorphone, methadone and codeine.

Alcohol and opiate drugs are CNS depressants ("downers") whereas marijuana and cocaine are CNS stimulants ("uppers"). Methamphetamine is also a CNS stimulant.

Addiction, as defined by the American Society of Addiction Medicine in 2011 is "characterized by inability to consistently abstain, impairment in behavioral control, craving, diminished recognition of significant problems with one's behaviors and interpersonal relationships, and a dysfunctional emotional response." [8]

Alcohol abuse and alcoholism is a primary chronic disease with genetic, psychosocial and environmental factors influencing its development and manifestation. It is characterised by impaired control over drinking, preoccupation with alcohol, use of alcohol despite adverse consequences and distortion in thinking, most notably denial, and each of these symptoms may be periodic or continuous.

Oral Manifestations of Substance Abuse

- **Alcohol** abuse can cause adverse psychosocial and physical changes. Dental aspects include poor oral hygiene, xerostomia, increase in decayed, missing and filled teeth, increased incidence of periodontal disease and dental attrition due to bruxism. Angular cheilitis, candidal infections and impaired wound healing are the other oral problems associated with alcohol abuse.
- **Cocaine abuse** can result in movement disorder. Additionally, buccolingual dyskinesia (diminished voluntary movements), which is commonly known as 'crack dancing' or boca torcida (twisted mouth), can also be present.
- In the case of **heroin abuse**, increased numbers of decayed, missing, and filled teeth can be detected in the mouth. This can result from chronic malnutrition, poor oral hygiene due to impaired motor function and neuropathology secondary to infection, vasculitis, septic emboli, thromboembolism, prolonged heroin-induced respiratory depression, hypotension, positional vascular compression, acute Parkinsonism, and spongiform leukoencephalopathy.
- **Marijuana abuse** can lead to acidic erosion of enamel due to cannabinoid hyperemesis, in which frequent episodes of vomiting occurs. Additionally, these patients develop dental caries, inflammation and hyperplasia of the gingival tissues, uvulitis, leukoplakia, oral papillomas, and tongue carcinoma. The underlying pathology includes poor oral hygiene due to short-term and long-term debilitating psychological effects, xerostomia, alveolar bone loss, and carcinogens in marijuana. Increased incidences of HIV have also been reported.
- **Chronic methamphetamine abuse,** which leads to "meth mouth", resulting in large carious lesions in buccal smooth surface areas and fractured teeth. Xerostomia is common in these individuals. Bruxism, clenching and fracture of the compromised dentition due to increased motor activity are also common. Periodontal diseases are

common. Osteoporosis has also been reported in a high percentage of methamphetamine abusers.

Dental management

Dentists should be vigilant in identifying patients with alcoholism or substance abuse.

History taking should be carefully conducted using neutral or non- judgemental questioning. Once the patient has been identified with alcoholism or substance abuse (from the history and oral exam), a physician should be contacted for more information on their systemic health. Screening for bleeding and clotting disorders must be carried out prior to any invasive procedures on these patients.

A decreased drug effect is common in alcoholics because of liver damage. Hepatotoxic drugs should be avoided.

Strict infection control measures must be followed at all times.

- **Dental management of patients on renal dialysis**

Dialysis is a medical procedure that artificially filters the blood. Techniques include haemodialysis and peritoneal dialysis.

- In haemodialysis, the patient's blood is pumped through the blood compartment of a dialyser, exposing it to a partially permeable membrane. There are clinical benefits of dialysing 5 to 7 times a week for 6 to 8 hours, preferably in a hospital renal unit.
- In peritoneal dialysis, a sterile solution containing glucose (called dialysate) is run through a tube into the peritoneal cavity, where the peritoneal membrane acts as a partially permeable membrane. Peritoneal dialysis is carried out at home by the patient, often without help. Peritoneal dialysis is repeated 4-5 times per day.

All modalities exchange solute and remove fluid from the blood using dialysis and filtration across permeable membranes.

Patients receiving peritoneal dialysis generally do not pose any problems in dental management. Those receiving haemodialysis may present problems. Dentally induced trauma in these patients may be a source of infectious emboli that can cause infective endocarditis.

The following precautions should be taken by the dentist in treating patients on renal dialysis:

1. Consult with the patient’s physician,
2. If kidney disease is poorly controlled, avoid dental treatment,
3. Screen for bleeding disorders before surgery,
4. Monitor blood pressure, but avoid using a blood pressure cuff and intravenous medications in the arm with a shunt,
5. Avoid dental treatment on the day of dialysis,
6. Avoid nephrotoxic drugs,
7. Manage oral infections,
8. Screen for HBsAg before treatment; treat the patient as a potential carrier.

