DIPLOMA IN REGISTERED NURSING

eLearning Training Program

Course Title: Medicine and Medical Nursing I

Acknowledgement

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COURSE OVERVIEW

You are welcome to our course on medicine and medical nursing. As you may know nurses have an important role in serving people's lives and as such they need to be knowledgeable about conditions that affect the human beings. This course intends to assist you with information which is essential in nursing patients with medical conditions. Knowing the medical conditions will assist you to carry out your nursing care to your patients effectively and efficiently as nursing care contributes much in the recovery of the patients.

Medicine and medical nursing course is offered using units. The splitting of the course in different units is to enable you understand each aspect of the course step by step. This course is associated with some of the courses that you studied in your foundation block such as Anatomy and Physiology, microbiology and nutrition. The course starts by explaining the principles of medicine and medical nursing, the nature and causes of the diseases and health assessment. It further goes on to describe conditions according to the systems of the body. Each system then starts with reviewing the anatomy and physiology which is an important aspect of understanding the condition as this states the parts involved and the functions which are disturbed, the common investigations done on those conditions, the definition of the conditions and so on.

Course Aim:

To equip students with knowledge and skills in the management of common medical conditions in order to provide comprehensive nursing care to clients and their families in hospitals, health centres and community settings.

Course Objectives

At the end of the course, you should be able to:-

1. Describe the common medical conditions affecting various systems of the body.
1. Explain the management, prevention and control of common infectious diseases in the hospital, health centre and community settings.
2. Interpret medical diagnostic investigations and prescribe appropriate interventions.
3. Apply the appropriate nursing model in the management of patients.

Course Content

This course is divided into 3 sections which are:

Medicine and medical nursing 1- MMN 019

You will cover this section in the first year. The section comprises of 4 units.

Unit 1: Introduction to medicine and medical nursing

This unit reviews principles of medicine and medical nursing, nature and causes of diseases and health assessment.

Unit 2: Digestive system

This unit reviews the anatomy and physiology of the digestive system and the role of the nurse in investigations and procedures done in digestive system disorders. The unit also describes management of patient with oral and esophageal disorders, stomach and duodenal disorders, intestinal disorders, hepatic disorders, biliary disorders and worm infestations.

Unit 3: The Respiratory System

In this unit you will review the anatomy and physiology of the respiratory system and outline the roles of the nurse in investigations and procedures which are done in respiratory disorders. You will also discuss the management of patients with upper and lower respiratory disorders.

Unit 3: Cardiovascular System

This unit reviews the anatomy and physiology of the cardiovascular system and outlines the role of a nurse in investigations and procedures done in the cardiovascular disorders. It goes on to discuss the management of the patient with blood disorders, heart disorders, hypertension, peripheral vascular disorders, malaria and filariasis.

Assessments

Your work in this course will be assessed by writing assignments and tests during the course and final examination at the end of the year. You will also be assessed in practical to see whether you have acquired the skill in the procedures needed. The breakdown of the assessments is as follows;

Continuous assessment - 40%

- Tests - 20%
- Assignments - 20%

Final examination - 60%

- Theory - 40%
- Practical - 20%

Learning Tips

This course will probably take you a minimum of 265 hours to cover the theory and 495hours practical. These hours should be spent on studying the course and readings, doing the activities and self-help questions and completing the assessment tasks. Note that the units are not of the same length; as such you must plan and pace your work to give yourself time to complete all of them.

Activities, Self-Help Questions And Case Studies

As you study the course, you will find activities, self-help questions and case studies. These are planned distance education programme and are intended to help you make your learning more active and effective as you progress and apply what you read. The activities will help you to employ new ideas and check your own understanding. It is therefore important to take time to complete them in the order they occur in this course. Ensure that you write full answers to the activities or take note of the discussions.

Readings

You will find a list of further readings at the end of this course. This includes books that are referred to in the course. The references will help you in case you wish to explore further. You are encouraged to read as widely as possible during and after the course, but you are not expected to read all the books on this list.

Unit 1: Introduction To Medicine And Medical Nursing

1.1 Unit Introduction

Welcome to our first unit in the medicine and medical nursing course. This unit introduces you to medicine and medical nursing. The introduction includes principles of medicine and medical nursing, nature and causes of disease, health assessment which will cover history taking and physical examination. Physical examination will include techniques which are; inspection, palpation, auscultation and percussion. Before you get into the actual content of this unit you will

find some key terms used in medicine and medical nursing which are defined. This is to make your understanding easier. The content in this unit will also help you to understand the information which is in the other units. By the end of this unit you should be able to apply the principles of medicine and medical nursing in the health assessment of clients with medical conditions. Let us start by reviewing our objectives for this unit.

1.2 Unit Objectives

By the end of this unit you should be able to:

1. Explain the principles of medicine and medical nursing.
2. Describe nature and causes of disease.
3. Explain the process of conducting health assessment

1.3 Definition of Terms Used in Medicine and Medical Nursing

There are quite a number of terms which are used in medicine and medical nursing. It is important for you to learn these terms so that you can understand the content of this course. We shall therefore start by defining these key terms.

Medicine

Medicine is the study of diseases. It looks at how diseases present their management and prevention. It is also defined as the art and science of the diagnosis and treatment of disease and the maintenance of health.

Medical Nursing

Medical nursing is a branch of nursing which deals with the care of patients/clients with medical disorders.

It can also be defined as an art and science applied to the care of patients with medical conditions. (It is the scientific approach to the care of patients with medical conditions).

Etiology

This term refers to the cause of disease.

Pathology

Pathology is a branch of medicine that deals with the essential nature of disease, especially of the structural and functional changes in tissues and organs of the body which cause or are caused by disease.

Pathophysiology

Pathophysiology refers to an alteration in functioning of the body as a result of disease. It clearly outlines how signs and symptoms come about.

Disease

This is a pathological process having a characteristic set of signs and symptoms.

Prognosis

This is a forecast of the probable course and outcome of an attack of disease and the prospects of recovery OR

It can also mean the art of telling the course, duration and termination of any disease.

Investigations

This simply refers to the search into the problem so that one comes up with the diagnosis.

Symptom

A symptom is what the patient is complaining of.or

A physical or mental feature which is regarded as indicating a condition of disease, particularly such a feature that is apparent to the patient.

Sign

Sign is a physical observations made on the patient on examination.

Complications

It is a lesion or symptom resulting from original disease.

Syndrome

Syndrome refers to a set of symptoms and signs which occur together and constitute the manifestation of some special condition.

Diagnosis

Diagnosis is the recognition of a particular ailment from symptoms, physical signs and any test which may have been performed.

Prophylaxis

Prophylaxis refers to prevention of disease and treatment one gets in order to avoid the disease.

Morbidity

Morbidity refers to a diseased state, disability, or poor health due to any cause. The term may be used to refer to the existence of any form of disease, or to the degree that the health condition affects the patient.

1.4 Principles of Medicine And Medical Nursing

What is a principle? Before you read on, do the following activity.

Activity 1.1
Write down the meaning of the word "principle" in your notebook

Well done! Now compare your definition with the one in the following discussion.

A principle is an established rule of action to follow in a given situation. It acts as a guide. For you to learn this course you need to be guided. The following are the principles of medicine and medical nursing:

- Autonomy
- Confidentiality
- Fidelity
- Justice
- Respect for persons
- Informed consent
- Cultural concerns
- Beneficence
- Sanctity
- Informed consent
- Sexual relationships

Let us look at each principle in detail starting with autonomy.

Autonomy – each person has individual rights, privacy and choice. The nurse must therefore respect the patient's autonomy which includes the patient's right to refuse therapy. The nurse must also actively seek to empower the patient with adequate information (Davison medicine, 2006)

Confidentiality - Confidentiality in relation to the appropriate management of patient-specific information is important in generating and maintaining trust in the nurse-patient relationship.This principle relates to the concept of privacy. Information which is obtained from an individual should not be disclosed to another without patients consent.

Fidelity – the duty to become faithful to one's promise.

Justice – Justice relates to the distribution of health services and allocation of resources. Justice is also equated with 'being fair' and 'even-handed',all patients should be treated alike.

Respect for persons – Have respect for clients regardless of gender or status in society.

Sanctity of life - life should take precedence in all practice.

Veracity – the obligation to tell the truth and not lie or deceive others.

Beneficence-This is the principle of doing good, or acting in another person's best interests. The term beneficence refers to actions that promote the well-being of others. In the medical context, this means taking actions that serve the best interests of patients.

Informed Consent-This term describes the participation of a patient in decisions about their health care. In ethics usually refers to the idea that a person must be fully informed about and understand the potential benefits and risks of their choice of treatment **Cultural concerns** – Cultural differences can create difficult medical ethics problems. Some cultures have spiritual or magical theories about the origins of disease, and reconciling these beliefs with the tenets of Western medicine can be difficult.

Sexual Relationships- Sexual relationships between health care providers (doctors/nurses) and patients can create ethical conflicts since sexual consent may conflict with the fiduciary (involving trust) responsibility of the physician. Doctors/nurses who enter into sexual relationships with patients face the threats of deregistration and prosecution.

In-text question 1.1

Match the following principles with their meaning in the other column

1. Fidelity	A. Equal treatment to persons
2. Autonomy	B. Duty to become faithful to one's promise
3. Confidentiality	C. Person's rights, privacy, choice
4. Justice	D. Concept of privacy

I hope you have now mastered the principles of medicine and medical nursing.

1.4 Nature and Causes of Disease

Activity 1.2

Using your own words, write down the meaning of the word "Disease" in your notebook

Good work! Now compare your definition with the one we shall discuss below.

The word **disease** refers to any condition that impairs normal function. It can also be defined as a departure from health due to an interruption or disorder of function. There are varying degrees of

departure from normal. They may be severe enough to cause incapacity of the individual, or may be less serious allowing an individual to remain active but without a sense of wellbeing. Diseases can occur naturally or can be acquired artificially. They may be caused by factors from outside the body such as cholera, which is a waterborne disease, or can originate from factors within the body, for example hypertension.

A person with a disease may present with certain signs and symptoms or may not due to a number of normal cells which maintain adequate degree of normal functioning or can be due to compensatory mechanism such as hypertrophy (an increase in the size of a tissue or structure caused by an increase in the size of cells). The effect of a disturbance in the function of one part of the body is likely be reflected in the functioning of other or all parts because of the dependence of each system on the others for oxygen, nutrients, elimination of wastes and other essentials. **Causes of Diseases**

What do you think are the causes of diseases?
Write your answers in your note book and compare your answers with the notes below.

Diseases can be caused by different factors of which some can be from within the body (internal) and some can be from outside the body (external). The following are recognized causes of diseases:

- Hereditary factors
- Developmental defects
- Biological agents
- Physical agents
- Chemicals
- Deficiencies and excesses
- Lack of normal oxygen supply to any tissue seriously impairs its function
- Emotions
- Tissue responses
- Unknown/Idiopathic

Let us look at each cause of disease in detail starting with hereditary factors.

- **Hereditary:** these are diseases passed from parent to child. Hereditary diseases may be transmitted from one generation to another by a genetic or chromosomal disorder in one or both gametes for example sickle cell disease.
- **Developmental Defects:** Failure or abnormality in the developmental process during the embryonic or fatal stage leads to developmental defects. The cause in most cases is unknown.

Developmental defects are seen in some infants born to mothers who have had a viral infection during the first trimester (first 3 months) of pregnancy. Toxic chemicals taken during pregnancy may disturb normal fatal development. Radiation is also thought to cause developmental defects examples spinal bifida and cleft palate

- **Biological agents:** one of the commonest causes of disease is the invasion of the body by bacteria, viruses, fungi or parasites. These harm and destroy the tissues by their direct action on the cells or by the toxins they produce. The disease caused by biological agent is referred to as an infection.
- **Physical Agents:** tissues may suffer injury or destruction as a result of external forces in the environment. These include pressure, blows, falls, lacerations and the entry of foreign bodies such as bullets.
- Cells may be destroyed when subjected to extreme heat or cold. Exposure to excessive sun rays and to radiation from x-rays or radioactive material may alter cell structure and activity or may actually cause destruction of the cells.
- **Chemicals:** when some chemicals are introduced into the body, they have a harmful effect on tissue cells. The chemical may disrupt normal cellular chemical reactions either by forming incompatible compounds or by interfering with normal enzymatic action within the cells.
- **Deficiencies and Excesses:** an inadequate supply of materials essential to normal tissue structure and activity may cause a variety of diseases. The deficiency may be due to an insufficient intake of nutritional substances or a specific element, lack of absorption form the intestine, or interference in the delivery of the essential substances to the cells by the circulatory system.
- **Lack of normal oxygen supply to any tissue seriously impairs its function**. If the supply is completely cut off, the cells quickly die. The deficiency may be local or general; local hypoxia may be due to a blockage of the vessels supplying the affected area. General oxygen deprivation may be due to respiratory insufficiency or a disturbance in the oxygen-carrying or delivery mechanisms.
- An excess of nutrients may also create problems, such as increased demands on body function and the storage of excess fat. Examples of diseases that occur as a result of excess nutrients include hypertension, certain cardiovascular diseases, and diabetes in obese persons.
- ***Emotions:*** psychological reactions to stressful situations may influence a person's autonomic nervous system and alter its control of visceral activities. Changes in autonomic innervations may increase or decrease the function of certain structures; this may have marked effects on total body functioning.
- ***Tissue responses*****:** Illness may be caused by the responses or reaction of tissues to an injury or irritation. Examples of this are inflammation and allergic reaction.
- ***Unknown/Idiopathic*****-** The cause of some diseases is unknown for example, causes of cancer, rheumatoid arthritis, leukemia and psychosis. In some diseases, predisposing and

perpetuating (precipitating) factors have been recognized even though the primary causative factor has not been identified. Such information contributes to preventive care. When the cause is unknown, care and therapy are based principally on the signs and symptoms.

In-text question 1.2

Do you still remember the causes of diseases which we have discussed in this lecture?
Write down in your note book any 5 causes of diseases

1.5 Health assessment

Health assessment refers to the process of obtaining data or information from the patient. The information to be collected has to be complete. Under this topic we will look at:

- History taking
- Physical examination

History Taking:

This is also referred to as 'clinical interview'. This is the major component of health assessment, the first thing done to find out why the patient comes to the health facility. It is a planned discussion that is aimed at establishing the health needs and health problems of the client. It is normally conducted before physical assessment.

What information would you need to collect under history taking?
Write your answers in the note book.

Well done

During history taking you should collect the following information:

- Demographic data e.g. age and sex; address; marital status
- Presenting complaints
- History of presenting illness; history of previous illnesses
- Treatment history
- Family history
- Social and occupational history

Let us now look at the information that you should collect under under history taking in detail..

Demographic data/biographical data

Under this heading you will need to collect the following information and record

- Name of client.
- Age
- Sex.
- Address. (residential)
- Marital status
- Next of kin.
- Religion.
- Denomination
- Nationality
- Tribe.
- Occupation

The Presenting Complaint

The presenting complaint is simply the problem, which made the patient seek medical help. Try to define the main complaint and its duration. The timing and duration of symptomatic events is especially important since the chronology (order) of the illness will provide valuable clues to the pathological process underlying it. Some patients find it difficult to remember the exact duration of their illness, especially the elderly. This is normal. Some will use events to signify the duration. Ask when the patient last felt perfectly well. This may help the patient to remember earlier symptoms, which he/she may have thought as unimportant.

The History of the Present Illness

Ask the patient to tell you the story of the illness from the beginning. Ideally, you should allow the patient to continue without interruption. If the patient is anxious or nervous, you will need to use tactful encouragement. For talkative patients, try to direct their account of events. Some patients use medical terminologies without knowing their meaning; encourage such patients to tell you what they actually feel to be wrong.

Friends/relatives may accompany the patient; always talk to the patient first. When the patient has given you an initial description of their symptoms, suggest that you would like to find out more about certain aspects. Try to clear up doubts about the time of onset and the duration of the main symptoms. Some symptoms come and go, then try to find out whether the relapses and remissions are related in any way to times, seasons or events in the patient's life. After the patient's story is clearly understood, examine each symptom in detail.

Symptom analysis

Pain is a symptom, which can be explored in clinical terms in order to direct physical examination, and then clinical investigation, toward description of the cause of the pain and its treatment. Other symptoms may have a physiological basis and thus can be considered more specifically. In symptom analysis, it is important to consider the course or shape of illness, reason for presentation, and finally review of systems

For example, to conduct an analysis of pain, you need to consider the following and ask your client these questions

- Onset: When did the pain start?
- Location: Where is the pain?
- Duration: For how long have you been feeling that pain? When did it start?
- Characteristics: How is the pain? Is it stabbing? Burning? Pricking? Is it localized or it originates from a certain body part and moves to other parts?
- Aggravating factor: What worsens the pain? Is it coughing? Breathing?
- Relieving factor: What lessens/reduces the pain?
- Treatment: What drugs or treatment are you receiving?

To help you remember the components of symptom analysis, you may use this mnemonic **OLDCART**. These letters stand for

O – Onset of illness

L – Location

D – Duration

C – Characteristics

A – Aggravating factors

R – Relieving factor

T – Treatment

S – Severity of the condition

The History of Previous Illness

Evaluation of the previous history will help you to get an insight about the health status of the patient.

The previous history should include all important illnesses, operations and injuries from infancy onwards. Ask questions about the nature of the illness to check whether the diagnosis seems likely. Inquire about treatment for the previous illnesses. Ask about previous admissions to hospital.

The Family History

It is important to evaluate the family history of your client. Evaluation of family history will help you to determine the health of the immediate family members. It will also help you to know whether your client is at risk of developing certain conditions which run in families.

Note the patient's position in the family and the age of the siblings/children, if any. Record the state of health, important illnesses and the cause of death of immediate relatives. Inquire about hereditary disorders in the family. Ask if there is any family member with similar symptoms.

The Social History

The patient's physical and emotional environment, including their surroundings both at home and work, and their habits and mental attitude to life and to work are essential components of the history that are important in assessing the effect of the illness on the patient and on their family. Ask your patient questions such as; what do you do during your spare time? Do you take alcohol? Smoke? Exercise?

Occupational History

Certain occupations may predispose to conditions such as prolonged exposure to radiation may lead to cancers. Ask your client whether s/he has been exposed to noxious substances at work, number of work hours, nature of work e.g. secretary, director, underground mine worker, street vendor, bar man, shop assistant or nursery nurse.

Menstrual History

Women should be asked about menstruation i.e. last normal menstrual period, regularity duration, amount of flow, dysmenorrhea, menstrual tension, history of taking oral contraception.

Obstetric History

Data on a woman's experience of childbirth, including abortions number of pregnancies, deliveries whether normal or had complications, health during pregnancy and whether those children are alive or they are dead.

I hope you now understand how to take the history of a patient or client. We are now going to discuss how to conduct a physical examination.

Physical examination

After taking the history of the patient, it is important to conduct a routine physical examination. This procedure may disturb the patient because of what is involved. For example, the patient is required to undress. So you will need to reassure and put the patient at ease. When conducting an examination, you should be gentle and avoid tiring and exposing the patient unnecessarily. **In acutely ill patients, it may be necessary to postpone a routine examination and to perform only the examination necessary for a provisional diagnosis and treatment**. The history and physical examinations are complementary.

A good physical examination requires a cooperative patient and a quiet, warm and well-lit room. Day light is better than artificial light, which may mask changes in skin colour, for example, the faint yellow tinge of slight jaundice. Attempt to make the patient to relax regardless of the circumstances. For a complete examination, the patient should be asked to undress, but should be covered with a bed sheet/blanket. The patient may be permitted to have underwear but remember to examine the buttocks and the genitalia as well.

A chaperone should be present when a male nurse is examining a female patient and during rectal and vaginal examinations, both to reassure the patient and to protect the nurse from subsequent accusations of improper conduct.

Four major techniques are used in performing the physical examination. These are:

- Inspection
- Palpation
- Percussion
- Auscultation

Let us look at each technique in further detail.