Chapter 22

Medical Emergencies

S. R. Prabhu

Abstract

This chapter deals with medical emergencies and their management aspects in the dental setting. Beginning with dental clinic preparedness and the procedures involved in recording vital signs in an emergency situation, topics then discussed include the management of syncope, hypoglycaemia, angina, acute myocardial infarction, cardiac arrest, asthma, hyperventilation, airway obstruction, urticaria, anaphylactic shock, angioedema, seizures, stroke, panic attack and adrenal crisis.

Introduction

Though uncommon, medical emergencies in dental practice can occur. Medical emergencies range from transient problems to serious emergencies that require the transfer of patients to medical facilities. The best way to avoid medical emergencies is by careful assessment of the patients when obtaining detailed medical and drug histories. When medical emergencies occur in the dental clinic, prompt diagnosis and immediate management can affect the outcome. Dental practitioners and staff must be trained and have a plan for emergency management. Dental students must receive adequate training in this regard as well. In this chapter some aspects of dental clinic preparedness and management of medical emergencies in dental practice are briefly discussed.

Dental Clinic Preparedness

For all new patients:

1. Obtain a thorough medical and drug history,

2. Perform a thorough physical examination, and
3. Record baseline vital signs.

Equip your clinic with:

Emergency equipment:

- Blood pressure monitor
- Tourniquets
- Stethoscope
- First-aid kit
- Oxygen tank (portable)
- Nasal cannula
- Masks (non-rebreather and bag-valve mask)
- Syringes: 3 cc disposable syringe
- Albuterol inhaler (with spacer) or solution and apparatus for nebulisation
- Blood glucose monitor
- Pulse oximeter
- Laryngeal airways
- Automated external defibrillator (AED)
- Emergency cabinet (centrally located)
- Emergency phone number call list (posted in the reception)

Emergency drugs:

- Albuterol (bronchodilator for asthma or COPD)
- Aluminium chloride (haemostatic)
- Aminocaproic acid (haemostatic)
- Ammonia spirit, aromatic (respiratory stimulant for fainting)
- Aspirin (analgesic for dental pain and for prophylaxis of MI and transient ischaemic attacks)
- Cellulose (oxidised/regenerated) to control bleeding during a dental procedure
- Collagen (absorbable) to control bleeding during dental surgery
- Dexamethasone (anti-inflammatory and antiemetic) for oral manifestations of an allergic, inflammatory or autoimmune nature
- Dextrose (oral) for hypoglycaemia
- Diazepam: an anti-anxiety agent for preoperative dental anxiety
- Diphenhydramine hydrochloride for hypersensitivity reactions
- Epinephrine for anaphylactic reactions (including Epipen)
- Fibrin sealant kit for haemostasis
- Gelatin (absorbable) for haemostasis
- Glucagon for hypoglycaemia
- Glucose for hypoglycaemia
- Hydrocortisone for allergic reactions

- Lorazepam for anxiety disorders
- Methylprednisolone, a corticosteroid (systemic) for inflammatory or allergic conditions
- Morphine sulphate, a narcotic for severe pain
- Nitroglycerine, an anti-angina agent (vasodilator)
- Oxygen, a resuscitative agent for medical emergencies in the dental office
- Promethazine, an antiemetic and for allergic reactions

Medical Emergencies in Dental Practice

Some Diagnostic Vital Signs of Medical Emergency

1. Pulse

Rapid (>120 bpm) and full: panic attack, high BP, heat stroke
Rapid and thready: shock, blood loss, heat exhaustion, diabetic coma
Slow and full: stroke
No pulse: cardiac arrest.

Respiration

Rapid, shallow: shock, cardiac dysfunction, insulin shock, heart failure
Deep, gasping, laboured: airway obstruction, heart failure, heart attack, diabetic coma
Snoring: CVA/stroke, drug or alcohol
Gurgling: airway obstruction, lung disease
Coughing blood: punctured lung, fractured rib.

Blood Pressure

BP <90 mmHg systolic: hypotension (patient seated or lying down)
BP drops to 90/60 mmHg: patient is going into shock
BP >140/90 mmHg: hypertension.