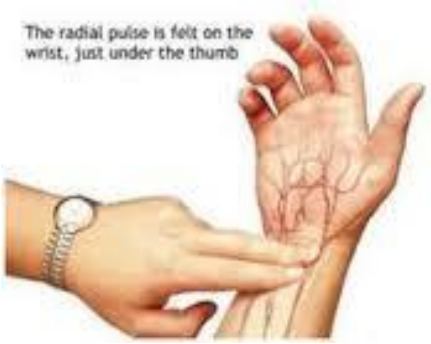
Inspection

Inspection is the visual examination of a part or region of the body to assess normal conditions or deviations from normal. Inspection is more than just looking. This technique is deliberate, systematic, and focused. You are required to compare what you can see with what are the known, generally visible characteristics of the body parts being inspected. Make sure adequate light is available and position and expose body parts in such a way that all surfaces can be viewed. Additional light is used to inspect cavities accurately.***Palpation***

Palpation is the examination of the body with touch. The use of light and deep palpation can yield information related to masses, pulsations, organ enlargement, tenderness or pain, swelling, muscular spasm or rigidity, elasticity, vibration of voice sounds, crepitus, moisture, and differences in texture. During your practical class, you will learn that different parts of the hand are more sensitive for specific assessments.

For example;

- The tips of the fingers are used to palpate lymph nodes and pulse rate
- The dorsa of hands and fingers are used to assess temperatures
- The palmar surface is best suited for feeling vibrations and abdomen for tenderness or any masses



FigureTable

Percussion

Percussion is an assessment technique involving the production of sound to obtain information about the underlying area. The percussion sound may be produced directly or indirectly. Direct percussion is performed by directly tapping the body with one or two fingers to elicit a sound. Indirect, or mediated, percussion is the more common technique. The middle finger (pleximeter) of the non-dominant hand is placed firmly against the body surface. The tip of the middle finger of the dominant hand (plexor) strikes the distal phalanx or the distal interphalangeal joint of the pleximeter finger. A relaxed wrist and rapid strike produce the best sounds. The sounds and the vibrations produced are evaluated relative to the underlying structures. Deviation from an expected sound may indicate a problem. For example, the usual percussion sound in the right lower quandrant of the abdomen is tympany. Dullness in this area may indicate a problem that should be investigated. (Specific percussion sounds of various body parts and regions are discussed in the appropriate assessment notes).

Ascultation

Auscultation is listening to sounds produced by the body to assess normal conditions and deviations from normal. Auscultation is usually indirect, using a stethoscope to clarify sounds by blocking out extraneous sounds. The bell of the stethoscope is more sensitive to low-pitched sounds. The diaphragm of the stethoscope is more sensitive to high-pitched sounds. Auscultation is particularly useful in evaluating sounds from the heart, lungs, abdomen, and vascular system.

Take Note:1.1
Physical assessment techniques are usually performed in the following sequence: inspection, palpation, percussion, and auscultation.
The only exception to this sequence is for the abdominal examination. In this situation, the sequence is inspection, auscultation, percussion, and palpation.

The following outline gives you a breakdown of what to assess in different parts of the body.

An Outline for Screening Physical Examination

General

- General appearance (does the patient look healthy, unwell or ill, well cared for or neglected?)
- Intelligence and educational level
- Mental state
- Expression and emotional state

- Build and posture
- Nutrition, obesity, oedema
- Skin colour, cyanosis, pallor, jaundice, pigmentation
- Body hair
- Deformities, swellings
- Temperature, pulse, respiration rate
- Features of endocrine disease, e.g. hyperlipidaemia, acromegally, cushing's syndrome.

Hair

- Note the distribution and Texture of hair and grooming
-

Eyes

- Simple tests of visual acuity: compare one eye against the other
- Check for exophthalmos or enophthalmoPtosis
- Oedema of the lids
- Observe the Conjunctivae for anaemia, jaundice or inflammation
- Observe the Pupils for the size, equality, regularity, reaction to light, accommodation
- Eye movement: nystagmus, strabismus
- Ophthalmoscopic examination of the fundi and ocular chambers

Face

- Check for facial symmetry or asymmetry
- Jaw movements
- RashFeatures of endocrine disease or hyperlipidaemia
- Inspect the for Nose/ Sinuses
- Inspect the external nose, nasal Mucosa and septum,
- Palpate frontal and maxillary sinuses for evidence of tenderness.

Mouth and pharynx (a touch and tongue depressor should be used)

- Breath odours
- Inspect the Lips for the colour and eruptions
- Check for any tongue protrusion and appearance
- Inspect the Teeth and gums (if patient has dentures, notice whether they fit and ask whether they are worn for meals or only for cosmetic reasons).
- Inspect the buccal mucous membrane for the colour and pigmentation
- Pharynx (movement of soft palate, state of tonsils)

Neck

- Palpate thyroid gland and cervical nodes
- Asculate carotids for pulse
- Note presence of jugular venous distension and angle of distension
- Note range of neck movements and neck rigidity

Upper limbs

- General examination of hands and arms
- Inspect the Fingernails for clubbing or koilonychias
- Pulse: rate, rhythm, volume and character
- State or arterial walls of radials and brachials
- Axillae: lymph glands
- Blood pressure
- Muscles: muscle wasting, fasciculation
- Tests for power, tone, reflexes and co-ordination
- Cutaneous sensation: check all modalities to exclude root or nerve lesions
- Joints: movement, pain and swelling

Thorax

Anteriorly and laterally

- Note the type of chest, asymmetry if anyInspect the breasts and nipples for size, shape, inversion, rashes, ulceration, and discharge.
- Observe the respiration movement rate, depth and character
-
- Dilated vessels
- Palpate for the Position of trachea
- Look for and palpate apex beat
- Palpate over pericardium for thrills
- Estimate tactile vocal fremitus
- Percuss the lungs
- Auscultate the heart sounds
- Estimate vocal resonance, cervical and axillary glands

Posteriorly (patient sitting)

- Inspect and palpate respiratory movement
- Estimate tactile vocal fremitus
- Percuss the lung resonance
- Ascultate the breath sounds
- Estimate vocal resonance
- Note movements and deformities of the spine
- Palpate from behind: cervical glands, thyroidLook for sacral oedema

- Note any kyphosis, scoliosis
- Note presence of tenderness and range of motion of Back

Abdomen

- Inspection: size, distension, symmetry
- On the Abdominal wall inspect the movement, scars, dilated vessels
- Inspect for visible peristalsis or pulsation
- Observe pubic hair
- Hernial orifices
- Palpation the abdomen for tenderness, rigidity, hyperaesthesia, splashing, masses, liver, gallbladder, spleen, kidneys, bladder
- Abdominal reflexes**Rectal examination**
- Examine anus and rectal wall for lesions, inflammation and sphincter muscles.
- Note any Nodules and other abnormalities
- Observe any fecal matter for occult blood
- In males palpate for prostate gland

Lower limbs

- General examination of legs and feet
- Check the stance, balance and gait
- Observe for the pedal OedemaInspect for varicose veins
- Muscles:, check for muscle wasting, fasciculation
- Tests for power, tones, reflexes (including plantar response) and co-ordination
- Joints, check for movements, pain and swelling
- Palpate for peripheral pulses
- Palpate for Temperature of feet

We have now come to the end of this unit. Let us review what you have learnt.

1.6 Summary

In this unit you've learnt the principles of medicine and some of these principles are; autonomy, confidentiality, fidelity, justice, respect for persons, sanctity of life, beneficence and so on. You have also learnt the nature and causes of diseases and seen that they may occur naturally or artificially. Disease can cause signs and symptoms or may not, depending on the degree of severity. Causes of disease are several; they include biological agents, physical agents, chemicals, deficiencies and excesses, emotions, tissue responses or may be unknown.

We also looked at health assessment which was defined as process of obtaining data from the patient. In our health assessment we looked at two main ways of obtaining data which are; history taking and physical examination. Under History taking we reviewed the types of data that should be obtained from the patient. The data include demographic/ biographic, present complaint, history of previous illness, social history, occupational history, menstrual and obstetric history. Physical examination which is one of the components of health assessment was defined as an evaluation of the body functions using inspection, palpation, percussion and auscultation. The examination uses four major techniques which are inspection, palpation, percussion and auscultation.

Hope you have enjoyed and learnt something from this lesson. In the next unit you will learn about conditions of the digestive system. But before then, test your understanding of this unit by completing the following self-test

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1.7 Self- Assessment Test

Section A: Indicate true or false

1. The following are principles of medicine and medical nursing:
   a. Autonomy
   b. Order
   c. Discipline
   d. Respect for persons
   e. Beneficence
2. Health assessment include
   a. Principles of medicine and medical nursing
   b. History taking
   c. Physical examinations
3. The following are causes of diseases
   a. Biological agents

b. Emotions
c. Good nutrition
d. Deficiencies and excesses
e. Tissue response

4. Diseases always cause symptoms

Section B: Choose the best answer

5. The information which is collected from a patient during history taking include:
   a. History of present illness
   b. Palpation
   c. Auscultation
   d. Inspection

6. Which technique of physical examination sense of sight
   a. Auscultation
   b. Palpation
   c. Percussion
   d. Inspection

Section c: Fill in the blanks

7. Any condition that impairs normal function is referred to as ----------
8. The principle of medicine and medical nursing that relates to concept of privacy is ---------

1.8Answers to self-Assessment test

1. a- True
   b. - False
   c. - False
   d. - True
   e – True

2. a –False
   b – True
   c – True

3. a –True
   b – True
   c – False
   d –True
   e – True

4. False
5. a
6. d
7. Disease
8. Confidentiality

1.9References

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UNIT 2: DIGESTIVE SYSTEM

2.1 UnitIntroduction

You have finished the first unit in this course and now moving on to the second unit which is on digestive system. In the first unit, you looked at the history of medicine and medical nursing. You have learnt about the the principles of medicine and medical nursing which included autonomy, confidentiality, fidelity, justice among others. You also learnt that diseases can arise from inside the body or can be from outside factors and some of the causes include; biological agents such as bacteria; physical agents, emotions, deficiencies and excesses, tissue responses or the cause may be unknown. Furthermor,youdiscussed about health assessment., history taking and physical examination..

You are now welcome to this unit. Inthis unit you will review the applied anatomy and physiology of the digestive system and desceibe the roles of the nurse in investigations and procedures of the digestive system.You will further learnthe common conditions of the digestive system and how they are managed.. By the end of this unit you should be able to achieve the unit objectives which are stated below.

2.2 Unit Objectives

By the end of this unit you should be able to:

2.3. Describe the anatomy and physiology of the digestive system.
2.4 Explain the role of the nurse in common GIT investigations and procedures.
2.5Discuss the management of a patient with oral and oesophageal disorders
2.6Discuss the management of a patient with stomach and duodenal disorders
2.7Discuss the management of a patient with intestinal disorders
2.8Discuss the management of the patient with hepatic disorders
2.9Discuss the management of a patient with biliary disorders
2.10Discuss the management of patientswith worm infestations

-

2.3 Applied Anatomy And physiology Of The Digestive System

You are welcome once again to this unit.However, this time you are going to apply the knowledge you acquired in the foundation blook when you were learning you Anatomy and Physiology course.This will help you to manage patients withdisorders of the digestive system

ACTIVITY 1

Before you go through this section of anatomy and physiology of the GIT, write down the main structures of this system and try to outline the functions of each.

Well done. Now we can proceed.

The digestive tract is 7 m to 7.9 m pathwaythat extends from the mouth to the esophagus, stomach,small and large intestines, and rectum to the terminalstructure, the anus.It is responsible for ingestion (taking in food and fluids), digestion (breaking up the food into manageable sizes/pieces, absorption (extracting the nutritional content of food) and elimination (expelling/excreting the waste products of digestion).

The mouth is the first part of the digestive system and consists of the oral cavity which is surrounded by two pairs of lips. It is lined with the mucous membrane inside. It also comprises of the tongue, 3 pairs of salivary glands (parotid, salivary and submandibular glands) and teeth. It carries out 3 important functions which are breaking down of food mechanically (chewing or mastication), initiating chemical breakdown of food (salivation) and swallowing food (deglutition).

The oesophaguswhich is a hollow muscular tube is located in the mediastinum in the thoracic cavity. This hollow muscular tube is approximately 25 cm in length and passes throughthe diaphragm at an opening called the diaphragmatichiatus.Its function is to propel food from the mouth to the stomach using the waves of rhythmic muscular contractions and relaxations called peristalsis. Peristalsis is entirely under involuntary control. Relaxation of the esophageal sphincter allows the food to enter the stomach.

The stomach is a J shaped hollow muscular organ which lies in the epigastric region and partly in the left hypochondria.It has the capacity ofapproximately 1500 mL.(Davidson's principles and practice of medicine,2007). It can be divided into 3 main regions; the fundus , body and antrum. The pyrolus is a small portion of the antrum and is proximal to the pyrolic sphincter. The stomach serves as a temporal storage for food, mixes the food with gastric secretions (mucus, hydrochloric acid and pepsin). Mucus, Hydrochloric acid and precursor of pepsin are secreted by the cells which line the stomach. The mucus coats the cells of the stomach protecting them from damage by the acid and the enzymes while the hydrochloric acid maintains the acidity of the stomach needed for pepsin to breakdown proteins. The release of the gastric juice is stimulated by sight, smell and thought of food. A hormone called gastrin which is released by stomach walls also stimulates the release of gastric juice. Digestion of food is controlled by several factors; the presence of food which stretches the stomach wall (activates stretch receptors and stimulates the release of gastric juices, presence of certain foods in the stomach such as caffein and proteins stimulates gastrin release.

The small intestine is the largest segment of the GIT (2/3 of total length) and extends fromthe pyrolic sphincter to the ileocaecal valve.This valve, or sphincter,controls the flow of digested material from the ileum intothe cecal portion of the large intestine and prevents refluxthe distribution of the branches of the superior mesenteric artery.It is divided into 3 parts; the duodenum, jujenum and the ileum. The small intestine is responsible for completion of digestion, absorption of nutrients and reabsorption of water that enters the digestive tract. The duodenum receives the pancreatic enzymes from the pancreas and bile from the liver which aid in digestion through the sphincter of oddi. The small intestine has minute hair like projections called villi

which are responsible for absorption. The intestinal wall releases mucus which lubricates the intestinal contents and water which helps to dissolve the digested fragments.

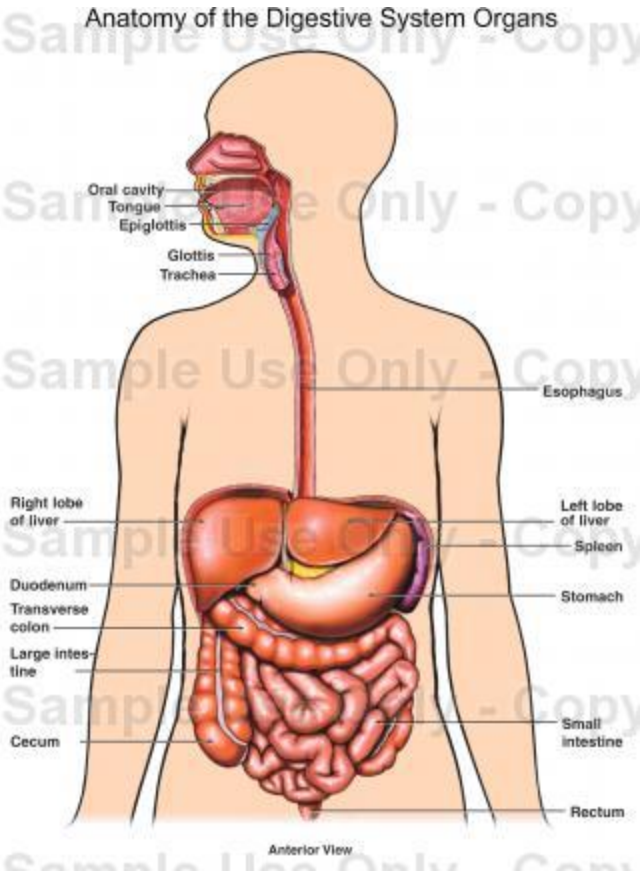
The large intestine is a hollow muscular tube which is about 1.5- 2m long. It has 4 parts; caecum and appendix, the colon (ascending, transverse and descending colon), rectum and the anus. Its main functions are; absorption of water and electrolytes, forms faeces and acts as a reservoir for faecal matter till defecation takes place.

The pancreas which lies below and behind the stomach secretes pancreatic juice enzymes and bicarbonate which aid in digestion of proteins, fats and carbohydrates and protects the duodenum by neutralising the acids respectively. The endocrine function of the pancreas is to excrete insulin and glucagon in response to fluctuating blood sugar levels. The initial stimulus of release of pancreatic juice is food entering the duodenum.

The liver isthe largest single organ in the body and is located in the upper right quadrant of the abdomen just below the diaphragm. The liver has many functions which include; manufactures bile which is stored in the gall bladder, metabolises proteins, carbohydrates and fats and also stores some glycogen among others.

The gall bladder is apear shaped organ located below the liver. Its function is to concentrate and store bile which is produced in the liver.

The diagram below summarizes the main structures of the GIT.



FigureTableFigure 1 Then Anatomy of the digestive system organs

Source:
http://www.bing.com/images/search?q=Digestive%20system&FORM=BILH1#view=detail&id=FB2A2BA3B9FFE7F8C4AA008FDE2706C0B152BE0D&selectedIndex=127

Hope you have remembered the anatomy and physiology of the GIT and now we can proceed and discuss on some of the common investigations which are done in GIT disorders.

2.4 The Role Of The Nurse In Investigations And Procedures

In text Question 2.1

Do you think the nurse has an important role to play in carrying out investigations and procedures on the patients?

The nurse has an important role to play in investigations and procedures that are done on the patients. The following are some of the duties which are carried out by the nurse in investigations and procedures:

- To prepare the patient psychologically and physically.
- To prepare the equipment to be used during the procedure.
- To comfort the patient during the procedure.
- To assist the doctor during the procedure.
- To make the patient comfortable during and after the procedure and to make the appropriate observations of the patient following the investigation, to record them and report any irregularities to the senior staff e.g. ward in-charge or doctor.
- To remove the used equipment and prepare it for use again.
- To ensure that specimen is correctly labelled in suitable containers and are sent to the laboratory.

There are various investigations and procedures that are done to come up with the correct diagnosis of which some may be invasive (penetrate patients' tissues and some may be not).

The following are the investigations and procedures that are done in GIT disorders::

- Endoscopies
- Liver biopsy
- Barium swallow
- Barium meal
- Barium enema
- Ultra sound scan
- Stool examinations
- Liver function tests
- Cholecystography
- Porto-splenography
- Paracentecis abdominis

Let us now discuss these investigations and procedures in detail.

Endoscopies

Endoscopy refers to direct visualization of the body structures through a lighted fibre-optic instrument (endoscope). The structures that can be examined by endoscopy include; oesophagus, stomach, duodenum and colon. When examining the oesophagus, the procedure is called **esophageoscopy**, stomach – gastroscopy, oesophagus, stomach and duodenum – **esophagogastroduodenoscopy (EGD),** rectum –sigmoidoscopy and colon up to ileocaecal is **colonoscopy**. The structures are visualised for motility, inflammation, strictures, ulcers, tumours, etc. The tube can either be inserted through the mouth or through the anus (sigmoidocsopy). The endoscopies are equipped with different instruments such as cameras to take pictures, small

clippers to remove tissue samples and electric probe to destroy abnormal tissues. Endoscopies therefore can be used for diagnosis and treatment.

FigureTableFigure 2: Endoscopy procedure

Figure 2.2 shows a physician performing an endoscopy procedure on a patient.

Liver biopsy

This is a diagnostic procedure which is done to obtain liver tissue for examination. It is used to diagnose hepatic (liver) diseases. A sample can be obtained during laparotomy or it can involve insertion of a needle between 6^{th} and 7^{th} or 8^{th} and 9^{th} intercostal spaces on the right. A CT (computed tomography) scan or ultrasound guide is used if a needle is used to obtain the sample.

Radiological studies

These involve injection or ingestion of a contrast medium followed by x-rays. The contrast medium will coat the gastro-intestinal tract to aid easy visualisation.

Barium Swallow

This procedure permits radiological visualisation of the oesophagus. It can be used to detect strictures, obstruction, ulcers, tumours, polyps, hiatus hernias and motility problems. It involves the patient ingestion of a radio-opaque contrast medium (barium) and the GI tract being

visualised on x-ray fluoroscopy. The lumen will be visualised by the barium so any mucosa destruction, distortion or deviation from the norm can be assessed.

Barium Meal

It is the radiographic examination of the stomach and duodenum after ingesting a radio-opaque contrast media. In a barium meal test, X-ray images of the stomach and the beginning of the duodenumare taken

N.B: A barium meal test is often performed straight after a barium swallow test.

Indications

The procedure is useful in detecting suspected peptic ulcers,gastric ulcers,cancer of the stomach and hiatus hernia.

Take Note 2.1
This procedure is rarely done nowadays because of the use of endoscopy.

Ultrasound scan

Ultrasound scanning or sonography is a non-invasive investigation that is used toobtain pictures or images from different organs inside the human body using high frequency sound waves. The patient is not exposed to radiation. In abdominal ultrasound the examiner passes a probe against person's abdomen. The pictures are displayed on a video screen and recorded on a film. Ultra sound can show size and shape of various organs such as the gallbladder, liver and pancreas and can detect abdominal masses, liver diseases, gallstones, etc. Little or no preparation of the patient is needed except explanation.

Stool examinations

Stool specimen can be collected to diagnose:

1) Occult blood: detects GI bleeding. Used for an early diagnosis of rectal cancers of the GIT and peptic ulcers.

2)Ova and parasites: stool specimen to detect intestinal infections caused by parasites and ova.

3)Stool cultures: identify pathogenic organisms in the GI tract. Sterile technique for collection of stool specimens is used.

4)Stool for lipids: normal dietary lipids are absorbed. Excessive secretion of faecal fats (stetorrhoea) may occur in various digestive and absorptive disorders. A high fat diet is eaten for 3 days prior to specimen collection, which conductedafter 72 hours. Failure of pancreatic lipase to reach the intestine results in undigested and unabsorbed fat.

Liver Function Tests

As the liver performs its various functions it makes chemicals that pass into the bloodstream and bile. Various liver disorders alter the blood level of these chemicals. Some of these chemicals can be measured in a blood sample. The tests that are commonly done on a blood sample are called liver function tests (LFTs). These usually measure the following

Alanine transaminase (ALT) - This is an enzyme that helps to process proteins. (An enzyme is a protein that helps to speed up chemical reactions). Various enzymes occur in the cells in the body.) Large amounts of ALT occur in liver cells. The levels of ALT usually raise when the liver is injured or inflamed (hepatitis).

Aspartate aminotransferase (AST) - This is another enzyme usually found inside liver cells. High levels of this enzyme in the blood indicate an injury to the liver. This test is not all that specific because it can also rise when there is injury to skeletal and heart muscles. For this reason, ALT is mostly requested than AST.

Alkaline phosphatase (ALP) - This enzyme occurs mainly in liver cells next to bile ducts, and in bone. The blood level is raised in some types of liver and bone disease. It also rises in biliary obstruction.

Albumin –It's a main protein which is made by the liver and circulates in the blood. A low level of blood albumin indicates liver disorders.

Bilirubin – It is a chemical that colours the bile. A high level of bilirubin in blood makes one to be jaundiced ('yellow'). Bilirubin is a by-product of haemoglobin during normal or abnormal RBC haemolysis.

Liver cells take in bilirubin and attach sugar molecules to it. This is then called 'conjugated' bilirubin which is passed into the bile ducts. A raised level of 'unconjugated' bilirubin occurs when there is excessive breakdown of red blood cellse.g., in haemolytic anaemia.

Prothrombin time - a laboratory test used to evaluate normal or abnormal blood clotting. Normal range is 11 to 13.5 seconds.

A high prothrombin time can be a sign of liver damage.

Fat metabolism: This looks at the total serum cholesterol and cholesterol esters.

The normal range is < 200mg/dl.

Liver function tests are used for diagnosis of liver conditions and also monitoring the toxic effects of some drug.(Davidson's Principles & Practice of Medicine, 2007)

Cholecystography

It's radiological examination of the gall bladder or cystic duct using a radio- opaque contrast medium. The evening before the test the patient ingests a radio-opaque dye. The dye isabsorbed by the gut, taken to the liver and secreted in bile, it therefore reaches the gall bladder.This enables the gall bladder to be visualised on a straight abdominal x-ray taken 24 hours afteringestion of the dye. It can be used to detect gall stones (cholelithiasis). X-ray film will show an opaque gall bladder and the gall stones will be seen as dark spots. Poor visualisation indicates gall bladder disease. Biliary obstruction prevents passage of the dye. A sensitivity test should be done first for the dye.

Porto-splenography

This is a radiological procedure which is used to detect conditions that affect the spleen.

Cholecystography and splenography are rarely done nowadays; investigations such as ultrasound are used instead.

Paracentesis Abdominis/ Abdominal Paracentesis

It is a procedure in which fluid is withdrawn from the abdominal cavity. It involves making an incision in the skin and passing a hollow trocar, cannular, or catheter through the incision into the cavity to allow out flow of fluid into a collecting device.

Paracentesis can be done to obtain a sample for analysis or to remove excess fluids in abdominal distension.

Activity:2.1

Write down the investigations that are commonly used in GIT disorders.

Well done and check your answers.

You have just finished looking at the roles of a nurse in investigations and procedures conducted in patients with digestive disorders. Am sure you have discovered that a nurse is a very important member of the health care team hence the reason to have the knowledge.

You will now learn on the management of patients with oral and esophageal disorders. Pay particular attention to these conditions to enable you manage these patients very well.

2.5 Management Of A Patient With Oral And Esophageal Disorders

Oral and esophageal disorders

STOMATITIS

Definition:.Stomatitis is inflammation of the mucosa of the mouth. It may be caused by one of many diseases of the mouth or it may accompany another disease.

Types Of Stomatitis

There are different types of stomatitis and these are:

SIMPLE CATARRHAL STOMATITIS

Simple catarrhal stomatitis is inflammation of the mucous membranes of the mouth with increased flow of mucus and exudates.

Its occurrence is more in children than in adults.

Causes

- Micro-organisms such as bacteria.
- Poor oral hygiene/neglected mouth.
- Hot foods/drinks.
- Wounds caused by foreign bodies in the mouth.
- Corrosion from strong acids or alkali.
- Systemic infections.

Signs and symptoms

- Low grade fever.
- Dry mucus membrane.
- Sores in the mouth.
- Pain in the mouth especially when eating.
- Red mucus membrane.
- Loss of appetite and craving for cold drinks.

Treatment

- Antipyretics such as paracetamol 1g tds fro three per day..
- Mouth wash with antiseptics.
- Soft diet.
- Treat the existing condition if cause is systemic condition

VINCENT'S sTOMATITIS

It is a severe inflammation of the mouth and gums caused by bacterial infection. Vincent's stomatitis is also considered as severe gingivitis.

Cause

- Bacteria
- Poor oral hygiene
- Immunosuppresion.

Signs and symptoms

- Pain in the mouth.
- Swelling of the affected parts.
- Bleeding from the gums.
- Redness of the mucous membrane.
- Bad taste
- Halitosis
- Fever due to infection.

Diagnosis

- Physical examination will reveal:
  - ✓ Mouth and gum swelling
  - ✓ Inflamed gums:
  - ✓ Gum redness
- Dental x-rays
- History taking

Treatment

- Oral antibiotics such as penicillin, erythromycin.
- Antiseptic mouthwash
- Hydrogen peroxide rinses

- Regular brushing
- Professional dental cleaning.

MONILIA STOMATITIS/ ORAL THRUSH

It is a mouth infection which is caused by a yeast-like fungus (Candida albicans)

Predisposing factors

It commonly occurs in people with:

- Lowered immunity
- Prolonged use of antibiotics such as tetracycline and chloramphenicol because the normal floras of the mouth which usually keep fungi at bay are destroyed by antibiotics.

Signs and symptoms

Lesion on the mucous membrane and on the gums which easily bleed when tempered with.

Treatment includes:

- Hydrogen peroxide and normal saline mouthwashes
- Clotrimazole tablets dissolved in the mouth five times/day
- Nystatin suspension or pastilles or amphotericin lozenges
- Fluconazole for oropharyngeal candidiasis
- Oral hygiene

HEPATIC STOMATITIS

It is a contagious viral infection of the mouth that causes ulcers and inflammation. It is common in children.

Causes

- Herpes virus hominis
- Epstein-Barr virus
- Varicella zoster

Signs and symptoms

- Blisters in the mouth, often on the tongue or cheeks.
- Decrease in food intake, even when the patient is hungry
- Dysphagia
- Drooling

- Fever which may occur 1 - 2 days before blisters and ulcers appear.
- Irritability
- Pain in mouth
- Swollen gums
- Ulcers in the mouth, often on the tongue or cheeks -- these forms after the blisters pop.

Diagnosis

History taking

Physical examinations

Treatment

- Antiviral - acyclovir.
- Liquid diet which are cool-to-cold, nonacidic drinks.
- For severe pain give oral topical anaesthetic –lidocaine. Give with caution because it can interfere with swallowing and can possibly cause burns on the mouth or throat.

PAROTITIS

Parotitis is inflammation of one or both parotid glands.

Causes

- Bacterial infection- staphylococcus aureus.
- Mycobacterium, the bacteria that causes tuberculosis.
- Mumps virus.
- HIV
- Blockage of the main parotid duct, or one of its branches.
- Systemic infection

Signs and symptoms

- Swollen and painful gland which is seen at the jaw angle.
- Dry mouth
- Severe pain when swallowing.
- Purulent exudates from gland.
- Erythema
- Ulcers
- Fever

Diagnosis

History taking

Physical examination- Enlarged gland will be seen on examination.

Treatment

- Antibiotics
- Mouth washes
- Warm salty water rinses may be soothing and keep the mouth moist.
- Warm compresses
- Increase fluid intake
- If an abscess develops, drainage is necessary.

We have now finished looking at the conditions of the mouth, let's now move andlook at theoesophagus.

ACHALASIA

Achalasia is a neuromuscular disorder of unknown cause in which propulsive (forward moving) peristalsis is absent and the lower oesophageal sphincter only relaxes to an insufficient extent.

It is a gastrointestinal tract condition which is characterised with lack of peristaltic movement in the oesophagus and failure of relaxation of lower esophageal sphincter.

It can also be defined as an esophageal motility disorder in which there is failure of the esophagogastric (cardiac) sphincter to relax in order to allow the passage of food into the stomach accompanied by a lack of tone in the musculature and normal peristalsis, particularly in the lower part of the oesophagus resulting in the accumulation and stagnation of food and fluids in the oesophagus causing irritation and inflammation of the oesophagus.

Cause

The exact cause is unknown but it has been associated with degenerative changes or malfunctioning in the nerve plexus that innervates the esophageal muscle tissue.

Signs and symptoms

- Progressive dysphagia.
- Regurgitation of undigested food.
- Weight loss,
- Halitosis caused by regurgitation of previously ingested food.
- Coughing when lying in a horizontal position.
- Chest pains that may increase after eating.

Diagnosis

- Barium swallow will show dilatation of the oesophagus, lack of peristalsis.
- Esophagoscopy – will show dilatation of the lower esophageal sphincter. It can also show changes associated with cancer or presence of candida.
- Oesophageal manometry – This will be done to measure muscle contractions in different parts of the oesophagus during the act of swallowing. Manometry reveals failure of the lower oesophageal sphincter to relax with swallowing and lack of functional peristalsis in the smooth muscle oesophagus.
- Biopsy, the removal of a tissue sample during endoscopy shows hypertrophied muscles and absence of certain nerve cells of the mesenteric plexus (network of nerve fibres that controls oesophageal peristalsis).

Treatment

- Achalasia may be treated conservatively by pneumatic dilation to stretch the narrowed area of the esophagus. Pneumatic dilation has a high success rate.
- Medication - include calcium channel blockers such as nifedipine and nitrates such as nitroglycerin to relax the lower oesophagus sphincter.
- Balloon (pneumatic) dilatation – This procedure is done to dilate the oesophagus at the point of narrowing. It involves inserting a balloon into the oesophagus inside the lower esophageal sphincter to stretch the lower sphincter.
- Surgery - Heller myotomy or cardiomyotomy (surgical division of the muscle of the lower end of the oesophagus.

GASTROESOPHAGEAL REFLUX

Definition

This is a condition in which there is backflow (reflux) of gastric and or duodenal content into the oesophagus which is not associated with either vomiting or belching.

Gastro esophageal reflux is a GIT condition that results from abnormal regurgitation of gastric contents into the oesophagus.

Cause

It is usually caused by changes in the barrier between the stomach and the oesophagus, including

- Abnormal relaxation of the lower oesophageal sphincter.
- Anatomical abnormalities such as hiatus hernia(condition where the upper part of the stomach and the Lower Esophageal Sphincter move above the diaphragm).

Predisposing factors

The following factors can increase the chance of developing gastroesophageal reflux:

- Obesity: increasing body mass index is associated with more severe gastresophageal reflux.
- Zollinger-Ellison syndrome, which can be present with increased gastric acidity due to gastrin production.
- Pregnancy
- Smocking
- Hypocalcaemia, which can increase gastrin production, leading to increased acidity.
- Food such as sodas that contain caffeine, chocolates, spicies, acidic foods like oranges, fat foods.
- Alcohol consumption
- Large meal
- Drugs such as anticholinergics, calcium channel blockers, nitrates.
- Systemic sclerosis
- Hiatus hernia
- Nasogastric tube placement for more than 4 days.

Signs and symptoms

- Heartburn – burning sensation behind the breastbone and usually occurs after meals. This is accompanied by regurgitation.
- Pain – this originates in the chest and radiates to the neck and throat. Pain is caused by acid regurgitation.
- Dysphagia(difficulties in swallowing).

Less-common symptoms include:

- Odynophagia (Pain with swallowing).
- Excessive salivation (also known as water brash) is common during heartburn, as saliva is generally slightly alkalineand is the body's natural response to heartburn, acting similarly to an antacid)
- Nausea
- Coughing, hoarseness, or wheezing at night.
- Belching
- Flatulence

Diagnosis

- Esophagoscopy is often used to identify the cause and examine the oesophagus for damage.
- Barium swallow to evaluate esophageal damage as well.

- Continuous oesophageal pH monitoring to evaluate degree of acid reflux.
- Oesophageal manometry
- A positive stool occult blood test may diagnose bleeding from the irritation in the oesophagus.

Treatment

- Life style changes
  - ✓ Avoid foods that often cause symptoms e.g. Alcohol, Caffeine, Carbonated beverages, Chocolate, Citrus fruits and juices, Tomatoes.
  - ✓ Advise patient to lose weight if obese.
  - ✓ Raise head of the bed 6-8 inches and patient should avoid lying down 3 hours after taking a meal.
  - ✓ Advise patient to stop smoking
  - ✓ Eat slowly and chew food thoroughly to reduce belching.
- Antiacid such as magnesium transilicate to neutralise the acid.
- Proton pump inhibitors are the most potent acid inhibitors e.g. omeprazole Proton pump inhibitors (PPIs) decrease the amount of acid produced the stomach.
- H2 antagonists such as cimetidine to decrease acid production in the stomach.
- Surgery - Nissen fundoplication – procedure to repair lower esophageal sphincter Repair a hiatus hernia
- Vagotomy (surgical removal of vagus nerve branches that innervate the stomach lining).

Thank you for your attention.You will now learn about hiccup

HICCUP

Definitions

A hiccup or hiccough is an esophageal contraction of the diaphragm that repeats several times per minute.

A spasm of the diaphragm resulting in a rapid, involuntary inhalation that is stopped by the sudden closure of the glottis and accompanied by a sharp, distinctive sound.

Causes

- Hiccups are caused by many central and peripheral nervous system disorders, all from injury or irritation to the phrenic and vagus nerves, as well as toxic or metabolic disorders.
- Chemotherapy—which can include a huge amount of different drugs—has been associated with hiccups.
- Hiccups often occur after drinking carbonated beverages or alcohol or ingesting spicy foods.
- Prolonged laughter is also known to cause hiccups.
- Eating too fast can also cause the hiccups.

Treatment

- Ordinary hiccups are cured easily without medical intervention. However, there are a number of anecdotal treatments for casual cases of hiccups. Some of the common home remedies include giving the afflicted a fright or shock.
- Eating peanut butter, taking a teaspoon of vinegar, drinking water, holding one's breath and altering one's breathing patterns.
- Hiccups are treated medically only in severe and persistent cases (termed "intractable“).
- Sedatives such as Diazepam and chlorpromazine are given.

We have now finished discussing the conditions of the mouth and the oesophagus and now moving on to the conditions of the stomach. But before we proceed, let’s discuss the are the common manifestations of the GIT and how they can be managed.

2.6 Management Of The Patient With Stomach And Duodenal Disorders

Gastritis

Definition

Gastritis is an inflammation of the gastric mucosa or lining of the stomach. It can be classified as either acute or chronic.

Pathophysiology of gastritis

The gastric mucosa is protected from acid auto-digestion by prostglandings. Injury occurs when there is a break in the protective barrier. The resulting injury is compounded by histamine release and vagal nerve stimulation which leads to hydrochloric acid diffusing back into the mucosa and injure small vessels resulting in oedema, haemorrhage, and erosion of the stomach’s lining. As the disease progresses, the walls and lining of the stomach thin and atrophy decreasing the function of the parietal cells. The source of intrinsic factor is also lost and the absorption of vitamin B 12 is impaired leading to pernicious anaemia(Smeltzer etal 2010).

ACUTE gASTRITIS

Definition: this is an acute inflammation of the gastric mucosa or submuosa with destruction of the superficial epithelial cells (Bloom, 2005). The condition lasts a few hours to few days.

Causes

- ✓ Thermal causes**:** very hot fluids or food.
- ✓ Chemical Causes
- Ingestion of corrosive substances.
- Food which cause irritation such as spiced food , alcohol.
- Drugs such as aspirin and other nonsteroidal anti-inflammatory agents (in large doses), cytotoxic agents, caffeine, corticosteroids, antimetabolites, phenylbutazone, and indomethacin excavates the mucosa of the stomach.
- ✓ Bacterial causes**:** endotoxins released from infecting bacteria such as staphylococci, Escherichia coli, salmonella and helicobacter pylori.
- ✓ Conditions-uraemia, shock, prolonged emotional tension, major burns, hepatic diseases, renal diseases, peptic ulcers, major surgery.
- ✓ Bile reflux: A backflow of bile into the stomach from the bile tract (that connects to the liver and gallbladder) which irritates the mucosa membrane of the stomach.
- ✓ Bile reflux: A backflow of bile into the stomach from the bile tract (that connects to the liver and gallbladder) irritates the mucosa membrane of the stomach.
- ✓ Exposure to certain procedures such as nasal gastric tube insertion and endoscopy.

Signs and symptoms

- Sudden onset of symptoms
- Anorexia
- Nausea and vomiting
- Dyspepsia
- Epigastric pains of varying severity
- Haematemesis and maleana stool if gastric bleeding occurs.
- Water brush syndrome – clear fluid which comes through the mouth due to reflex salivation in response to duodenal ulceration.
- Signs and symptoms of pernicious anaemia (lack of cynocobalamine – Vitamin responsible for maturation of RBC) due to loss of intrinsic factor.
- Colic diarrhoea 2 – 4 hours following intake of contaminated food if gastritis is due to food poisoning.
- Fever may be present.

Diagnosis

- Endoscopy/ gastroscopy will show inflamed gastric membranes

- FBC for laboratory tests will reveal low Haemoglobin level due to concealed bleeding as occult blood will be present in vomitus and stool. Erythrocyte Sedimentation Rate (ESR) will also be raised due to infection.
- Histological examination of biopsy specimen
- Serologic testing for antibodies for Helicobacter pylori or breath test.
- Gastric acid analysis which will reveal increased HCL secretion

Medical and Nursing Management

The gastric mucosa is capable of repairing itself after an attack of less severe gastritis.

Patient recovers in about a day, although the appetite may be diminished for an additional 2 or 3 days.