Other:

Temperature: cold and clammy, a sign of anxiety
Pupil size: equality and response
Lips, tongue, ear lobes and nail beds: colour
Breath odours
Muscular activity: spasms, weakness

Emergencies

Loss of Consciousness

Syncope

Definition: Syncope is a transient self-limiting loss of consciousness (simple fainting) due to the decreased circulation of blood to the brain (transient cerebral hypoperfusion).

Causes: Neurally mediated syncope is vasovagal or vasodepressive. Orthostatic (including due to the effect of drugs) and cardiac causes (arrhythmia or structural heart disease) can also cause syncope. Vasovagal syncope is the most common form.

Symptoms include anxiety, pallor, cold and clammy (diaphoresis), rapid pulse, decreased BP, loss of consciousness, dilatation of pupils and nausea.

Management of Syncope: (this also applies to orthostatic hypotension)

If the patient feels faint:

1. Tilt the chair back to a *horizontal* position. (NOT 'head lower than heart' position). This is called the Trendelenburgh position
2. Raise patient's legs slightly
3. Assess patient's level of consciousness by talking to them.

If the patient is unconscious:

1. Tilt the chair back to a horizontal position (NOT 'head lower than heart' position)
2. Place the patient on their side. (If pregnant, on to her left side)
3. Stimulate (by placing a crushed ammonia capsule under the nose) and cool the patient by placing a cold compress on their forehead
4. Monitor vital signs.

Those with vasovagal syncope recover rapidly. Reassure and comfort the patient when they awake.

If there is delay in response (unconscious for nearly a minute and with unstable vital signs), consider:

- Hypoglycaemia
- Seizure
- Cardiac arrest
- Anaphylaxis
- Cerebrovascular accident (CVA) or
- Transient ischaemic attack (TIA).

Call for Medical Assistance

Hypoglycaemia

Definition: Low blood glucose levels (less than 3 to 5 mmol/L) can cause symptoms and signs.

Causes include inappropriately high doses of insulin (in diabetes patients), delayed or forgotten meals, insufficient carbohydrates (especially in patients taking rapid-acting insulin) or unaccustomed or unplanned exercise.

Symptoms: These include pale skin, sweating, drooling from the mouth, shaking, palpitations, hunger, suboptimal intellectual function, confusion and inappropriate behaviour, seizures (uncommon), or coma.

Management: If the patient is conscious and cooperative:

- Stop dental treatment
- Place the patient in a semi-reclining position (supine if the patient loses consciousness)
- Monitor vital signs
- Administer oral sugar (food or drink) followed by longer acting carbohydrates (a sandwich, for example)
- If the patient is unconscious, DO NOT attempt to use the oral route. Administer 50% dextrose (intravenous) or a glucagon (intramuscular) injection.

Call for Medical Assistance

Note: Diabetic acidosis may lead to diabetic coma due to prolonged hypoglycaemia. This occurs slowly.

Chest Pain

1. Angina

Definition: Pain caused by temporary myocardial ischaemia resulting from a demand for more blood flow. In dental patients, this is usually precipitated by stress and anxiety.

Cause: A thrombus is the most common cause of myocardial ischaemia which may result in angina.

Symptoms and signs: Crushing pain in the chest which may persist for 3-5 minutes. Pain radiates to the left arm, neck or jaw. Vital signs are generally satisfactory.

Management: IF ANGINA OCCURS:

- Stop dental treatment
- Tilt the chair upright or semi-reclining
- Administer glyceryl trinitrate spray, a 400 microgram metered dose sublingually, and repeat the dose once in 5 minutes (maximum twice over 10 minutes).
- Monitor vital signs and administer oxygen

- Reassure the patient: do not proceed with dental treatment.
- Refer for medical evaluation.

If the pain persists for more than 10-15 minutes, treat as acute myocardial infarction and call for medical assistance.

Note: Avoid nitrates if the patient has used Viagra (sildenafil) in the previous 24 hours or Cialis (tedalafil) in the previous 5 days. Patients with a history of frequent angina attacks must be advised to bring their medication (glyceryl trinitrate spray or tablets) with them.

2. Acute Myocardial infarction

Definition: Death of a part of the cardiac muscle (infarction) due to the occlusion of coronary arteries constitutes acute myocardial infarction.

Cause: Common causes include occlusion of coronary arteries due to thrombus or embolus.