Management is largely symptomatic. Oral fluids and food are withheld while patient is vomiting. Oral fluids and non-irritating food should be re-introduced gradually according to the patient's tolerance. Patient will then progress to normal diet. Intravenous fluids are administered if the gastritis lasts longer than 12-18 hours, or if there is evidence of dehydration and electrolyte imbalance, in this case, the patient will be hospitalized and will need complete bed rest.

The following drugs may be prescribed:

- Antiemetic such as promethazine to relieve nausea and vomiting but avoid when the cause is due to corrosives.
- Anticholinergic such as atropine to decrease gastric secretions and to relax smooth muscle.
- Cimetidine is usually given to reduce gastric acid secretion when there is haemorrhage associated with gastritis.
- 'However,if gastritis is caused by ingestion of strong acids or alkalis,emergency treatment consists of diluting and neutralizing the offending agent. To neutralize acids, common antacids (eg, aluminum hydroxide) are used; to neutralize an alkali, diluted lemon juice or diluted vinegar is used(Smeltzer etal 2010)'.

CHRONIC gASTRITIS

Definition

It is a chronic degeneration of the mucosa membranes of the stomach which may follow prolonged dietary indiscretion or alcohol abuse.

Causes

- Helicobacter pylori (most cause).
- Alcohol abuse
- Thyroid disease
- Diabetes mellitus
- Radiation therapy

- Repeated attacks of acute gastritis
- Auto immune condition such as pernicious anaemia
- Smoking

Signs and symptoms

- Anorexia leading to weight loss
- Nausea and vomiting (heamatemesis) which relieves pain due to irritations of the gastric mucosa.
- Dyspepsia due to impaired gastric function
- Flatulence (presence of gas) due to impaired gastric function.
- Epigastric pain (heart burn) due to regurgitation of gastric contents
- Abdominal pain due to erosion of the gastric mucosa
- Melena stool due to gastric bleeding
- Constipation which is later followed by diarrhoea caused by enteritis.
- Slow in progression

Diagnosis

- History from the patient will reveal recurrence of acute gastritis.
- Barium meal will reveal inflammation of the gastric mucosa
- Endoscopy/Gastroscocpy reveal inflammation of the gastric mucosa
- Stool for occult blood
- FBC will reveal low HB due to bleeding
- Gastric acid analysis which will reveal increased HCL secretion.

Treatment

- Chronic gastritis is managed by modifying the patient's diet, promoting rest, reducing stress and initiating pharmacotherapy.
- Advise the patient to chew food thoroughly well before swallowing
- Helicobactre Pylori may be treated with combination of drugs such as Amoxicillin, Flagyl and omeprazole (triple therapy).
- Antiemetic drugs are administered to treat nausea and vomiting e.g. promethazine 25-50mgs either IM or IV for 3 days or Plasil 10-20mgs t.d.s by 3day.
- Antiacids to relieve pain or discomforts. such as Aluminum hydroxide 200-400mgs t.d.s for 7days.
- Histamine receptor blockers these drugs help to reduce production of hydrochloric acid e.g. cimetidine 200-400mgs t.d.s for 14days.

Nursing care of gastritis

Aims

- Prevent complications

- Relieve signs and symptoms
- Help in healing process.
- Reducing anxiety.

Environment

Patient is nursed in a general medical ward. Environment should be clean , well ventilated to promote air circulation and quite to promote rest.

Position

Nurse the patient in any comfortable position preferablyin a semi-fowlers position to prevent regurgitation of gastric juice.

Rest

Ensure noise free environment to promote rest. Do nursing activities collectively to avoid disturbances thereby promoting rest.

Observations

Observe general condition of patient to see whether improving, static or worsening. Monitor vital signs such as temperature, pulse, respirations and blood pressure and record the findings. High temperature indicates presence of infection, rapid pulse and low BP will indicate bleeding. Observe stool and vomitus for colour, consistency, amount and odor. If blood is present in stool and vomitus, give ice drinks to patient to constrict blood vessels thereby arresting the bleeding. Check for abdominal tenderness and also observe patient for any complications such as gastric ulcers. Patient should be weighed daily.

Psychological care

Explain condition to patient and his/her relatives in simple terms and this should include possible causes, disease process, treatment and why certain things are not allowed. This is to alley anxiety and gain cooperation. Allow patient to ask questions and answer them correctly. This is to help patient understand his/her condition. Involve patient and relatives in the plan of their care to avoid dependency.

Nutrition and fluids

Provide nutritious balanced meals containing proteins and vitamins to promote healing, carbohydrates to provide energy and vitamins to boost the immunity. Since patient may have anorexia and vomiting, serve food in small frequent amounts to promote appetite and prevent vomiting. Avoid spiced foods for this may worsen the condition. Give fluids either orally or intravenously to prevent dehydration and also to flush out toxins.

Patients should be discouraged to consume alcohol because it can worsen the condition

Elimination

Observe intake and output. Observe stool and vomitus for consistency, colour, amount and odour and then record and report. Observe the bowel patterns of the patient. Give patient food rich in roughage and encourage patient to take a lot of fluids to prevent constipations.

Exercises

Initially patient may be on total bed rest but as the condition improves, encourage patient to do passive exercises to prevent complications such as deep vein thrombosis and to promote blood circulation.

Hygiene

If condition is bad and patient is unable to bath herself, do bed bath to promote hygiene, comfort and blood circulation. Do oral care to prevent mouth infections and promote appetite. Do nail care also to prevent accumulation of dirt. Change bed linen when soiled or dirty.

IEC

Explain to patient the importance of rest.

- Teach patient the disease process.
- Advise patient to avoid spiced foods and also dangers of taking unprescribed drugs.
- Help the patient to identify factors that may aggravate the symptoms.
- Advise patient on importance of drug compliance.
- Advice on importance of following the review dates.
- Teach patient signs and symptoms of complications.
- Emphasise on the need of changing life style such as to stop drinking.

Complications

- Gastric ulcers
- Haemorrhage
- Anaemia
- Obstruction
- Perforation
- Peritonitis
- Cancer of the stomach

<u>Peptic ulcers</u>

Definition

Peptic ulcer is defined as “an erosion of the GI mucosa resulting from the digestive action of hydrochloric acid (HCl) and pepsin” (Basavanthappa, 2005).

Peptic ulcer is an erosion or break in the mucus or tissues of the digestive tract that comes in contact constantly with gastric juices. This erosion may penetrate the muscle layer up to the outside causing bleeding or perforation.

A peptic ulcer is an ulceration involving the mucosa and deeper structures of the upper gastro-intestinal tract and is due to action of the gastric juice.

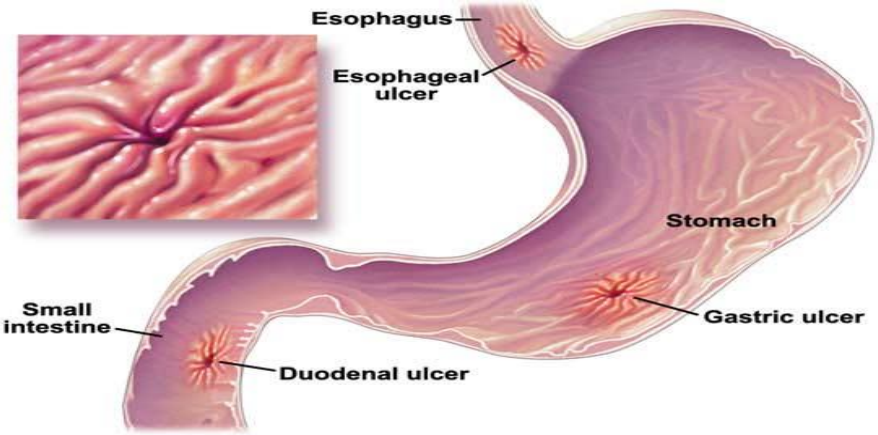
A peptic ulcer is an excavation formed in the mucosal wall of the oesophagus, stomach, or duodenum.

Epidemiology

- Greatest frequency between 40 and 60yrs.
- Peak age in duodenal ulcers is 30-60.
- Peak age in gastric ulcers is 50 and above
- Ratio of male: female in duodenal is 2-3:1.
- Ratio of male: female in gastric is 1:1.
- 80% of PUD are duodenal., 15% are gastric (Smeltzer & Bare, 2004)

Common Sites:

- Lower oesophagus
- Stomach
- Proximal portion of the duodenum.



FigureTableFigure 3: stomach – Sites which are commonly affected by ulcers

Types

Ulcers can be classified according to degree and location.

Type according to degree

Peptic ulcers may be acute or chronic. Acute ulcers are often multiple and are usually located in the fundus of the stomach. These ulcers are thought to be stress related, superficial, and self-

limiting. Occasionally, they penetrate a blood vessel causing haemorrhage of varying degrees of severity.

Chronic ulcers are more common than acute ulcers. These ulcers usually occur as a single lesion with margins that are thickened, hyperaemic, and oedematous. Chronic ulcers tend to recur frequently, causing extensive scarring.

Type according to location

Esophageal ulcers – these ulcers affect the lower portion of the oesophagus and are usually due to weaker esophageal sphincter which allows gastric contents to escape into the oesophagus.

Gastric ulcers – These occur in the stomach mainly in the lesser and greater curvatures and pyloric antrum. The occurrence is due to defective ability of the stomach to heal faster.

Duodenal ulcers – These are the commonest and are seen in the first portion of the duodenum. Ulcers result from high secretion of the hydrochloric acid.

Aetiology

The erosion of the gastric mucosa is caused by the digestive action of hydrochloric acid and pepsin, however, the real cause is not fully understood but there are some factors that precipitate the occurrence. These include

1. Gram negative bacteria Helicobacter pyloriwhich is present in 70% of patients with gastric ulcers and 95% of patients with duodenal ulcers has been associated with peptic ulcers.
2. Emotional Factors: emotional tension, anxiety, frustration and stress may cause an imbalance in the autonomic nervous system, resulting in increased vagal stimulation of gastric secretion.
3. Inflammation: gastritis and trauma of the mucosa reduces the resistance of the membrane to digestion. Cell destruction is accelerated and cell reproduction, which normally renews the superficial layers quickly, may be retarded.
4. Hereditary: gastric ulcers are common in people with type A blood while duodenal ulcers are common in people with type O blood. Duodenal ulcers are three times more common in first-degree relatives of duodenal ulcers patients than in the general population.
5. Trauma and serious illness: critical illnesses especially if it is characterized by hypotension or respiratory insufficiency may complicate into peptic ulcer. Conditions such as severe burns, shock, etc. may lead to peptic ulceration.
6. Prolonged use of irritants: certain drugs including Non-Steroidal Anti-inflammatory Drugs (NSAIDs) may predispose to peptic ulcer disease (e.g. acetylsalicylic acid, adrenal steroids, indomethacin and phenylbutazone). Alcohol inhibits prostaglandin secretion. Nicotine in cigarette smoking inhibits pancreatic secretion of bicarbonate, it also may accelerate the emptying of gastric acid into the duodenum and promote mucosal breakdown.

7. Bile reflux: the reflux of bile and pancreatic enzymes into the stomach due to an incompetent pyrolic sphincter may lead to a gastric ulcer. The bile salts damage the gastric mucosa, predisposing it to ulceration.
8. Normal aging may also wear down the pyloric sphincter, which in turn, permits the reflux of bile into the stomach.
9. Zollinger Ellisons syndrome; Condition characterized by excessive production of hydrochloric acid which erodes the gastric mucosa.

Epidemiology

Duodenal ulcers are very common, 2 to 3 times more than gastric ulcer disease.

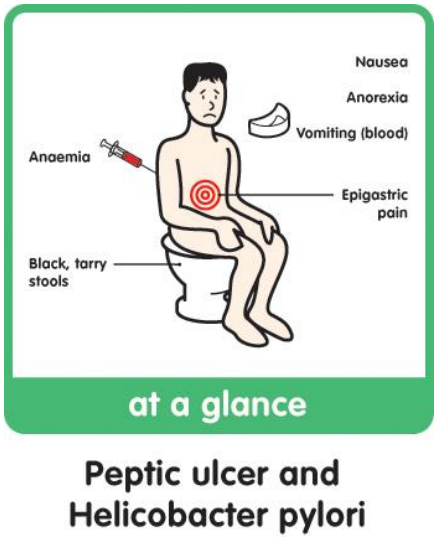
Pathophysiology

Ulceration occurs due to decrease in resistance of the Gastric mucosa to pepsin and acid injury. After ingestion of food acetylcholin, gastrin and histamine bind to specific receptors and stimulate the parietal cells in the fundus of the stomach to secret gastric acid. The parietal cells with the assistance of H+,K+ ATPase transport the HCL to the stomach lumen where the chief cells in the stomach secrete pepsinogen which convert to pepsin in the presence of HCL break down food.

Duodenal cells in the epithelium secret mucus barrier to protect the lining of the gastric duodenal area which dilute the secretion of acid and provides a protective coating against acid action. Peptic ulcer may develop when the secretory output of Hydrochloric acid is more than pepsin thereby eroding the gastric mucosa membrane due to inadequate defense secretion of mucous to neutralize the imbalance thereby promote ulcer development.

Signs and symptoms

What do you think about this picture?



FigureTableFigure 4: Common signs and symptoms in peptic ulcers

Having looked at the pathophysiogy of peptic ulcers what could be some of the signs and symptoms?

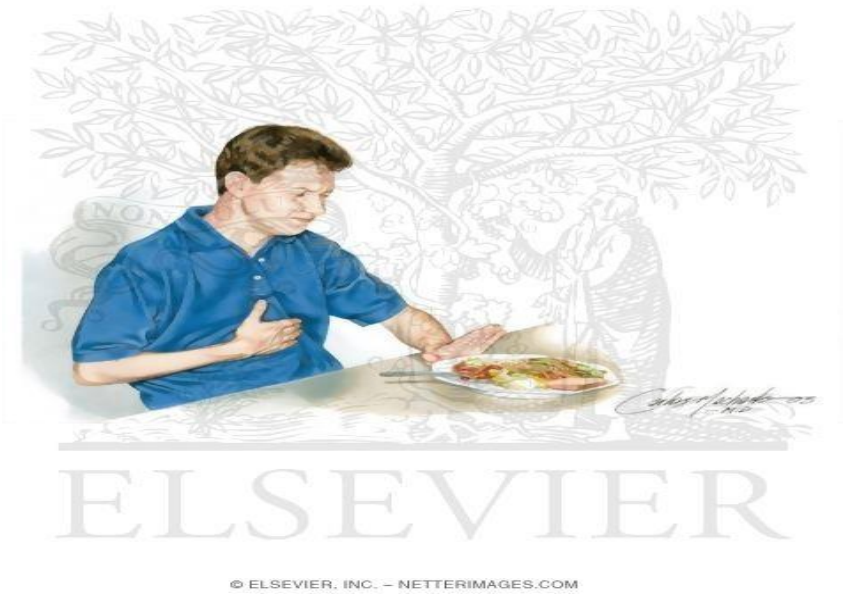
The signs of peptic ulcers include:

- Epigatric pain – a gnawing or burning pain, related to food, radiating to the back due to the erosion of the mucosa lining.
- Heartburn (pyrosis)-burning sensation experienced in some patients due to excessive presence of acid in the oesophagus and stomach
- Vomiting due to severe pain and maybe due to obstruction caused by muscular spasms of the pylorus
- Weight loss especially in those with gastric ulcers.
- Dyspepsia, including belching, bloating, distention, fatty food intolerance
- Chest discomfort
- Anorexia
- Hematemesis or melena resulting from gastro intestinal bleeding from eroded small blood vessels
- Malena stool – passing out black tarry stool. This is more common in duodenal ulcers.
-
- Constipation due to inadequate intake of fluids and fibre foods.

Table Differences between Duodenal and Gastric ulcers

Characteristic	**Gastric ulcers**	**Duodenal ulcers**
Location of lesion	Predominantly antrum, also in the body and fundus of the stomach	First 2cm of the duodenum
Lesion	Superficial with smooth margins, round, oval or cone shaped.	Penetrating and is associated with deformity of duodenum from healing of current ulcers
Gastric secretion	Normal to decreased secretion	Increased
Incidence	Higher in women Peak age 50-60yrs More common in persons of lower socioeconomic status	Higher in men but higher in post-menopausal women Peak age 35-45 yrs. Associated with psychological stress

	Increased with Incompetent pyloric sphincter and bile reflux	Associated with other diseases such as pancreatic disease, Zollinger-Ellison disease
Clinical manifestation	Burning or gaseous pressure in the high left epigastric Pain occurs 1 – 2 hours after meals. Food aggravates the pain. Weight loss Haematemesis	Burning and crampy pain across the mid epigastrium and upper abdomen Pain occurs 2 – 4 hours after meals. Pain is relieved with food. Weight may be normal or patient may gain more weight Meleana
Blood group	No difference	Common in blood group O
Cancerous tendencies	More common to develop cancer	Less common



FigureTableFigure 5: Pain caused by gastric ulcers

Management
Diagnosis

- History taking involves assessment for abdominal pain; determine its location, timing and severity of pain along with associated symptoms and precipitating factors.
- Physical assessment; Examine and palpate the abdomen carefully for pain, which is usually present in the upper epigastrium, left of the midline.
- Endoscopy (esophagogastroduodenoscopy) to visualize and identify inflammatory changes, ulcers and lesions. The duodenal mucosa is visualized.
- Special radiology (Barium swallow and Barium meal) is done to visualize the ulcer, after patient has taken barium sulphate.
- Stool examination to detect presence of blood (occult or fresh) in stool which may be a sign of bleeding from the gut.
- Biopsy may be done for gastric ulcers to diagnose benign status.
- Gastric analysis to help in differentiating gastric ulcer from gastric cancer
- Exfoliative cystology (examination of the secretions and cells that are brushed or scraped from the mucous membranes.

Drug therapy

Aims

1. To provide pain relief
2. To eradicate H. pylori infection
3. To heal ulcerations by reducing gastric secretions and protecting the mucosa from further damage,
4. To prevent recurrence

Eradication therapy

The following regimes are recommended for eradication of helicobacter in patients with gastric and duodenal ulcers, (Boon, et al, 2006).

First line therapy Use proton pumps inhibitor, and a combination of two antibiotics for example Omeprazole, clarithromycin and metronidazole.

Omeprazole 20mg orally twice daily for 4 to 8 weeks

Action: inhibits the activity of the acid pump and binds to hydrogen-potassium adenosine triphosphatase to block the formation of gastric acid.

Side effects: headache, hallucinations dizziness, diarrhoea, abdominal pain, nausea, vomiting, constipation.

Nursing Implications: caution patient not to perform hazardous activities if dizziness occurs; tell patient to swallow capsules whole and not to open or crush them.

Amoxyl 500mg tds orally.

Action: blocks protein synthesis

Side effects: Nausea and vomiting, diarrhoea, abdominal pain or discomfort.

Nursing Implications: use cautiously in patients with hepatic or renal impairment; obtain urine specimen for culture and sensitivity tests before first dose.Begin therapy pending results; monitor patient for super infection.

Metronidazole 400-500mg orally tds

Side effects: metallic taste, nausea and vomiting.

Nursing Implications**:** instruct patient to take drug with food**.**

Second line therapy

Proton pump inhibitor, bismuth, metronidazole and tetracycline.

Bismuth 120mg qid

Metronidazole 400mg bd

Tetracycline 500mg bd

Other Drugs

1. Antacids: These decrease the acidity by neutralizing gastric juice. They do not influence healing or prevent reoccurrence. They raise the pH in the stomach, aiming for a pH of 3.0-3.5. These are taken after meals. Example of antiacids, Aluminium hydroxide, magnesium transilicate.
2. Histamine receptor antagonists -These block histamine-secreted acid, therefore are effective in the management of ulcer disease e.g. Ranitidine 150mg bd or Cimetidine 400mg bd for 4-6 weeks which block the action of Hydrogen receptors and inhibit pepsin secretion and decreases gastric secretion.
   Mucosal Barrier Fortifiers such as sucralfate 1g PO qid or 2g BD 1hr before and meals and at bed time; do not give within 30minutes of giving antacids or other drugs.
   These stimulate mucus production therefore increase ulcer healing and prevent damage to the mucosa by forming a sticky gel and adheres to the ulcer surface forming a protective barrier. Mucosal barrier fortifiers do not inhibit acid secretion and have minimal acid-neutralizing ability.
3. Anticholinergic drugs such as propantheline - These are not often used, as Hydrogen antagonists are preferred. They decrease vagal stimulation (the action of the vagus nerve) therefore reduce gastric motility and secretion. Gastric emptying time is delayed which possibly increases the rate of ulcer healing and enhances pain relief. Not used if there is bleeding or there is a pyloric obstruction

If the above treatment fails, surgery can be performed.