Symptoms and signs: Crushing chest pain radiating to the arm and jaw as in angina, but not relieved by the administration of nitrates. Other symptoms include palpitations, tachycardia, thready pulse, dyspnoea, cyanosis, and a feeling of impending doom.
Management:

If acute myocardial infarction is suspected:

- Tilt the chair position to upright or semi-reclining
- Monitor vital signs
- Administer oxygen
- Give aspirin (300 mg) to be chewed or dissolved before swallowing (only if the patient is not allergic to aspirin)
- Give glyceryl trinitrate spray as for angina
- Reassure patient and CALL FOR MEDICAL ASSISTANCE

IF PATIENT LOSES CONCIOUSNESS (cardiac or respiratory arrest)

Call for Medical Assistance

- Administer CPR and continue oxygen
- Use an automated external defibrillator (AED) if available
- Monitor the patient until assistance arrives.

Respiratory Distress

1. Asthma attack/Bronchospasm

Definition/description: Spasm and constriction of the bronchi causing severe respiratory distress.

Cause: Intrinsic (allergic) and extrinsic (respiratory irritants such as tobacco smoke) factors.

Symptoms: Laboured breathing, coughing and wheezing (in a severe attack, no wheezing). Cyanosis indicates a life threatening attack.

Management:

- Stop dental treatment
- Chair position should be upright
- Monitor the patient's pulse (<100 bpm in mild attack, 100-120 bpm in moderate attack and >120 bpm in severe attack)
- Administer a bronchodilator (albuterol 4 puffs initially, to be followed by another 4 puffs 4 minutes later). Assess the patient. If recovered fully, discharge from care.

IF THE ATTACK IS MODERATE OR SEVERE

- Administer bronchodilator as above
- CPR if necessary
- Administer oxygen
- Call for medical assistance - hospital admission is required for a severe attack.

2. Hyperventilation

Definition/description: Characterised by over-breathing due to anxiety or a panic attack, resulting in a lowering of the carbon dioxide level in the arterial blood.

Cause: Anxiety or panic attack.

Symptoms and signs: light-headedness, shortness of breath, a feeling of panic, blurred vision, tingling of the fingers, toes and lips, rapid breathing, rapid pulse, altered consciousness, and carpopedal spasm of the fingers and hands.

Management:

- Chair position should be upright
- Encourage patient to slow their breathing
- Have the patient rebreathe expired air by cupping their hands close to the mouth and nose

DO NOT ADMINISTER OXYGEN

- Monitor vital signs
- If symptoms persist for more than 5 to10 minutes or carpopedal spasm is extensive

CALL FOR MEDICAL ASSISTANCE

3. Airway obstruction

Definition: An accidental inhalation of a foreign body lodged in larynx and pharynx.

Symptoms and signs of obstruction: Choking, gagging, violent expiratory effort, substernal notch retraction, cyanosis, laboured breathing, rapid pulse (initially) and cardiac arrest.

Management

- Stop treatment
- Chair position should be upright. If unconscious, supine.

IF A PARTIAL OBSTRUCTION: remain with the patient to reassure and encourage them to cough up the foreign body.

IF A TOTAL OBSTRUCTION, CALL FOR MEDICAL ASSISATANCE

- Turn the patient on their side
- Attempt to clear and open the airway by manually removing the object
- Check for signs of breathing. If there are no signs of breathing;
  - Give 5 back blows between the shoulder blades using the heel of the hand. Check for the effectiveness of each blow.
  - Give 5 chest thrusts (identical to cardiac compression but harder). Check for effectiveness.
- If total obstruction continues, perform cricothyroidotomy:
  - Extend the neck
  - Feel the cricothyroid ligament
  - Incise through the skin and the ligament or insert a large-bore needle
  - Maintain airway until medical help arrives.

ALLERGIC REACTIONS

Definition/description: Hypersensitive responses to allergens.
Cause: Common allergies include antibiotic hypersensitivity, latex allergy, food allergy and rarely, allergic reaction to a local anaesthetic agent.
Types of allergic reactions include urticaria, angioedema and anaphylaxis.

Urticaria

Definition: Transient raised erythematous and oedematous weals of different shapes and sizes. Eruptions are common on the neck, face, hands and arms. Itching (pruritus) is present.
Management:

- Chair position: upright or semi-reclining
- Administer Benadryl (diphenhydramine hydrochloride) 25-50 mg orally. If the reaction is severe involving the lips and eyelids, administer Benadryl (25-50 mg) by

IV or IM injection. Subcutaneous or intramuscular injection of epinephrine (0.3-0.5 ml) 1:1000 solution is recommended if the reaction is very severe (true anaphylaxis).

CALL FOR MEDICAL ASSISTANCE

Anaphylactic shock

Definition: Anaphylactic shock is a severe IgE mediated allergic response due to immediate hypersensitivity reaction.