Nursing care

- To relieve symptoms and promote healing of ulcer.
- To reduce anxiety
- Maintain nutrition requirements
- Provide knowledge about disease and management.
- To Prevent complications

Hospitalization is not always necessary, however, if patient can not adhere to treatment regimen or home situation is not conducive, the patient may be hospitalized until symptoms are relieved and patient and relatives appreciate treatment plan. Treatment and care is individualized for all patients.

Environment

The environment should support both mental and physical rest, therefore, nurse patientin a quiet environment to promote rest as patient with peptic ulcers experience severe pain. Environment should be well ventilated to promote comfort. Ensure that environment is free from stressful stimuli as stress increases vagal activity which worsens the condition.

Rest

Mental and physical rest is necessary to reduce gastric activity. A brief period of bed rest is essential. The patient may remain ambulatory with some restriction in activity and an increase in his hours of rest. A period away from work situation is necessary if the symptoms are severe, but for some persons it may be considered better to let them carry on rather than impose the anxiety created by the loss of financial income or by the disorganization of their work.

A quiet pleasant environment, physical comfort, undisturbed rest periods, avoidance of visitors who may arouse unpleasant feelings, diversion therapy and avoidance of delays in relation to his treatments and requests. Anxiety should be relieved: psychological stress may aggravate the ulcer, and the symptoms in turn evoke further anxiety. The nurse should take time to listen and encourage the patient to develop coping mechanisms for stressful situations that tend to aggravate gastric hypersecretion and hypermotility. She should help the patient see the problems in normal perspective and may make constructive suggestions. A sedative or tranquilizer to promote rest and relaxation may be prescribed in ulcer therapy. Discourage any visitors that agitate the patient until his/her condition improves.

Pain relief

Assess the patient's pain by asking him/her and observing for non-verbal signs of pain such as grimacing and rubbing the affected area.

Administer prescribed medication such panadol to relieve pain and promote comfort. Adminiser also other prescribed medication that help in ulcer healing. Advice the patient to avoid highly

spiced foods to alleviate pain and promote rest. Provide diversional therapy such as reading books watching TV to relieve pain.

Psychological care

This is a very important aspect of treatment because most of the ulcers are duodenal and may be associated with emotions. As such explain the condition to the patient and significant others in simple terms. The explanation should involve the disease process, parts affected in relation to signs and symptoms, causes, treatment and complications that may occur. This is to impart knowledge in the patient and allay anxiety. Advise the significant others to avoid alarming the patient by telling him bad news or involving him/her in serious decision making as this may cause more stress to the patient. Encourage patient to express his/her fears and concerns. Involve him in making plans about his /her care to gain cooperation.

Observations

Patient's general condition should be observed to determine whether it is improving or not. Establish a base line data of vital sign observations which will be compared to subsequent findings. Vital signs should include pulse and blood pressure which would be affected when there is haemorrhage, temperature and respirations also get affected in severe peptic ulcers. Observe patient for pain, site and the food which aggravate the pain. Observe patient for vomiting and the contents of vomitus and also the stool should be observed. Record and report the findings to the sister in charge. Observe patient's response to treatment and also observe for the side effects of the drugs. Monitor patient for any complications such as hemorrhage which will be manifested by dizziness, low BP, rapid and feeble pulse; pyloric obstruction – vomiting of undigested food, etc. Observe eating habit of the patient and help patient to identify foods that cause or relieve pain

Nutrition/Fluids

Nutrition also plays an important role in treatment of patient with peptic ulcers since it can either cause pain or reduce it. Foods which cause pain should be identified and avoided as well as the highly seasoned or gas forming foods. These foods may worsen patient's condition. Provide food that is well balanced that should include in proteins to promote healing of ulcers and roughage to prevent constipation that is common. Serve food in small frequent amounts to promote appetite and prevent vomiting. Fluids should be encouraged either orally or intravenously depending on the condition to prevent dehydration due to vomiting and they also help to neutralise the hydrochloric acid in the stomach.

Elimination

Observe intake and output and record to prevent fluid overload. Provide diet rich in roughage to prevent constipation. Observe for blood in stool and vomitus and notify the physician. Provide an emesis bowl for vomiting.

Exercises

Initially patient should be put on total bed rest as excessive exercises can stress the patient and stimulate the vagus nerve to produce more hydrochloric acid. As the condition improves, exercises should be introduced slowly as prolonged bed rest can cause complications like deep vein thrombosis.

Hygiene

Assist patient with bathing to promote comfort, promote blood circulation and to remove dirty. Do oral care to promote appetite and prevent halitosis. Change linen whenever necessary.

Patient teaching

- Instruct the patient to avoid precipitating factors such as caffeine drinks, alcohol ingestion.
- Assess the knowledge that the patient has on the management of the condition so as to establish the baseline data for patient teaching.
- Assess the patient's ability and willingness to learn and ensure understanding of the condition.
- Help the patient to identify stressors and initiate modification in the daily routine as stress causes hypersecretion of HCL and pepsin which can alter mucosal barrier.
- Discuss diet plan and assist with implementation at home and work setting.
- Teach warning signs and symptoms of recurrent ulcers or complications and the importance of early treatment.
- Explain harmful effects of smoking which directly irritates gastric mucosa.
- Explain the rational for elimination of alcohol, spicy foods, coffee, tea and cocacola from the diet.
- Provide written material on the condition.
- Advise patient the importance of taking prescribed medication as prescribed and teach on side effects of the drugs. Discourage patient to take unprescribed drugs such as aspirin
- Reinforce the need for follow up appointments.

Complications

Haemorrhage– It is the most common complication of peptic ulcer disease. It occurs when the ulcer erodes one of the blood vessels. Haemorrhage is manifested by haematemesis and melena. If bleeding is massive, the patient experiences weakness, apprehension, dizziness and faintness which may progress rapidly to prostration and loss of consciousness. The skin becomes pale, cold and clammy; the pulse is rapid and thready and the blood pressure is abnormally low. Rapid respirations manifest air hunger and hypoxemia. If a large vessel is eroded, the signs and symptoms appear more rapidly and collapse occurs quickly.

Perforation – Most considered lethal complication and is due to progressive erosion of the ulcers through the submucosal, muscular, and serous layer of the gastrointestinal wall. When the serous membranous layer is penetrated, some of the stomach or duodenal content escapes into the peritoneal cavity and causes a generalized peritonitis by chemical irritation and infection. Perforation has a higher incidence in duodenal ulceration and may occur in a few persons with no previous history of ulcer.

The patient experiences sudden, incapacitating abdominal pain that begins in the midepigastric region and spreads over the whole abdomen. The patient will have pallor, a cold clammy skin, rapid pulse, shallow grunting respirations and probably nausea and vomiting. The abdomen becomes tender, rigid and board like. The patient assumes a knee-chest position to decrease the tension on the abdominal muscles.

Perforation complicates to bacterial septicaemia, hypovolaemic shock, and paralytic ileus.

Peritonitis – this may result fromperforation through the peritoneal cavity.

Gastric outlet obstruction – This isas a result of oedema, inflammation as well as fibrous scar formation. Symptoms of gastric outlet obstruction include abdominal bloating, nausea, and vomiting. Persistent vomiting leads to alkalosis from loss of large quantities of acid gastric juice (hydrogen and chloride ions) in the vomitus. Hypokalaemia may also result from vomiting or metabolic alkalosis.

Malignancy- Malignancy may result from regeneration of epithelium caused by chronic ulceration. (MacSween and Whaley, 2001).

Penetration: an ulcer located along the posterior wall of the duodenum or stomach may perforate into contiguous structures such as the pancreas, liver, or biliary tree. Patient complains of a change in the intensity and rhythmicity of the ulcer symptoms. The pain becomes more severe and constant, may radiate to the back, and is unresponsive to antacids or food.

Activity
Write differences between gastric ulcers and duodenal ulcers
Well done. You are a fast leaner.

2.7Management Of A Patient With Intestinal Disorders

Malabsorption Syndrome

Definitions

Malabsorption is the inability of the digestive system toabsorb one or more of the major nutrients (Smeltzer etal 2010).

This a condition in which there is failure of the small intestines to absorb the products of digestion (Bloom, 2005)**.**

Malabsorption is a syndrome associated with a variety of disorders in which there is a disruption of digestion and nutrient absorption.

Aetiology and Classification

The fundamental basis of this syndrome is faulty absorption of one or more essential nutrients as a result of a large variety of diseases. Faulty absorption is commonly due to either faulty digestion, where nutrients are not changed into an absorbable nature, or because nutrients which have been digested are not being transported across the brush border of the villi.

Diseases that Cause Malabsorption

1. Coelic disease
2. Dermatitis herpertiform
3. Tropical sprue
4. Bacterial over growth
5. Whipple's disease (stunted growth of villi).
6. Short bowel syndrome
7. Lactase deficiency
8. Parasitic infestation e.g. Giardia intestinalis

Clinical Manifestations

A patient who is admitted to hospital with malabsorption may be in a state of starvation and therefore acutely ill. He/she will present with the following features:

1. Steatorrhoea: this is excretion in faeces of more than 7g of fat daily. The stools are characteristically pale, bulky, offensive, and difficult to flush down the toilet.
2. Wasting: due to failure of absorption of essential body building nutrients. Weight loss may not be evident by weighing because the patient may have ascites and/or oedema.
3. Abdominal distension, borborygmi (sounds of flatus in the abdomen)**,** flatulence, and abdominal discomfort due to increased bulk of intestinal contents and gas production.

The signs and symptoms of specific deficiencies commonly seen in malabsorption are:

1. Fatigue due to anaemia and/or hypokalaemia
2. Oedema due to hypoproteinaemia and/or anaemia
3. Tetany due to hypocalcaemia

4. Haemorrhage (in the form of ecchymosis, purpura) due to failure of absorption of vitamin K.
5. Anaemia due to iron deficiency
6. Decreased libido
7. Glossitis and stomatitis due to folic acid and vitamin B dificiencies
8. Osteoporosis, osteomalacia, and bone pain due to hypocalcaemia and vitamin D deficiency
9. Infections due to globulin deficiency
10. Pigmentation of skin and mucous membranes due to hypoadrenalism
11. Hypotension, hypothermia, and inhibition of growth due to depression of endocrine activity
12. Mental changes occur, particularly in gluten enteropathy.

Diagnosis

- Stool analysis for bacterial growth and parasites. For example isolating cysts of Giardia Lamblia.

Jejunal biopsy showing the mucosal appearance which may show malignancy, Villi atrophy. The biopsy can be obtained through jejunoscopy.

Radiological:-

- Ultra Sound scan, Computed Tomography and Magnetic Resonance Imaging
- Barium meal - small intestine is dilated, segmented and loss of the normal feathery appearance of jejunum
- Ultrasound scan, and can revealpancreatic or intestinal tumors.
- Acomplete blood cell countto rule out anemia.

Medical and Nursing Management

The principles of treatment are:

1. To treat or remove the primary cause, e.g. gluten-free diet in celiac disease, pancreatic extract by mouth in chronic pancreatic disease or antibiotic therapy in diverticulosis
2. Where specific treatment is not possible, such as following extensive resections of stomach or intestine, to try to overcome the malabsorption by increasing the intake of essential nutrients and kilojoules
3. To replace the specific deficiencies detected. Intravenous therapy is often necessary to achieve this.
4. To reduce the symptoms associated with steatorrhoea

Drugs

Drugs prescribed for the patient with malabsortion will be dictated by the underlying cause and any known deficiencies, e.g.:

- Anti-bacterials, example Metronidazole can be used

- Mode of action; it is bactericidal. It inhibits bacterial DNA synthesis thereby leading to death of the bacteria.
- Dose: 200 – 400mg TDS PO for 7 -10 days.
- Side; dry mucous membranes, metallic taste

Take Note:

Drugs are selected according to the cause to eliminate the causative organism

A. Diet:-

- Gluten free diet for celiac disease.
- Low fibre diets.
- Low fat as medium chain triglycerides.
- No irritant diet, for instance with a lot of spices.

B. Parenteral fluids and feeding with supplements of the deficient elements.

- Total parenteral nutrition via central venous line can be required.

Management of some individuals with malabsorption syndrome may require injections of vitamin B_{12} and oral iron supplements. The doctor may also prescribe enzymes to replace missing intestinal enzymes, or anti-spasmodics to reduce abdominal cramping and associated diarrhoea. People with cystic fibrosis and chronic pancreatitis require pancreatic supplements. Those with lactose intolerance or gluten enteropathy (non-tropical sprue) will have to modify their diets to avoid foods that they cannot properly digest.

If or when an oral diet is tolerated the patient is usually prescribed a high protein, low fat, high calorie diet with added nutritional supplements. A specific diet should be prescribed where applicable, e.g. a gluten-free diet for a patient with coelic disease. The nurse should help the patient understand the importance of taking the prescribed diet and ensure that the patient eats. A strict fluid balance chart and a food intake chart should be maintained.

Specific Nursing Management

The nurse should use a problem-solving approach such as nursing process when caring for the patient because of the multiple causes and the varying degrees of severity of the syndrome. The 3 main areas to be assessed are:

1. Problems associated with diarrhoea and steatorrhoea (incontinence, excoriation of the perianal area and fatigue)
2. Problems associated with ensuring that the patient receives adequate nourishment
3. Psychological status. Depression, grief and anger may be a response to the loss of normal good health.

The nurse should also provide full general nursing care to a very ill patient who is on complete rest in bed. Special attention should be paid to the mouth and pressure area care, as well as deep breathing exercises.

Crohn's Disease

Definitions

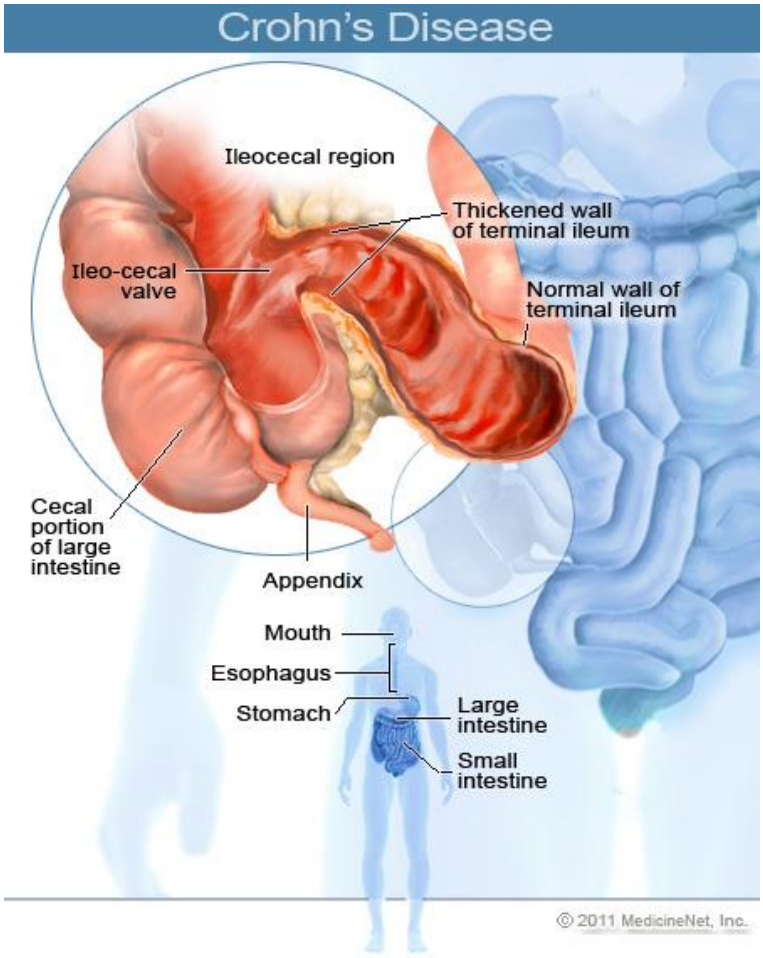
Crohn's disease is a chronic inflammatory disease of the gastrointestinal system that mainly affects the distal ileum and the colon. It may also affect any part of the gastrointestinal tract from the mouth to the anus (Bloom, 2005).

Crohn's disease is a chronic, nonspecific inflammatory bowel disorder of unknown origin that can affect any part of the gastro intestinal tract.

Crohn's disease is an idiopathic inflammatory process that can affect any portion of the alimentary tract from the mouth to the anus.

What do you think about this picture?

This picture shows some parts of the lower gastrointestinal tract and also the parts which are affected by chrohn's disease.



FigureTableTableFigure 6: Lower GIT parts-sites commonly affected by Crohn's disease

Epidemiology

Crohn's disease may occur at any age, but occur most often between the age of 25 and 30 years. Both sexes are affected with slightly high incidence in women.

One-third of cases involve only the small bowel, most commonly the terminal ileum (ileitis). About half of cases involve the small bowel and colon, most often the terminal ileum and adjacent proximal ascending colon (ileo-colitis). In 15-20% of cases, the colon alone is affected.

Causes

The exact cause of chrohn's disease is unknown but it is associated with the following:

- *Genetic factors* - it can run in families, genes that are inherited from parents may increase the risk of developing Crohn's disease
- *Immune disorders* - the inflammation may be caused by a problem with the immune system (the body's defence against infection and illness) that causes it to attack the normal flora of the gastrointestinal tract.

- *Previous infection* - a previous infection may trigger an abnormal response from the immune system. Smoking - smokers with Crohn's disease usually have more severe symptoms than non-smokers

Pathophysiology

Crohn's disease can affect any part of the GI tract, but is most often seen in the terminal ileum, jejunum and colon. The inflammation involves all layers of the bowel wall (i.e. transmural). Areas of involvements are usually not continuous with segments of normal bowel occurring between diseased portions. Typically ulcerations are deep and longitudinal and penetrate between islands of inflamed oedematous mucosa causing the classic cobblestone appearance. Thickening of bowel wall occurs as well as narrowing of the lumen with stricture development. Abscesses or fistula tract that communicates with other loops of bowel, skin, bladder, rectum or vagina may develop.

Clinical Manifestation: manifestations mainly depend on –

- Anatomical site of involvement
- Extent of the disease process
- Presence or absence of complications
- Crumpy abdominal pains due to inability of the intestines to transport products of the gastrointestinal tract through the constricted lumen.
- Chronic diarrhoea due to constant irritating discharge that is emptied into colon from weeping, swollen intestines. The diarrhoea may be accompanied by blood.
- Weight loss due to malabsorption and reduced intake of food.
- Fever due to infection
- Nausea and vomiting may indicate the beginning of small bowel obstruction

Constipation- while most patients have diarrhoea, some patients may have constipation which comes as a result of having inflammation on the anus, this makes it difficult to defecate.**Management**

Aims of management

1. To control the inflammatory process
2. To relieve symptoms
3. To correct metabolic and nutritional problems and promote healing

Diagnostic Studies/ investigations

- Proctosigmoidoscopy is usually performed initially to determinewhether the rectosigmoid area is inflamed
- Stool for occult blood
- Barium enema will reveal strictures. This test will also show fissures.

- Small bowel x-ray may show irregular mucosa, ulcerations and stiffening.
- Sigmoidoscopy and colonoscopy will show patchy areas of inflammation.
- Biopsy obtained during sigmoidoscopy will reveal granulomous tissue.
- Blood tests will show increased white blood cell count, reduced erythrocyte sedimentation rate (ESR) and reduced haemoglobin level.