Symptoms: Progressive respiratory and circulatory failure, itching of the nose and hands, a flushed face, a feeling of substernal pressure, laboured breathing, stridor, coughing, sudden hypotension, cyanosis, loss of consciousness, and incontinence. Sometimes, there may also be angioedema, swelling of the tongue and oropharynx. Symptoms occur within minutes of parenteral or mucosal exposure to an allergen (a drug, for example), or 30 minutes to several hours after drug ingestion.

Management:

- Stop dental treatment
- Chair position: supine
- Administer oxygen by face mask (6-8 L/minute)
- Inject adrenaline 1:1000 intramuscularly 0.50 ml (or through an auto injector into the anterolateral thigh or tongue)
- Monitor vital signs
- For bronchospasm, use albuterol 2-4 puffs initially, repeating twice at 4 minute intervals
- Administer hydrocortisone 2-6 mg/kg intramuscularly
- For urticaria, give Benadryl 25-50 mg by the oral or IM route.

CALL FOR MEDICAL ASSISTANCE. HOSPITALISATION IS NECESSARY

Angioedema

Definition: Angioedema is characterised by acute oedema of the subcutaneous tissues.

Symptoms: Non-itchy lesions on the face, periorbital region, lips, tongue, glottis, dorsa of the feet and hands, and genitalia. Swelling of the lips, tongue, and eyelids are common. Involvement of the larynx can cause airway obstruction.

Management:

- Chair position: upright or semi-reclining
- Administer Benadryl (diphenhydramine hydrochloride) 25-50 mg orally. If the reaction is severe involving lips and eyelids, administer Benadryl (25-50 mg) via IV or IM injection. Subcutaneous or intramuscular injection of epinephrine (0.3-0.5 ml) 1:1000 solution is recommended if the reaction is very severe (true anaphylaxis).

CALL FOR MEDICAL ASSISTANCE

Neurological Emergencies

Seizures

Definition: Seizures are an intermittent disorder of the nervous system.

Cause: Epilepsy, hypoglycaemia, hypoxia following syncope, and local anaesthetic overdose.

Symptoms and signs: Pre-seizure aura, sudden spasm of muscles, jerky movements of the head, arms and legs, noisy breathing, salivation and loss of consciousness. In status epilepticus, recurrent seizures occur without recovery of consciousness between attacks.

Management:

- Stop treatment
- Chair position: supine
- Loosen clothes
- Protect the patient from falling
- Relocate all instruments
- Avoid restraining the patient
- Check conscious status
- If there is vomitus, remove it using high volume suction
- If the patient recovers completely, keep them under observation for at least 30 minutes. They must not be allowed to drive.

IF RECURRENT SEIZURES OCCUR WITHOUT RECOVERY OF CONCIOUSNESS BETWEEN ATTACKS, SEEK MEDICAL ASSISTANCE. THE PATIENT NEEDS TO BE HOSPITALISED.

Stroke (cerebrovascular attack) and Transient Ischaemic Attack (TIA)

Symptoms: Muscle weakness, loss of consciousness, confusion, headache, dizziness, dysphagia, paralysis (difficulty in moving one side of the body).

Cause: Cerebral thrombosis or haemorrhage.

Management:

Stroke:

If the patient is conscious:

- Chair position: semi-reclining
- Monitor vital signs
- Provide oxygen
- Seek medical assistance

If the patient is unconscious:

- Chair position: supine
- Record vital signs

- Provide basic life support
- Provide oxygen
- SEEK MEDICAL ASSISTANCE AND TRANSFER THE PATIENT TO HOSPITAL. DO NOT give any anti-platelet medication (aspirin, for example).

Panic Attack

Cause: Acute anxiety.

Symptoms: Anxiety, chest pain, hyperventilation, nausea, dizziness, numbness in the extremities and restlessness.

Management:

- Chair position: for patient comfort
- Monitor vital signs
- Call for medical assistance if the patient's vital signs are unstable.

Adrenal Crisis

Definition: Adrenal crisis can occur 6 to 12 hours after surgical stress in patients who are taking corticosteroids and have not had them increased before surgery. This may happen at home.

Symptoms: Hypotension is progressive. Patient may feel faint and confused, and collapse. This may even result in death at home while asleep.

Management:

- Always organise appointments in the morning
- Advise the patient to have their steroid dose increased (doubled) before surgery
- If the patient is unconscious and help is not immediately available, administer hydrocortisone 200 mg intramuscularly or intravenously.