Drug therapy

There is no specific therapy that exists but the following drugs can help:

1. Corticosteroids, example Prednisolone: Prednisolone 5-60mg daily PO given as single dose or in divided doses.

    Maximum daily dose is 250mg
    Mode of action; stimulates the synthesis of enzymes needed to decrease the inflammatory response
    - Side effects: suppresses the immune system by reducing activity and volume of the lymphatic system, thus producing lymphocytopenia

2. Aminosalicylates, example Sulfasalazine initial 3-4g PO daily in divided doses, then maintenance dose 2g PO daily in divided doses

    - Mode of action; unknown but believed to be metabolised by the intestinal flora in the colon thereby producing anti-inflammatory effects and antibacterial action.
    - Side effects; reduced appetite, stomatitis, vertigo

3. Antibiotics, example Metronidazole200 – 400mg TDS PO for 7 -10 days.

    - Mode of action; it is bactericidal. It inhibits bacterial DNA synthesis thereby leading to death of the bacteria.
    - Side; dry mucous membranes, metallic taste

4. Anti-diarrhoeals, example Loperamide 4mg stat PO, then 2mg per loose stool

    Maximum dose 16mg in 24 hours
    - Mode of action; reduces intestinal motility by acting directly on intestinal mucosal nerve endings leading anti-peristaltic action. May also inhibit fluid and electrolyte secretion by unknown mechanism.
    - Side effects; constipation, abdominal pain, dry mouth and drowsiness

5. Analgesics may be given such as Paracetamol 500- 1000mg tds po

    - Mode of action; It is a non-narcotic analgesic, anti-pyretic and anti-inflammatory
    - Side effects; Rare but may have Nausea, Vomiting, liver damage.

Other measures are;

- Vitamin supplements to compensate for the bowel's inability to absorb them.
- In severe disease, the patient should be nil orally and parenteral feeds given.
- Surgery may also be done in severe cases to
    - ✓ remove the affected part,
    - ✓ repair fistulas
    - ✓ Drain abscess.

Complications

- **Abscess:** the presence of a tender abdominal mass with fever and leukocytosis suggests an abscess. Patient should be given broad-spectrum antibiotics and, if malnourished, maintained on Total Parenteral Nutrition (TPN). Percutaneous drainage or surgery is usually required.
- **Obstruction:** Small bowel obstruction may develop secondary to active inflammation or chronic fibrotic structuring and is often acutely precipitated by dietary indiscretion..
- **Fistulas:** the majority of enteromesenteric and enteroenteric fistulas is asymptomatic
- **Perianal Disease:** patients with fissures, fistulas, and skin tags have perianal discomfort that is treated conservatively with sitz baths and cotton pads to absorb drainage. **Carcinoma:** patients with colonic Crohn's disease are at increased risk of developing colon carcinoma. Screening colonoscopy is recommended by some authorities.
- **Haemorrhage:** unlike ulcerative colitis, severe hemorrhage is unusual in Crohn's disease.
- **Malabsorption:** Malabsortion may arise from bacterial overgrowth in patients with enterocolonic fistulas, strictures and stasis, extensive jejunal inflammation, and prior surgical resections.

Nursing care

Environment

Nurse the patient in the acute bay for easy observations. Environment should be well ventilated to eliminate odours.

Rest

Promote rest by creating a quiet environment, do nursing activities collectively so as to reduce disturbances. Give prescribed analgesics to reduce pain.

Observations

Observe general condition of the patient to see whether patient is improving or not. Check vital signs frequently depending on the patient's condition which can be two hourly or four hourly. Observe intake and output of fluids both oral fluids and intravenous fluids to watch out on complications like renal failure. Observe the vomitus and stool for consistency, colour and amount. Monitor for side effects of the drugs.

Observe for complications of the disease like abdominal distension which may indicate intestinal obstruction.

Medication

Give prescribed medication like Metronidazole and watch for side effects.

Psychological care

Explain the disease process to the patient and the family so that they can have knowledge about the condition. Inform patient on the causes, signs and symptoms, and the treatment. Tell patient that the outcome of the treatment will depend on his/her compliance in taking the prescribed medication. Provide emotional support to the patient and family to allay anxiety and answer patient's questions faithfully so as to gain confidence. Establish rapport by being attentive, calm and confident.

Hygiene

Provide good patient hygiene like baths to promote blood circulation. Oral care to promote salivation, prevent halitosis and mouth infections and to improve the appetite. After each bowel movement, provide skin care.

Nutrition/fluids

Provide patient with a diet which is rich in proteins to repair worn out tissues and carbohydrates to provide energy and vitamins to boast the immunity.If the condition is serious feed patient parentally. Give patient food in small frequent amounts rather than larger amount of foods to prevent vomiting. Encourage patient to increase fluid intake.

Elimination

Provide bed pans in initial phase but as the patient improves nurse patient near the toilet.

Observe stool for consistency, amount, and odour.

Patient's teaching

- Teach patient on disease, signs and symptoms and complications.
- Teach patient on the importance of rest, explain that limiting physical activity helps to reduce intestinal motility and promote healing.
- Explain the importance of drug compliance
- Explain to patient the importance of coming for review on the given date or whenever necessary.
- Teach patient on the importance of having good nutrition which is well balanced.

Complications

- **Abscess:** the presence of a tender abdominal mass with fever and leukocytosis suggests an abscess. **Obstruction:** Small bowel obstruction may develop secondary to active inflammation or chronic fibrotic structuring and is often acutely precipitated by dietary indiscretion..
- **Fistulas:** the majority of enteromesenteric and enteroenteric fistulas is asymptomatic
- **Perianal Disease:** patients with fissures, fistulas, and skin tags have perianal discomfort that is treated conservatively with sitz baths and cotton pads to absorb drainage. **Carcinoma:** patients with colonic Crohn's disease are at increased risk of developing colon carcinoma. Screening colonoscopy is recommended by some authorities.
- **Haemorrhage:** unlike ulcerative colitis, severe hemorrhage is unusual in Crohn's disease.
- Intestinal obstruction due to adhesions and narrowing of the intestinal lumen
- **Malabsorption:** Malabsortion may arise from bacterial overgrowth in patients with enterocolonic fistulas, strictures and stasis, extensive jejunal inflammation, and prior surgical resections.
- **Malnutrition** due to Malabsorption
- **Perforation** due to inflammation complications

Dysentry

Definitions

Have you ever heard of this condition (dysentery) before? I'm sure you have and how can you define it then?

Dysentery is an acute or chronic disease of the large intestine,characterized by frequent passage of small, watery stools, often containing blood and mucus, accompanied by severe abdominal cramps.

Causes: the main causative organisms of dysentery are:

- Shigella species
- Enternal invasive E. coli
- External Hemorrhagic E. coli

- Yersinia enterocolitica
- Salmonella species
- Entamoeba hystolytica

Types of dysentery

There are two main types of dysentery:

- Bacillary dysentery or shigellosis
- Amoebic dysentery or amoebiasis

Bacillary Dysentery /Shigellosis

Bacillary dysentery is an acute inflammation and ulceration of large intestines characterized by small frequent bowel movements consisting of blood and mucous in stool.

Bacillary dysentery is caused by non-motile gram negative bacteria of the genus Shigella.

Causes

Shigella is in 4 strains:

1. Shigella flexneri
2. Shigella boydii
3. Shigella dysenteriae
4. Shigella sonnei

Predisposing factors

These are best summarized by the 6 F's which are Formites, Food, Feaces, Fingers, Fluids and Flies. If these are well taken care then the problem is solved. The predisposing factors include;

i. Poor feeding methods, example, use of dirty feeding bottles for infants, eating unboiled and improperly prepared foods.
ii. Poor personal hygiene especially hand hygiene(hand washing, long unkempt finger nails and so forth)
iii. Poor source, treatment and storage facilities for water to drink
iv. Poor sanitation - Rubbish pits or dumping sites; Sewerage lines.
v. Overcrowding

Epidemiology

It is most prevalent in unhygienic areas of the tropics, but, because it is easily spread, sporadic outbreaks are common in all parts of the world.

Baccillary dysentery occurs among confined populations, such as those in nursing homes, large institutions subject to overcrowding (Diseases, 1992).

Mode of transmission

Look at this picture carefully. What comes in your mind when you see such a picture?

The route of transmission of shigella is fecal oral route. The bacilli are excreted in feces and through poor sanitation and bad hygiene, food and water can then become contaminated. Flies also frequently cause contamination of food and are prevalent mode of spread of dysentery. Figure 8 illustrates the mode of transmission of dysentery.

FigureTableTableFigure 7:Mode of transmission in dysentery

Incubation period

The incubation period of shigella is 1 - 7 days.

Pathophysiology

When the bacillus enters the GIT, it invades the large intestine causing inflammation of the mucosa. This results in ulceration and bleeding of the mucosa. The stool becomes blood stained and mucoid. In the later stage, pus forms due to infection. Adjacent lymph nodes may be affected resulting into fever.

After going through the pathophysiology of bacillary dysentery, what do you think can be some of the signs and symptoms? Write them down in the following activity.

Activity
Write down the signs and symptoms of dysentery in your notebook

We hope your answers included the following signs and symptoms.

Signs and symptoms

- Sudden onset of sign s and symptoms.
- Fever which results from infection and inflammatory reaction.
- Signs and symptoms of dehydration such as loss of skin turgor, as a result of diarrhoea. Dehydration may or may not be present in this condition because patient passes small amount of stool, but it's the frequency which is increased.
- Abdominal discomfort: This may be due to irritation of the mucosal lining of the gastro-intestinal tract by the bacteria and usually it is an early symptom of dysentery.
- Nausea and Vomiting: This may be due to irritation of mucosal lining of the GIT (stomach).
- Colic abdominal pains: May be due to inflammatory reaction in the mucosal lining of the intestines.
- Bloody diarrhoea - This may be due to damage of the mucosal lining of the large intestines during inflammation. Damage to the mucosal lining may also cause damage to the capillaries.
- The passage of bloody diarrhoea is usually accompanied by Urgency and tenesmus. (Urgency is the urge to open bowels at very frequent intervals even if small amounts of stool are passed and tenesmus is a painful ineffective straining to empty the bowels.

Management

The aims of management are to:

1. Correct electrolyte and fluid imbalance
2. Eliminate the causative organism
3. Prevent and manage complications

Investigations

The main investigations include:

- Microscopic examination of a fresh stool specimen and a rectal swab for culture and sensitivity. Stool should be cultured within a few hours of collection. Detection of the organism in stool confirms diagnosis.
- Immunofluorescent techniques to detect organism in stool.
- Sigmoidoscopy reveals a red, bleeding mucosa with patches of necrotic membrane which may separate to leave ulcerated areas.

Therapy

Fluid and electrolyte replacement: oral rehydration is usually required to restore fluid and electrolyte imbalances. However, each patient should be assessed for the degree of dehydration and the appropriate fluid replacement therapy given.

Drugs: antibiotics are administered to shorten the duration of illness and prevent relapse. Any of the following are given while waiting for result of culture and sensitivity:

- Nalidixic acid 1g PO qid for 7 to 14 days
- Ciprofloxacin 500mg PO BD for 5 days
- Trimethoprin-Sulfamethoxazole (Septrin, Co-otrimoxazole) 960mg PO BD for 5 days
- Chloramphenicol 50 to 100mg/kg body weight qid for 5 days
- Ampicillin 500mg qid for 5 days

Amoebic Dysentery/Amoebiasis

This is the second type of dysentery. Amoebic dysentery or amoebiasis is an infection caused by a pathogenic amoeba *Entamoeba histolytica.* This is a chronic enteric infection caused by protozoa known as Entamoeba hystolytica (Billings and stokes, 1982).

Amoebiosis is an infection of the large intestines caused by Entamoeba hystolytica a single celled parasite (Berkow et al, 1997).

Cause

The cause of amoebic dysentery is entamoeba hystolytica.

Predisposing Factors

Same as for bacillary dysentery.

Epidemiology

Entamoeba histolytica has a worldwide distribution and is endemic in most countries with poor sanitation and low socioeconomic conditions. Use of nights soil for agricultural purposes favours the spread of the disease. The organism is acquired when cysts are ingested.

Mode of Transmission

Faecal-oral route, vectors such as flies, cockroaches and rodents are capable of carrying cysts and contaminating food and drink.

Incubation Period

It may take 2 weeks or years. Human beings are the principal reservoirs/carriers.

Pathophysiology

Ingested cysts enter the alimentary tract through the mouth to the stomach where they eccyst during digestion. Motile trophozoites are released which multiply. They then invade and ulcerate the intestinal mucosa of the large bowels. The ulcers they form are flask like.

Some of the amoeba goes through the mesenteric artery and reach the liver causing total destruction of the liver resulting in amoebic hepatocellular necrosis and then liver abscess.

Signs and Symptoms

The onset is gradual and associated with abdominal discomfort. Other signs and symptoms are:

- Mildly loose stools or frank diarrhoea with or without blood and mucus. Diarrhea may alternate with constipation.
- Tenderness may develop over the caecum, transverse colon or sigmoid
- Fever may be present
- Abdominal pains that may be on and off.
- If there is hepatic amoebiasis there would be body malaise, swinging temperature, sweating, and enlarged tender liver.
- Foul-smelly stool.
- Weight loss in chronic cases.

Investigations

The following tests are useful in making a diagnosis:

- Stool for m/c/s
- History of blood stained stool.
- Physical inspection will reveal dehydration.
- Rectal swab culture.
- Blood for Hb.
- Sigmoidoscopy will review ulcers.
- Liver scan will review Liver abscess.

Treatment

The disease is treated with the following:

- Flagyl 200-400mg tids
- Septrin 960mg bd x 5-7 days
- Furamide[diloxanide furoate] 500mg tds for 10 days
- For Herpatic Amoebiasis give Flagyl and Chloroquine 600mg od for 2 days and then 300mg od for 21 days.
- Panadol
- Intravenous fluids [Ringers Lactate)

Nursing care of dysentery

The aims of nursing care are to:

- prevent further spread of infection
- replace lost fluids and electrolytes

- prevent complications such as shock
- identify any contacts

Environment

Admit the patient in an isolation room away from other patients to prevent spread of infection to other people. The room should be well lit for easy observation and ventilated environment to promote air circulation. Patient should be nursed near the toilet for convenience. Equipment such as drip stands, intravenous set and observations tray should be within patient's environment.

Observation

Observe general condition of patient. Monitor vital signs such as temperature, pulse, respirations and blood pressure frequently. The frequency of vital sign observations depends on patient's condition. Observe the quality and amount of stool passed by patient. Monitor the intake and output and record on the fluid balance charts. Monitor stool for amount, consistency and colour and report.

Observe for any signs of dehydration such as loss of skin elasticity, sunken eyes, and thirsty and dry mucus membranes of the mouth.

Infection prevention

Isolate patient away from other patients to prevent spread of infection.

People who come in contact with this patient should observe isolation techniques such, putting on gowns and masks whenever they enter the room, washing hands before and after attending to the patient. Restrict visitors because they can also get the infection. The linen which is used by the patient should be disinfected with JIK 1:6 and should be labelled **"infectious**" before sending it to the laundry. It should not be mixed with other linen from the wards. Administer prescribed medication to treat the causative organism. All utensils used by patient should be disinfected.

Nutrition

Give some copious drinks and a light diet free from irritants.

If patient is unable to take food and fluids orally commence him/her on intravenous fluids. Maintain strict intake and output. Record intake and output, time commenced IVF, type of fluid and date started.

Hygiene

Assisted /bed bath can be given depending on the condition of the patient to promote comfort, self esteem and to remove dirty.

Assist the patient with oral care to prevent complications of a dirty mouth such as mouth infections and also promote salivation as the patient's mouth can be dry due to excessive loss of fluids. Change linen whenever soiled and disinfect the linen with Jik 1:6 before sending to the laundry. Ensure perineal area is cleaned.

Psychological care

Patients with dysentery may feel as if they have been neglected. The nurse needs to give proper psychological care to allay anxiety. Educate the disease process to patient which should include the cause, mode of transmission, signs and symptoms, treatment and complications. Explain to the patient the reason for isolation which is prevention of spread of infection. Explain also to the significant others on why they are not allowed to visit the patient. Any procedure which is done to patient should be explained to gain his/her cooperation. Allow patients to ask questions and answer them truthfully.

Medication

Administer prescribed drugs as prescribed and observe for side effects. Administer fluids according to patient' condition.

Elimination

Observe intake and output and record. Observe stool for amount, contents and odour. Provide bed pan in the initial stage but as condition improves, encourage patient to go to the toilet.

Prevention And Control Of Dysentery

We have discussed the mode of transmission and predisposing factors of dysentery. From our discussion, how can dysentery be prevented?

Dysentery can be prevented by doing the following measures:

Improved Environmental Sanitation: measures include:

- Provision of safe and adequate water supply.
- Safe and adequate disposal of human excreta through use of pit latrines or toilets
- Food safety against faecal contamination
- Provision of information, education and communication about dysentery.
- Discourage use of untreated human excreta for manure.

Early Diagnosis and Treatment of Cases and Carriers

- Prompt detection and appropriate and adequate treatment of both cases and carriers
- Regular screening of food handlers

Improved Personal and Communal Hygiene

- Adequate hand washing with soap under running water after using the toilet and before handling and eating food.
- Use of pit latrines or toilets for defaecation.
- Children should not be allowed to defaecate on the ground. Toilet training pots should be used and disinfected after use. Children's stools should be disposed off in the toilet or pit latrine
- Boil water for drinking and for washing vegetables and fruits.
- Avoid eating vegetable and fruits salads.

Complications of Dysentery

1. Perforation of the colon dure to ulceration
2. Peritonitis due to perforation
3. Rectal prolapse due to tenesmus
4. Hematogenous dissemination of the shigellas (rare) causing abscesses and meningitis
5. Acute, nonsuppurative arthritis involving large weight-bearing joints may occur during convalescence
6. Conjuctivitis, iritis and peripheral neuropathy (rare).
7. Haemolytic uraemic syndrome (7-10 days after the onset of disease).
8. Toxic megacolon
9. Hemiplegia
10. Encephalopathy
11. Septicaemia due to the presence of the pathogens
12. Hyponatraemia due to electeolyt imbalances
13. Reiter's syndrome
14. Liver abscess
15. Renal failure due to excessive dehydration

Cholera

Definition: Cholera is an acute infectious disease caused by **vibrio cholerae** characterized by copious rice water diarrhoea, vomiting, muscle cramps, severe dehydration and vascular collapses.

Incubation Period

From few hours to 5 days.

Transmission

Oral faecal route through ingestion of contaminated food and water.

Causative Organism

Cholera is caused by vibrio cholerae. There are two (2) bio-types namely:

1. Classical cholerae vibrio
2. Haemolytic el-tar vibrio

Characteristics Of Vibrio Cholerae

1. Comma shaped
2. Aerobic
3. Gram negative
4. Non spore forming
5. Motile organism (possess both flagella and somatic antigens)
6. Killed by heat at 55^{o}C for 15 minutes and by phenolic and hypochlorite disinfectants
7. Can survive aquatic environments for extended periods in a 'dormant state'

Epidemiology

Cholera is a disease of low socioeconomic groups living in unsanitary conditions with poor health services, unsafe water supply, inadequate or absent sewage disposal. It is common in Asia and Africa. Infection spread from contaminated feaces and water. Other portable foods include milk, cold cooked foods and unwashed fruits and vegetables.

Pathogenesis: The vibrio cholerae remain in the gut and does not penetrate into the blood stream. It adheres to the mucosa of the small intestines by both outer membrane protein and flagella adhesions. Vibrio cholerae produces enterotoxin that causes excessive fluid and electrolyte loss. Sodium Chloride absorption is inhibited and therefore excreted resulting in water, sodium chloride, and potassium and bicarbonate loss.

Immunity to both cholera toxin and bacterial surface antigens follows natural infection.

Signs and Symptoms

Evacuation phase

1. Mild to acute onset of diarrhoea which initially contains faecal matter but later becomes watery, with flecks of white described as rice water stool.
2. Projectile vomiting may be present
3. Severe dehydration due to diarrhoea and vomiting

Collapse phase

1. Muscle cramps due to loss of electrolytes
2. Metabolic acidosis indicated by signs of air hunger with deep sometimes rapid breathing.