CARDIOPULMONARY RESUSCITATION (CPR) AND BASIC LIFE SUPPORT (BLS)

Cardiopulmonary resuscitation - (CPR)

These guidelines are applicable to adults, children and infants. [8, 9]

What is CPR?

Cardiopulmonary resuscitation (CPR) is a technique of chest compressions combined with rescue breathing.

What is the purpose of CPR?

The purpose of cardiopulmonary resuscitation is to temporarily maintain a circulation sufficient to preserve brain function until specialised treatment is available.

When to start CPR?

Rescuers must start CPR if the victim is unresponsive and not breathing normally. Even if the victim takes occasional gasps, rescuers should start CPR.

CPR should commence with chest compressions. Interruptions to chest compressions must be minimised.

Compression ventilation ratio

- Current consensus is that a universal compression-ventilation ratio of 30:2 (30 compressions followed by 2 ventilations) is recommended for all ages regardless of the numbers of rescuers present.
- Compressions must be paused to allow for ventilations.

Steps of Resuscitation

Initial steps of resuscitation are: DRS ABCD

- Check for **D**anger (hazards/risks/safety)
- Check for **R**esponse (if unresponsive)
- **S**end for help
- Open the **A**irway (head tilt-chin lift)
- Check **B**reathing (if not breathing/abnormal breathing)
- Give 30 chest **C**ompressions (almost two compressions/second) followed by 2 breaths
- Attach an AED (**A**utomated **E**xternal **D**efibrillator) if available and follow the prompts.

When providing 30 compressions (at approximately 100/min) and giving 2 breaths (each given over one second per inspiration), this should result in the delivery of five cycles in approximately two minutes.

Chest compressions only

If rescuers are unwilling or unable to do rescue breathing they should do chest compressions only. If only chest compressions are given, they should be continuous at a rate of approximately 100/min.

Multiple rescuers

When more than one rescuer is available ensure:

- That an ambulance has been called;
- All available equipment has been obtained (e.g., AED).

Duration of CPR

Rescuers should minimise interruptions of chest compressions and CPR should not be interrupted to check for response or breathing. Interruption of chest compressions is associated with lower survival rates.

The rescuer should continue cardiopulmonary resuscitation until:

- The victim responds or begins breathing normally
- It is impossible to continue (e.g., exhaustion)
- A health care professional arrives and takes over CPR

- A health care professional directs that CPR be ceased

If CPR is required in a heavily pregnant woman it should be performed with the patient lying in the left lateral tilt position of up to 30^{o}. To achieve this, place a wedge under the patient's right hip. Alternatively, if a second rescuer is available, the uterus may be manually moved to the left side. The intention with either method is to shift the uterus off the major abdominal vessels.

Risks

The risk of disease transmission during training and actual CPR performance is very low. A systematic review found no reports of transmission of hepatitis B, hepatitis C, human deficiency virus (HIV) or cytomegalovirus during either training or actual CPR when high-risk activities, such as intravenous cannulation, were not performed. If available, the use of a barrier device during rescue breathing is reasonable. After resuscitation, all victims should be reassessed and re-evaluated for resuscitation-related injuries.

ANY ATTEMPT AT RESUSCITATION IS BETTER THAN NO ATTEMPT

Sources:

Meiller, T. F. et al. (2006). *Dental office medical emergencies*: *a manual of office response protocols* (2nd edition). Hudson, Ohio: Lexi-Comp.

Australian Resuscitation Council. *Guideline 8: cardiopulmonary resuscitation* [online]. 2010 [2014 August 7]. Available from: http://www.resus.org.au/policy/guidelines /section_8/ cardiopulmonary_resuscitation.htm.

Appendices

Appendix 1.

Table 1. Vaccination schedule

Hepatitis B: Birth, 2 mos, 4 mos, 6-12 mos.
Diphtheria, pertussis and tetanus (DPT): 2, 4 and 6 mos, 4 yrs and 15-17 yrs.
Haemophylus Influenza Type B: 2, 4, 6 and 12 mos.
Pneumococcal: 2, 4, 6 mos and 65 yrs.
Measles, mumps and rubella (MMR): 12 mos, 4 yrs.
Varicella: 18 mos.
Meningococcus: 12 mos.
Human papilloma virus (HPV): 10-13 yrs.
Influenza: 65 yrs (and then annually).