3. Hypovolaemic shock due to vascular depletion leading to vascular collapse detected by hypotension, tachycardia, and pulse may be impalpable at the wrist, cold clammy skin, olyguria. If no intervention shock can complicate into acute renal failure and death

Recovery phase

Patient improves and the signs and symptoms decline

Diagnosis

- In epidemics, presumptive diagnosis is made on clinical presentation or epidemiological grounds. Example, a patient 5 years or older, who develops acute watery diarrhoea with or without vomiting residing in an area where cholera is likely to occur.
- Laboratory diagnosis
    1. Dark field microscopy of stool may show the characteristic darting movement of vibrio. Inhibition of movement by 01 antisera provide strong evidence
    2. Stool or vomitus or rectal swab for microscopy, culture and sensitivity confirms the diagnosis
    3. Immunoflorecent allows for rapid diagnosis

Management

Aims of management

1. To correct fluid and electrolyte imbalance
2. To eliminate the causative organism
3. To prevent the spread of infection
4. To prevent complications due to loss of fluid and electrolytes

Therapy

Correction of fluid and electrolyte imbalance: Start intravenous infusion of fluids such as Ringer's Lactate or normal saline immediately. (Give ORS while setting up the drip if patient is able to drink).

In severe dehydration, give 30mls/kg body weight in the first 30 minutes rapid I.V. then 70mls/kg body weight in the next 2½ hours. The aim is to restore normal hydration and acid-base balance within 2-3 hours. Continue rehydrating the patient at a slower rate until the pulse and BP return to normal. When the patient can drink orally, give 5mls/kg body weight/hour (250mls/hr).

Monitor vital signs every 30 minutes. If signs of circulatory overload are detected, slow down the rate of flow. Monitor urine output every hour (normal is 30-40mls/hy). If less patient has acute renal failure. Give ORS as soon as the patient can drink.

Maintain the patient on fluid by equal amount from stool losses. In this case oral rehydration is as required may be done.

Antimicrobial Agents

1. Tetracycline 500mg QIDfor three days
2. Doxycycline 300mg stat dose is a drug of choice
3. Cotrimoxazole 960 BD for three days
4. Ciprofloxacin 500mg BID for three days
5. Erythromycin 500mg QID for three days

Complications

- Paralytic ileus
- Muscle weakness
- Cardiac arrythymias
- Renal failure
- Metabolic acidosis
- Hypoglycaemia
- Pulmonary oedema

Prevention and control of cholera

- Proper disposal and treatment of the germ infected fecal waste (and all clothing and bedding that come in contact with it) produced by cholera victim is of primary importance
- Sewage: treatment of general sewage before it enters the waterways or underground water supplies prevent possible undetected patients from spreading the disease
- Sources: warnings about cholera contamination posted around contaminated water sources with directions on how to decontaminate the water
- Sterilization: boiling, filtering, and chlorination of water kill the bacteria produced by cholera patients and prevent infections, when they do occur, from spreading. All materials (clothing, bedding, etc.) that come in contact with cholera patients should be sterilized in hot water using (if possible) chlorine bleach. Hands etc. that touch cholera patients or their clothing etc. should be thoroughly cleaned and sterilized. All water used for drinking, washing or cooking should be sterilized by boiling or chlorination in any area where cholera may be present.
- Improve water supply and sanitation
- Contact tracing
- Personal hygiene
- Postpone festivals and gatherings
- Change of attitudes/behaviours e.g. wash hands, boil water, heat food before eating, use toilet or latrine
- Adequate treatment of cases
- Active reporting of suspected cases in areas previously uninfected (notification).

Typhoid Fever /Salmonella Infection

Definition

Typhoid fever is a systemic infectious (enteric fever) disease caused by salmonella typhi characterized by lessions in the payer's patches,mesentery and spleen with a high remittent fever, malaise, and headache and abdominal pain.

Causative Organism

A bacterium called Salmonella typhi. The organism is readily killed by drying, pasteurization, and common disinfectants.

Reservoir of infection

Man is the only reservoir of infection. Both cases and carriers are infectious as long as the bacilli appear in stools or urine. Carriers may be temporary (incubatory, convalescent) or chronic. Convalescent carriers excrete the bacilli for 6 to 8 weeks.

Source of infection

The primary sources of infection are faeces and urine of cases or carriers; the secondary sources are contaminated water, food, fingers and flies.

Environmental and Social Factors

Incidence of typhoid rises during the rainy season. During this period, there is also an increase in fly population. Outside the human body, the bacilli are found in water, ice, food, milk and soil for varying periods of time. Typhoid bacilli do not multiply in water; many of them die within 48 hours to 7 days. The bacilli may survive for over a month in ice and icecream, up to 70 days in soil irrigated with sewage under moist conditions, and about 30 days or more in dry conditions. The bacilli multiply and survive in food especially milk. Vegetables grown in sewage farms or washed in contaminated water are a health hazard. Social factors such as contaminated water supply, open air defecation and urination, low standards of food and personal hygiene influence the disease.

Incubation Period

Usually 10 to 14 days. Depending on the dose of the bacilli it may take as short as 3 days and as long as 21 days.

Mode of transmission

Oral faecal route through ingestion of contaminated water and food especially raw fruit and vegetables.

Pathogenesis

Infection occurs through ingestion of the organism which rapidly penetrates the intestinal mucosa and multiplies in the lumen for a short period and stools can be cultured positive during the 1st 4 days of the incubation period.

From the mucosa, the organisms travel to mesenteric lymph nodes. After a brief period of multiplication here, the organisms enter the blood stream via the thoracic duct (transient primary bacteraemia) and are transported to the liver and spleen. After a period of further multiplication at these sites, huge numbers of organisms enter the blood stream, making the onset of clinical illness (secondary bacteraemia). During this secondary bacteraemia, which continues for the greater part of the illness, very few organs escape invasion but the involvement of the gall bladder and peyer's patches in the lower small intestine have clinical importance. The gall bladder is probably infected via the liver and the resultant cholecystitis is usually sub-clinical. The infected bile renders stool cultures positive. Pre-existing gall bladder disease predisposes to chronic bile infection, leading to chronic faecal carriage.

Invasion of the peyer's patches occurs either during the primary intestinal infection or during the secondary bacteraemia, and further seeding occurs through infected bile. The peyer's patches become hyperplastic, with infiltration of chronic inflammatory cells. Later necrosis of the superficial layer leads to formation of irregular, ovoid ulcers along the long axis of the gut, so that stricture formation does not occur after healing. When an ulcer erodes into a blood vessel, severe haemorrhage results and transmural perforation leads to peritonitis.

Clinical Manifestations of Typhoid Fever

The course of untreated typhoid fever is divided into four individual stages, each lasting approximately one week. The symptoms may be mild or severe.

Week 1:

Features are non-specific in the first week. The onset is insidious in adults, but may be abrupt in children.

- The early symptoms are severe headache, malaise, anorexia, body pains, and epistaxis.
- The temperature rises in a step ladder fashion (remittent fever) of about 40^{o}C by the end of the first week.
- A mild, non-productive cough (bronchitis)
- Constipation
- Usually enlarged and tender spleen causing abdominal pain

Week 2:

- Patient lies prostrate, looks toxic with sustained (continuous) high temperature of 40^{o}C.
- Patient may become confused and disoriented with hallucinations
- Slight abdominal distension and tenderness in the right lower quadrant.
- Patient becomes delirious, and sometimes agitated hence the nick name "nervous fever".

- Rose spots (crops of 2mm – 4mm diameter, pink papules) that fade on pressure, develop on the upper abdomen and lower chest, between the 7^{th} and 12^{th} days. They are difficult to detect in dark-skinned individuals. The spots are caused by bacterial embolization and rose spot cultures may be positive.
- Relative bradycardia, a pulse lower than anticipated in a febrile patient.

Week 3:

- Patient becomes more toxic and ill
- Continuous high fever persists
- Delirium continues
- Abdominal distension becomes more pronounced, with scanty bowel sounds.
- Crackles may develop over the lung bases
- Diarrhoea (6 – 8 stools in a day), green, foul smell, comparable to pea soup.
- Hepatosplenomegaly and elevation of liver transaminases
- Considerable weight loss
- Widal reaction is strongly positive with antiO and antiH antibodies.
- Blood cultures are sometimes still positive at this stage.

A number of complications can occur in the third week:

- Intestinal haemorrhage due to bleeding in congested Peyer's parches; this can be very serious and usually not fatal.
- Intestinal perforation in the distal ileum: this is a very serious complication and is frequently fatal. It may occur without alarming symptoms until septicaemia or diffuse peritonitis sets in.
- Encephalitis
- Neuropsychiatric symptoms (described as "muttering delirium" or "coma vigil"), with picking at beddings or imaginary objects.
- Metastatic abscesses, cholecystitis, endocarditis and osteitis
- Dehydration develops and patient is still delirious (typhoid state)/
- By the end of third week, the fever starts to reduce.

Week 4:

In patients who survive:

- The fever, mental state and abdominal distention slowly improve over a few days but intestinal complications may still occur.
- Convalescence is usually a slow process

Prognosis:

Prognosis is good with early diagnosis and/or appropriate treatment

Management Of A Patient With Typhoid Fever

Investigations

- Full Blood Count will show mild leuchocytosis, leucopenia and neutropenia, normocytic anaemia, mild thrombocytopenia
- Elevated serum transaminases and mild proteinuria
- Blood and bone marrow culture will isolate causative organism (definitive diagnosis)
- Faecal and urine cultures
- Faecal cultures are usually positive in the first week (presumptive evidence)
- Urine cultures are positive less often
- Serology: Widal test will be positive. Widal reaction detects antibodies to the causative organism but has limitations in that patients with previous immunization to typhoid fever and those who had other salmonella infection will have a positive widal test
- Detection of IgG and IgM antibodies

Drug Therapy

Treatment of Cases

1. Antibiotics e.g.
    - Ciprofloxacin tab 750mg orally BD for 14 days. If patient is vomiting persistently, 400mg I.V. 12 hourly for 7 days or
    - Amoxycillin cap 500mg orally 8 hourly for 2 weeks, or
    - Co-trimoxazole tab 960mg orally BD for 1 week, or
    - Azithromycin cap 500mg orally as a single dose followed by 250mg daily on day 2 to day 7

2. Antipyretic drug e.g

Paracetamol 1g tds for 5 days

3. Fluid and Electrolyte replacement therapy according to the level of dehydration.

Prevention and control of typhoid fever

There generally three lines of defence against typhoid fever:

1. **Control of Reservoir/cases**
    - **Early diagnosis**: early symptoms are non-specific. Culture of blood and stools are important investigations in the diagnosis of cases
    - **Notification:** this should be done following the notification procedure
    - **Isolation**: since typhoid fever is infectious and has a prolonged course, enteric precautions should be observed. Patients should be admitted to a health facility and isolated until three bacteriologically negative stools and urine reports are obtained on three separate days.
    - **Treatment**: appropriate and effective treatment should be administered.
    - **Disinfection**: stool and urine are the sole sources of infection. They should be received in closed containers and disinfected using the recommended disinfectant. All soiled clothes and linen should be soaked in a recommended disinfectant. Hand

washing should be observed by all who get in contact with the patient and contaminated items.

- **Follow up**: follow up examination of stools and urine should be done for S. typhi 3 to 4 months after discharge of the patient, and again after 12 months to prevent the development of the carrier state.

Carriers

- **Identification:** carriers are identified by cultural and serological examinations. Duodenal drainage establishes the presence of salmonella in the biliary tract in carriers.
- **Treatment:** the carrier should be given an intensive course of effective drug therapy.
- **Surveillance:** carriers should be kept under surveillance. They should be prevented from handling food, milk or water for others.

- **Information, Education and Communication:** regarding hand washing with soap after defecation or urination and before preparing food, use of toilets/pit latrines, control of fly population, terminal disinfection of all contaminated articles.

2. Control of Sanitation

- Protection and purification of water supplies, Improvement of basic sanitation, Promotion of food hygiene are essential measures to interrupt transmission of typhoid fever.

3. **Mass Immunization with Typhoid Vaccine**
    - Those living in endemic areas
    - Household contacts
    - Grip[s at risk of infection such as school children and hospital staff
    - Travelers proceeding to endemic areas,

Complications of typhoid fever

1. Intestinal haemorrhage: This occurs when the sloughs overlaying the peyer's patches separate during the late second or early 3rd week of the illness.

Signs and Symptoms

- Sharp fall in body temperature and blood pressure, and sudden tachycardia.
- Passage of bright red blood per rectum but may be absent in the presence of paralytic ileus.

Management: Sedation and blood transfusion

3. **Intestinal perforation**: also occur when the sloughs overlaying the peyer's patches separate during the late second or early 3rd week of the illness.

Signs and Symptoms

- Recognition of perforation is difficult.
- Usually pain and tenderness worsens

- Pulse rate rises
- Body temperature falls suddenly
- Discovery of free fluid in the abdomen
- Demonstration of gas under the diaphragm

1. Complications in the liver, gallbladder and pancreas

- Hepatitis
- Cholangitis
- Cholecystitis
- Pancreatitis

2. **Cardiorespiratory complications**

- Toxic myocarditis (tachycardia, weak pulse and heart sounds, hypotension, ECG abnormalities
- Mild bronchitis
- Bronch-pneumonia and lobar consolidation

3. **Complications in the nervous system**

- Toxic confusional state (disorientation, delirium and restlessness)
- Facial twitching or convulsions
- Paranoid psychosis or catatonia
- Meningitis
- Encephalomyelitis

4. **Haematological and renal complications**
    - Subclinical disseminated intravascular coagulation
    - Haemolysis
    - Immune complex glomerulitis
    - Nephritic syndrome
5. **Musculoskeletal complications**
    - Arthritis
    - Osteomyelitis

Paratyphoid Fever

This is a similar to typhoid fever but it is less severe disease. It is caused by salmonella paratyphi types A, B or C. Pathological and clinical conditions are very similar to typhoid fever, though they may produce only acute gastroenteritis.

Nursing care Of Typhoid And Paratyphoid

<u>Nursing care:</u>

Close monitoring of vital signs to detect abnormalities promptly

- record temperature, pulse, blood pressure and respirations 4 hourly, but more frequently if they are severely abnormal

- report abnormalities to nurse in charge/doctor
- avoid giving aspirin due to gastric irritation
- monitor for signs of intestinal bleeding i.e. malena, hypotension

Enteric isolation to prevent cross infection

- careful hand washing after each contact with the patient
- gloves should be worn when handling faeces, vomit, soiled bed linen
- careful disposal of all excretions and rubbish from the patient is essential
- disinfect bed pans/vomit bowls thoroughly
- thoroughly clean all articles used by the patient
- careful disposal of linen
- provide hand washing facilities for the patient

Careful monitoring of fluid balance

- record all input and output on fluid balance chart
- intravenous infusion may be necessary to maintain hydration
- encourage oral fluids if able

Relieve abdominal pain to aid patient comfort

- give prescribed analgesia e.g. Pethidine/morphine for severe abdominal pain
- monitor effectiveness of analgesia by asking the patient, liase with medical staff if analgesia not relieving pain
- assist with positioning comfortably

Meet hygiene needs to maintain good skin integrity

- assist with bed bath
- ensure anal region kept clean if experiencing diarrhoea to prevent skin breakdown
- mouth care to reduce the risk of oral infections and dry mouth
- pressure area care to prevent skin breakdown
- change bed linen as required ensuring careful disposal of soiled linen
- provide hand washing facilities for the patient

Provide for elimination needs

- position patient near to toilet or keep bed pan readily available
- record stool type on chart and keep medical staff informed
- ensure privacy when using bed pan
- nurse on a plastic and draw sheet if experiencing diarrhoea

- provide hand washing facilities

- *Provide diet and fluids to meet nutritional needs*
- give a high calorie, low fibre diet, a soft or liquid diet will be more easily managed

2.8 Management Of A Patient With Hepatic Disorders

Review Of The Anatomy And Physiology Of The Liver And The Biliary System

The Liver

The liver is the largest organ of the body situated in the upper abdominal cavity immediately below the diaphragm. It is divided into four lobes and is highly vascular, receiving its blood supply from two sources. The portal vein carries blood from the stomach, intestines, spleen and pancreas into the liver. The hepatic artery delivers blood from the aorta. The blood from both sources leaves the liver by a common pathway, the hepatic vein, which joins the inferior vena cava.

The liver tissue is organized in functional units called lobules. Each lobule consists of rows of cells radiating out from a central vein. Subdivisions of the hepatic artery and portal vein deliver blood into small spaces called sinusoids between the rows of cells, bringing the blood in direct contact with the hepatic cells. From the sinusoids it enters the central vein. Large phagocystic, reticuloendothelial cells called Kupffer cells lie scattered within the sinusoids to ingest and destroy organisms and other foreign material within the blood. The central veins from the lobules empty into sub lobular collecting vein, which unite to form the hepatic vein.Minute ducts into which bile is discharged are also formed between the rows of hepatic cells. The small lobular bile ducts are directed toward the surface of the lobules where they unite to form larger ducts. Eventually, the bile from the lobules is transmitted in one main channel, the hepatic duct, which joins the bile duct from the gallbladder (cystic duct) to form the common bile duct.

Manifestations Of Impaired Liver Function

Jaundice (icterus): This is an indication of an excess of bilirubin in the blood, resulting in a yellowish staining of the tissues that may be seen in the sclera, mucous membranes and skin. The cause may be intrahepatic or extrahepatic disease, and according to the cause, the jaundice may be classified as hepatocellular, obstructive or haemolytic.

The hepatocellular type of jaundice is associated with intrinsic liver disease and is due to failure of the hepatic cells to take up the bilirubin resulting from the breakdown of red blood cells and excrete it as bile. Jaundice may not be present in chronic liver disease (e.g., cirrhosis), especially in the early stages, since regeneration of hepatic cells may parallel the damage.

Obstructive Jaundice is caused by an interference with the flow of bile in the extrahepatic ducts. It is most often due to the impaction of gallstones in the common bile duct, but may occur as the result of a stricture in the duct or neoplastic disease in neighboring structures (e.g., pancreas) compressing the duct.

Haemolytic Jaundice occurs when there is an excessive destruction of red blood cell, resulting in excessive bilirubin formation.

The jaundiced patient's urine is likely to be dark because of the bilirubin or urobilinogen content. Urobilinogen is not present in obstructive jaundice, since it is formed in the intestine. The stools are pale gray in obstructive and severe hepatocellular jaundice.

***Pruritus*:** The itching of the skin experienced by many patients with jaundice is attributed to irritation of the cutaneous sensory nerves by the retained bile salts

Hepatitis

Definition

Hepatitis is an acute or chronic inflammation of the liver (Lewis et al 2011).

Hepatitis is inflammation and injury to the liver due to viruses, bacteria, toxic substances and infections.

Classifications

It is usually classified according to the cause and duration i.e.:

- Toxic hepatitis
- Auto immune hepatitis
- Drug induced hepatitis
- Hepatitis A
- Hepatitis B
- Hepatitis C
- Hepatitis D
- Hepatitis E
- However the commonest forms of viral hepatitis are Hepatitis A. B, and C
- It may also be called acute or chronic

HEPATITIS A (HAV)

- Hepatitis A is a contagious viral infection caused by Hepatitis A virus (an RNA picornavirus).

Mode Of Transmission

1. Fecal-oral (fecal contamination and oral ingestion)

2. Spread by direct contact

3. Ingestion of contaminated food, milk, or water. Food preparers who are infected can pass the virus on if they do not wash their hands with soap and water after having a bowel movement, especially when they prepare uncooked foods.

4. May spread parentally though rare.

Prevalence

More prevalent in underdeveloped countries and overcrowded poorly sanitized conditions.

Incubation Period

Incubation period is 2 – 6 weeks.

Pathophysiology

Hepatitis virus invades the liver cells and destroys it after reproducing. The body attacks the HV with antibodies. This leads to the destruction, regeneration and resuming of normal function of the liver. A person who recovers remains protected for life.

Signs and symptoms

It is possible to experience mild or no symptoms whatsoever, but even if this is the case the person's faeces will still be infectious to others. Many people who become infected with HAV will have symptoms that include

- A short, mild, flu-like illness.
- Nausea, vomiting and diarrhoea.
- Loss of appetite.
- Weight loss.
- Fever
- Jaundice (yellow skin and whites of eyes)
- Darker yellow urine
- Pale faeces (clay coloured stool).
- Itchy skin.
- Abdominal pain especially around the right hypochondriac region

- Diarrhoea
- The infection usually clears up in to 2 months, but may occasionally recur or persist longer in some people.