Table 2. Some useful reference values

- **Red Blood Cells:**

Haemoglobin: 135-175 g/L (M) and 120-160 g/L (F)
Glycosylated Haemoglobin (HbAlc): at age 50 yrs 3.6-5.0% of Hb (M) and 3.9-5.3% of Hb (F)
Haematocrit (Hct): 0.4-0.53 (M) and 0.36-0.48 (F)
Mean Cell Volume (MCV): 76-100 fL (M) and 78-101 fL (F)
Red Blood Cell Distribution Width (RDW): 11.5-14.5%
Mean Cell Haemoglobin (MCH): 0.39-0.54 fmol/cell
Mean Corpuscular Haemoglobin Concentration (MCHC): 4.8-5.4 mmol/L
Red Blood Cells (RBC): 4.2-5.7 mln/mm^3 (M) and 3.5-5.1 mln/mm^3 (F)
Reticulocytes 0.5-1.5 % of RBC (Adult)

- **White Blood Cells**

White Blood Cell Count (WBC): 3.5-9.0 $x10^9$/L
Neutrophil Granulocytes (PMNs, Polys, Grans or Segs): 1.3-5.4 $x10^9$/L
Lymphocytes. 0.7-3.5 $x10^9$/L
Monocytes: 0.1-0.8 $x10^9$/L
Mononuclear Leukocytes (lymphocytes + monocytes): 1.5-5 $x10^9$/L
CD4+cells: 0.4-1.5 $x10^9$/L
Eosinophil Granulocytes 0.0-0.44 $x10^9$/L
Basophil Granulocytes: 40-100 $x10^9$/L

Table 2. (Continued)

- **Coagulation**

Platelet Count (Plt): 150-450 $x10^9$/L
Prothrombin Time (PT) 10-13 s (seconds)
International Normalised Ratio (INR): 0.9-1.2
Activated Partial Thromboplastin Time (APTT): 18-28 s (seconds)
Thrombin Clotting Time (TCT): 11-18 s (seconds)
Fibrinogen: 1.7-3.6 g/L
Bleeding Time (BT): 2-9 mts (minutes)
Viscosity: 1.5-1.72 cP

- **Immunology**

Erythrocyte Sedimentation Rate (ESR): 0 – Age divided by 2 for males and age plus 10 divided by 2 for females (mm/hr)
C-Reactive Protein (CRP): n/a-5 mg/L Alpha1-antitripsin (AAT): 89-170 mg/dL
IgA: 70-360 mg/dL
IgD: 0.5-3.0 mg/dL
IgE: 0.01-0.04 mg/dL
IgG: 800-1800 mg/dL
IgM: 54-220 mg/dL
Anti-SSA(Ro): >25 U/mL. Positive
Anti-SSB (La): >4 U/mL. Positive
Anti-ds-DNA: >60 U/mL. Positive
Anti ss-DNA 10 U/mL. Positive
Cytoplasmic Anti-neutrophil Cytoplasmic Antibodies (c-ANCA): >30 U/mL. Positive
Perinuclear Anti-neutrophil Cytoplasmic Antibodies (p-ANCA): >5 U/mL. Positive
Anti-mitochondrial Antibodies (AMA): >10 U/mL. Positive
Rheumatoid Factor (RF): 30 U/mL. Positive
AntistreptolysinO titre (ASOT) in preschoolers: >100 U/mL. Positive
ASOT in adults: >125 U/mL. Positive

- **Other Enzymes and proteins**

Lactate dehydrogenase (LDH): 50-150 U/L
Amylase 25-110 U/L
Lipase 7-60 U/L
Angiotensin Converting Enzyme (ACE): 23-57 U/L
Acid Phosphatase: 3.0 ng/mL
Urea: 3.0-7.0 mmol/L
Uric acid: 2.1-8.5 mg/dL (M) and 2.0-7.0 mg/dL (F)
Creatinine: 60-118 mg/dL (M) and 50-90 mg/dL (F)
BUN/Creatinine Ratio: 5-35
Plasma Glucose (fasting): 3.8-6.0 mmol/L
Full Blood Glucose (fasting): 3.3-5.6 mmol/L

- **Iron and trace metals**

Sodium (Na) 135-145 mmol/L
POTAQSSIUM (k): 3.5-5.0 mmol/L
Chloride (Cl): 95-105 mmol/L
Ionized calcium (Ca): 1.03-1.23 mmol/L
Total Ca: 2.1-2.5 mmol/L
Total serum iron (TSI): 65-176 µg/dL (M) and 26-170 µg/dL (F)

Total Iron Binding Capacity (TIBC): 240-450 µg/dL
Transferrin: 190-326 mg/dL
Ferritin: 12-300 ng/mL (M) and 12-150 ng/mL (F)
Ammonia: 10-35 µmol/L
Copper: 70-150 µg/dL
Phosphate: 0.8-1.5 mmol/L
Inorganic Phosphorus (serum): 1.0-1.5 mmol/L
Magnesium: 1.5-2.0mg/dL

- **Acid-Base and Blood gases**

pH Arterial: 7.34-7.44 and Venous: 7.31-7.41
Oxygen partial pressure (pO_2):75-100 mmHg (or Torr)
Oxygen saturation Arterial: 94-100% and Venous: Approx 75%
Carbon Dioxide Partial Pressure (PCO_2) Arterial: P_aCO_2: 33-44 mmHg and Venous: 41-51 mmHg
Bicarbonate (HCO_3) Arterial and Venous: 18-23 mmol/L

- **Liver Function Tests (LFTs)**

Total Protein: 60-78 g/L
Albumin: 35-48
Globulins: 23-35 g/L
Total Bilirubin: 0.1-1.0 mg/dL
Direct/Conjugated Bilirubin: 0.0-0.3 mg/dL
Alanine Transaminase (ALT): 5-20 IU/L (ALT is also called Serum Glutamic Pyruvic Transaminase (SGPT))
Aspartate Transaminase (AST): 6-34 IU/L (F) and 8-40IU/L (M) (also called Serum Glutamic Oxaloacetic Transaminase (SGOT))
Alkaline Phosphatase (ALP): 53-128 U/L (M) and 42-98 U/L (F)
Gumma Glutamyl Transferase (GGT): 0.92 µkat/L (M) and 0.63 µkat/L (F)

- **Cardiac Tests**

Creatinine Kinase (CK): 24-174 U/L (M) and 24-140U/L (F)
Myoglobin: 17-106 µg/L (M) and 1-66µg/L (F)
Troponin-I: > 0.2 µ/L and Troponin-T: > 0.2 µg/L (suggestive of cardiac muscle damage)

- **Lipids**

Triglycerides: 0.6-1.7 mmol/L (Therapeutic target: 1.1 mmol/L)
Total Cholesterol: 3.0-5.0 mmol/L (Therapeutic target: 3.9 mmol/L)
HDL Cholesterol: 0.9- 2.0 mmol/L (M) and 1-2.2 mmol/L (F) (Therapeutic target: 1-1.6 mmol/L)
LDL Cholesterol: 2.0-3.0 mmol/L
LDL/HDL quotient: n/a-5.

- **Thyroid Function Tests**

Thyroid Stimulating Hormone (TSH): 0.3-4.0 mIU/L
Free Thyroxin (FT4): 0.7-1.4 ng/dL
Total Thyroxine: 4-11 µg/d/dL
Free Triiodothyroxine (FT3): 0.2-0.5 ng/dL
Total triiodothyronin: 60-1755 ng/dL
Thyroxin-Binding Globulin (TBG): 12-30 mg/L
Thyroglobulin (Tg): 1.5-205 µg/dL

Table 2. (Continued)

- **Other hormones**

Adrenocorticotropic Hormone (ACTH): 4.4-18 pmol/L
Cortisol: 5-25 µg/dL (at 9.00am) and 2.9-13 µg/dL (at midnight)
Growth Hormone (Fasting): 0-5 ng/mL
Prolactin: 2.7-13.0 µg/dL (M) and 3.4-16.4 µg/dL (F)
Parathyroid hormone (PTH): 1.1-6.9 pmol/L
25-hydroxycholecalciferol (a Vitamin D): 20-95 nmol/L. (Therapeutic target range: 85-120 nmol/L)
Aldosterone: 19-34 ng/dL

- **Vitamins**

Vitamin A: 30-65 µg/dL
Vitamin B_9 (Folic acid, Folate): 450-1400 nmol/L (RBCs) and 6.8-36 nmol/dL (serum)
Vitamin B_{12} (Cobalamin): 130-700 ng/L
Vitamin C (Ascorbic acid): 0.4-1.5 mg/dL
25-hydroxycholecalciferol (a Vitamin D): 20-95 nmol/L. (Therapeutic target range: 85-120 nmol/L)
Vitamin E: 1.2 mg/dL

- **Tumour markers**

Carcinoembryonic antigen (CEA): 3.4-5 µg/l
Prostate specific antigen (PSA): <2.5 µg/L

Recommended Further Reading

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