Diagnosis

- Obtain a thorough patient history to assess the likelihood of hepatitis A infection.
- Use clinical evaluation to support the diagnosis of hepatitis A.
- Confirm the diagnosis of hepatitis A with appropriate laboratory testing. IgM anti body to HAV.
- Use serologic testing to exclude HAV infection in persons with unexplained acute liver failure.
- Consider the presence of other liver diseases in persons with unexplained acute hepatitis. Blood to r/o hepatitis B and C
- liver function (laboratory evaluation of: urine bilirubin and urobilinogen, total and direct serum bilirubin, ALT and/or AST, alkaline phosphatase, prothrombin time, total protein, albumin, IgG, , IgA, IgM.
- Liver scan will show change in the liver
- Stool for electro microscopy
- Urine for urobilinogen

Treatment

- No specific treatment is necessary for hepatitis A. Disease
- IV fluids to prevent dehydrated, 5% Dextrose1litre/24hours.
- Anti-emetics to counteract nausea nausea and vomiting, e.g. phenegan 10mg bd
- Anti-histamine to control itching phenegan 5-10mg
- Enough rest to promote recovery
- Glucose to promote rest of the liver there by promoting recovery
- Vitamin B complex
- Vitamin A 10mg im
- Prednisolone 30mg od for 14/7 then reduce till you stop
- Analgesics for pain e.g. panadol 500mg tds for 3/7

Prevention

Advise the community people to have:

- Clean water supply
- Good sanitation
- Good personal hygiene
- Vaccination against hepatitis A .
- Prophylaxis with anti-hepatitis immunal globulin.
- Avoiding overcrowding.

HEPATITIS B (HBV)

Definition

Hepatitis B is an inflammatory liver disease caused by the hepatitis B virus (HBV) which is a DNA hepadevirus that results in liver cell damage.This damage can lead to scarring of the liver (cirrhosis) and increased risk of liver cancer in some people.

Mode of transmission

Hepatitis B can be spread in the following ways:

- By unprotected (without a condom) penetrative sex with someone who is infectious.
- By sharing contaminated needles or other drug-injecting equipment.
- By using non-sterilised equipment for tattooing and body piercing.
- From an infected mother to her baby, most commonly during delivery. Immunisation of the baby at birth prevents the transmission of hepatitis B.
- Through receiving blood (blood transfusion) which is infected with hepatitis B virus.

Incubation Period

Incubation period is 1 – 6 months

Phases of infection

Hepatitis B infection has 2 phases: acute and chronic.

- Acute (new, short-term) hepatitis B occurs shortly after exposure to the virus. A small number of people develop a very severe, life-threatening form of acute hepatitis called fulminant hepatitis.
- Chronic (on-going, long-term) hepatitis B is an infection with HBV that lasts longer than 6 months. Once the infection becomes chronic, it may never go away completely.

About 90-95% of people who are infected are able to fight off the virus before it becomes chronic.

Only about 5-10 percent of adults infected with Hepatitis B develop chronic infection.

Pathophysiology

- Viral hepatitis causes diffuse inflammatory infiltration of the hepatic tissue with mononuclear cells, spotty or singular necrosis. The liver may be swollen. Inflammation and regeneration occur simultaneously distorting the normal lobular pattern and creating pressure within and around the portal vein and obstruction the normal bile channel. The pathological change in the hepatocytes is not always related to the effect of the virus itself but rather the injurious response of the body's own immune system attempting to clear out the virus. The changes are associated with impaired liver functions.

  Signs and symptoms

  We have discussed the pathophysiology of Hepatitis B infection, now let us look at the signs and symptoms.

Half of all people infected with the hepatitis B virus have no symptoms. Symptoms develop within 30-180 days of exposure to the virus. The symptoms are often compared flu like. Most people think they have flu and never think about having HBV infection. The signs and symptoms include:

- Loss of Appetite
- Fatigue (Feeling tired)
- Nausea and vomiting
- Itching all over the body
- Pain over the liver (on the right side of the abdomen, under the lower rib cage)
- Fever
- Jaundice - A condition in which the skin and the whites of the eyes turn yellow in colour
- Dark urine
- Pale stools
- Diarrheal especially in acute hepatitis.

Treatment

- Usually acute hepatitis is self-limiting (it may not require medical treatment).
- Fluids to replace lost ones and electrolytes may be required if symptoms like diarrhoea and vomiting exist. 2000mls of Ringers Lactate may be required in 24 hours.
- Glucose to promote rest of the liver there by promoting recovery
- Vitamin B complex.
- Lamivudine and alpha interferon can be given in chronic cases.
- Vitamin A 10mg .

Prednisolone 30mg OD for 14/7.

Anti-emetics such as phernegan.

Prevention

Avoid contact with body fluids by:

- Using condoms during sexual intercourse. Do not share needles.
- Avoiding sharing instruments such as razor blades, needles and teeth brushes.

- Wearing gloves whenever coming in contact with body fluids.
- Vaccination with hepatitis B vaccine.

HEPATITIS C (HCV)

Definition

It is the inflammation of the liver that is caused by Hepatitis C virus (RNA virus).

Mode of transmission

- Exposure to blood or blood product, such as using needle stick used by infected person, blood transfusion,
- Un protected sexual intercourse with infected person.
- Faecal-oral transmission routes possible

Incubation period

Incubation period takes 6 – 7 weeks.

Signs and symptoms

- At the beginning of an hepatitis C virus infection, only about 25% of patients exhibit the characteristic symptoms of acute (rapid onset) hepatitis.
- These symptoms include;
- fatigue,
- muscular aches
- Anorexia
- Low-grade fever.

Diagnosis, treatment and prevention

As for hepatitis B

TOXIC HEPATITIS

Definition

Toxic hepatitis is the inflammation of the liver caused by toxic agent.

Treatment

- Identify and remove the damaging agent e.g. by gastric lavage.
- Where there is a known treatment for the poison it may be given.
- For instance for Acetaminophen over, Acetyl cysteine will be given.

DRUG INDUCED AND tOXIC hEPATITIS

Definition

Toxic hepatitis is the inflammation of the liver caused by toxic agent.

The toxic agent may include:

- Drugs such as isoniazide
- Chemical such as Carbon tetrachloride, phosphorus and chloroform.
- Alcohol
- Industrial toxins such as pollutants.
- Plant poisons such as certain mushrooms.

Signs and symptoms

- Anorexia, nausea, and vomiting are the usual symptoms.
- Jaundice and hepatomegally are noted on physical examination, and
- dark urine
- Symptoms are more intense for the severely poisoned patients.

There may also be

- fever,
- petechiae (hemorrhage under the skin) due to severe clotting abnormalities,
- rash, pruritis,
- arthralgia,
- delirium,
- convulsions and coma.

Diagnosis

- History from patient will confirm exposure to causative agent e.g. chemicals.
- Blood test for plasma protein to determine liver function.
- Blood test for liver enzymes which are elevated in liver cell damage. These are aspirate aminotransferase and alanine transferase.
- Liver scan may show enlargement and damage.

- Thrombin time: reflects thrombin activity- it is prolonged due to coagulation inadequacy in decreased thrombin activity, more than 12 seconds.
- Blood Bilirubin level: high conjugated Bilirubin in obstructive jaundice.

Treatment

- Recovery from acute toxic hepatitis is rapid if the hepatotoxin is identified early and removed, or if exposure to the agent has been limited. If there is prolonged period between exposure and onset of symptoms, recovery is unlikely. There are no effective antidotes. Therefore, there is need to immediately withdraw or stop the causative agent.
- Restore and maintain fluid and electrolyte balance by giving intravenous and oral fluids.
- Vitamin K administration to increase prothrombin time to promote coagulation.
- Blood transfusion may be done,

FULMINANT HEPATITIS/ACUTE LIVER FAILURE

Definition

It is a severe hepatic failure with development of hepatic encephalopathy within eight weeks due to any cause. It is characterized by massive hepatocellular injury and necrosis, leading to dramatic and rapid clinical deterioration .70% are due to acute viral hepatitis. Other causes are drugs, shock, malignancy (commonly lymphoma).

Pathophysiology

Following the introduction of a causative agent into the body, such as poisons, there is acute inflammatory reaction with hepatic vascular occlusion, leading to ischaemia and hypoxia of hepatic tissue. Ischaemia will lead to progression of hepatocellular injury and necrosis with development of hepatic encephalopathy. Mortality rate is extremely high (60% to 85%), despite intensive treatment.

Signs and symptoms

Jaundice due to accumulation of bilirubin.

- Urine which becomes frothy when shaken.
- Pruritis caused by bile salts deposited on the skin,
- Steatorrhoea and diarrhoea due to poor absorption of food,
- Peripheral oedema caused by hypoproteinaemia that lowers the oncotic pressure, resulting in fluids moving from intravascular to interstitial spaces.
- Ascitis due to portal circulation hypertension.
- Bleeding tendencies due to coagulopathy from protein deficiency,
- Irritability, confusion, coma as the level of consciousness alters.
- Hepatic coma/hepatic encephalopathy.
- Fever.

Diagnosis

- Liver function tests
- EEG for encephalopathy
- Toxicology
- Viral markers, autoantibodies, serum and urinary copper,
- Abdominal ultrasound.

Treatment

- Administration of potassium-sparing diuretic such as spinorolactone 100mg per day orally, to reduce the oedema but conserve potassium for cell metabolism.
- Manage cerebral oedema by giving mannitol.
- Vitamin A supplement, vitamin B complex 1 tablet daily, vitamins C and K, to improve integrity of the mucous membranes of the gastrointestinal tract, increase prothrombin levels for good coagulation of blood.
- Antibiotics such as amoxicillin to treat any bacterial infections that may be suspected.
- Abdominal paracentesis is performed; in the presence of Ascitis.
- Anti-acids administration and H2 -receptor antagonists, such as magnesium tricylicate to reduce the risk of bleeding from stress ulcers.
- Liver transplantation is the treatment of choice where available.
    - Steroids such as prednisolane
    - 10% dextrose

Nursing care of patient with hepatitis

AIMS:

- To prevent spread of the infection
- To promote optimal functioning of the liver and prevent complications
- Improve patient's knowledge about the condition

Environment

Nurse patient in isolation to prevent spread of infection. The environment should be well ventilated to prevent other respiratory tract infections as dust harbour organisms and may cause irritation to the respiratory passage. Patient should be nursed in a well lit room for easy observation and for orientation to time and place. Ensure all necessary equipment are within reach for use if needed.

Position

Nurse patient in in fowler's position to promote lung expansion and relieve dyspnea. Change patient's position two hourly to prevent development of pressure sores. As the condition improves let the patient adopt any position of comfort to promote rest.

Rest

Nurse patient in noise free environment to promote rest. Do related procedures collectively to avoid disturbing the patient during her periods of rest. Administer prescribed analgesics if patient is in pain to relieve pain thereby promoting rest.

Ensure that squeaking trolleys are oiled to prevent noise and there by promote rest.

Observations

Do vital signs such as temperature, pulse, Blood pressure and respirations to act as base line data and compare with subsequent ones. This helps to know if the condition is improving or deteriorating. Observe for oedema if improving or getting worse and elevate the foot end of the bed to promote venous drainage. Observe for the itching if present and offer ant histamines. Assess the pressure area to detect on set of pressure sore. Observe the stool and urine for colour and note any improvement towards normal.

Take Note 2.3

The patient's facial expressions to detect pain and administer prescribed analgesics like panadol. Observe the feeding pattern of my patient and take measures like giving small frequent meals to promote appetite. Observe also the mental status of the patient.

Psychological care

Explain the disease process in order to raise the knowledge levels and thereby alley anxiety. Encourage patient to ask question and answer accordingly and if unable refer to other health team members such as in charge or physician. Explain all procedures to the patient in order to allay anxiety. Involve a successfully managed case to come and talk to the patient in order to raise hope. This will also expel any misconceptions and instil a sense of hope. Explain the reason for isolation to alley anxiety. Provide diversional therapy in order to shift the patient's mind from the hospital routine and his condition. Involve patient in planning his/her own care to promote self-esteem and gain cooperation.

Hygiene

Assist patient to take a bath in order to remove dead epithelium and promote comfort. Do hair care to promote self-esteem and also prevent pediculosis. Do nail care to prevent auto infection and mouth care to prevent halitosis and promote appetite. Change any soiled linen and clothes to promote comfort.

Elimination

Provide a lot of fluids and roughage to prevent constipation. Encourage patient to take a lot of fluids to prevent renal problems and to flush out toxins. Offer a bed pan if he is confined to bed to ensure bowel movement. Apply infection prevention techniques when disposing patient's

excreta to avoid cross infection and further spread. Disinfect the feces and vomitus before disposal.

Nutrition

Provide diet that is nutritious and appetizing to the patient. Small, frequent feedings may be in offered. The diet should include carbohydrates such as nshima to provide energy, proteins such as beans, fish to repair worn out tissues and Vitamins such as vegetables and fruits to boost the immunity and promote skin and mucus membrane integrity. If patient is vomiting administer I.V fluids rich in electrolytes and glucose. Intake and output chart should be maintained to avoid renal failure and also to monitor fluid overload. Serve meals in pleasant surroundings to stimulate patient's appetite. Take weight on same scale, at the same time, with same clothing to monitor weight loss secondary to poor appetite. Avoid fat foods until patient is able to tolerate it.

Exercises

If patient is confined to bed, help patient do passive exercises like limb movement and massage in order to prevent muscle atrophy and promote blood circulation. Encourage the patient to do deep breathing exercises in order to promote lung expansion. Encourage early ambulation as soon as the condition permits in order to prevent deep vein thrombosis and other complications of immobility.

Medication

Administer prescribed drugs at the right time to promote quick recovery. Observe side effects of the drugs.

Health education

- Instruct patient to practice good personal hygiene.
- Stress on the importance of washing hands.
- Encourage optimum sanitation practices.
- Employ proper measures safeguards to prevent use of blood and its components from infected donors.
- Screen food handlers carefully.
- Practice safe preparation and serving of food.
- Educate the patient about his condition in order to create awareness and prevent recurrence of the condition.
- Explain the importance of drug compliance to prevent drug resistance.
- Educate the patient on the sign and symptoms of the condition for early diagnosis and treatment . Educate the patient about the need keep the review dates so that his progress is monitored to ensure full recovery.
- Advise the patient to avoid overcrowding to prevent spread of infection.
- Explain to the patient about the need to take a balanced diet using locally available foods in order to boost the immunity, provide energy and promote healing of worn out tissues.

- Encourage patient to stop taking alcohol.
- Encourage patient to avoid unprotected sexual intercourse until antibodies test negative to avoid reinfection.
- Explain to the patient the importance of balanced diet.
- Stress on the importance of rest.

Complications of hepatitis

- Liver failure - due to sudden and massive destruction of liver cells.
- Chronic hepatitis due to untreated and repeated attacks of hepatitis.
- Hepatic coma resulting from toxins that invade the brain cells.
- Liver cirrhosis resulting from extensive degeneration and destruction of the liver parenchymal cells.
- Cancer of the liver which comes in as a result of chronic inflammation of the hepatocytes leading to repeated cycles of cell death and regeneration resulting into preneoplastic changes such as hepatocytes dysplasia.
- Encephalopathy - this is a neuropsychiatic manifestation of liver damage a terminal complication due to destruction of the astrocytes in the brain by the nitrogenous bacterial waste products not detoxified in the liver but absorbed from the colon together with systemic ammonia remains, characterized by apathy, disorientation, muscular rigidity, delirium and coma.

Liver cirhosis

Liver cirrhosis is a chronic progressive disease of the liver characterized by extensive degeneration and destruction of the liver parenchymal cells (Lewis et al, 2011).

Liver cirrhosis is a progressive disease of the liver with a degeneration of the liver cells and infiltration of the liver by fibrous tissue.

Causes

- Dietary deficiency of protein (PEM and severe Kwashiorkor).
- Alcoholism - It is believed that the combined impact of malnutrition and alcohol causes damage to the hepatocytes. Alcohol alone has a direct hepatotoxic effect on the liver because its known to produce necrosis of cells and fatty infiltration.
- Associated malnutrition (reduce protein intake) and damaged liver cells.
- Viral or toxic hepatitis - chronic inflammation and cell necrosis results in fibrosis and ultimately cirrhosis

- Parasitic infection such as schistosomiasis, and repeated bouts of heart failure with liver congestion can all lead to liver cirrhosis
- Metabolic disorders such as diabetes mellitus.

- Blocked bile ducts - When the ducts that carry bile out of the liver are blocked, bile backs up and damages liver tissue.
- Autoimmune hepatitis- This condition can be caused by the immunologic damage to the liver causing inflammation and eventually scarring and cirrhosis.

Pathophysiology

Early in the disease the liver is enlarged and loaded with fat. As liver cells are destroyed and replaced by scar tissue, it shrinks and becomes hard with a rough surface. As the liver fails to function properly the patient becomes jaundiced and has digestive problems. Blood from the digestive tract which normally flows through the portal system to the liver is slowed down resulting in portal hypertension. This causes ascites, spleenomegally (large spleen), hemorrhoids, and oesophageal varices which may cause haemorrhage. Since albumin is not produced well, there may be oedema and ascites.

Signs and symptoms

- Gastrointestinal problems: Anorexia, nausea and vomiting, dull abdominal pains, diarrhoea or constipation, dyspepsia due to the livers altered metabolism of carbohydrates and fats.
- Hepatomegaly due to fat infiltrating the liver cells.
- Jaundice due to inability of the liver to conjugate bilirubin, compression of bile ducts by connective tissue overgrowth. The jaundice may be minimal or severe depending on the degree of hepatic damage.
- Portal hypertension due to obstruction of the venous system as a result of changes in hepatic vasculature
- Fatigue due to decreased energy reserve as a result of the inability of the liver to metabolize carbohydrates
- Hematologic problems-Anaemia due to increased destruction of red blood cells as a result of unconjugated bilirubin, poor diet, poor absorption of folic acid and bleeding from varices. Coagulation problems result from the inability of the liver to produce prothrombin which is essential for blood clotting. These problems are manifested by bleeding tendencies such as epistaxisis, petechiae, easy bruising, gingival bleeding and heavy menstrual bleeding.
- Skin lesions: spider angiomas (telangiectasis or spider nevi) are small dilated blood vessels with a bright red centre point and spider like branches. These occur on the nose, cheeks, upper trunk, neck and shoulders. Palmar erythema- a red area that blanches with pressure is located on the palms of the hands. These lesions are attributed to an increase in the circulating oestrogen as a result of the damaged livers inability to metabolize steroid hormones.
- Endocrine disturbances. Normally, the liver is supposed to metabolize hormones such as oestrogen and testosterone. When the liver is damaged, its unable to carry out these

functions. In men, gynacomastia, loss of axillary and pubic hair, testicular atrophy, impotence with loss of libido may occur due to oestrogen accumulation. In younger women, amenorrhoea may occur and in older females vaginal bleeding may occur. Aldosterone metabolism is impaired resulting in hyperaldosteronism with subsequent sodium and water retention and potassium loss.

- Finger clubbing
- Ascites - Accumulation of fluid in the peritoneal cavity
- Pluritis

Diagnosis

- History taking – Patient may reveal excessive alcohol intake or recent exposure to hepatitis B or C.
- Physical examination may reveal yellowish dis colouration of the mucous, hepatomegaly and ascites.
- Liver function test will show elevated levels of AST (Aspartate Amino Transferase) and ALT (Alanine Amino Transferase) bilirubin and serum albumin will be reduced.
- Liver biopsy for analysis to detect chronic active disease,, progression and response to therapy.
- Barium swallow will show oesophageal varices.
- Bleeding and clotting time-bleeding time will be increased whereas clotting time will be reduced.
- Computed axial tomography (CAT) scan will show calcification of the liver.
- Ultrasound scan will show enlargement of the liver.

Treatment

Treatment depends on the cause and stage of the cirrhosis. It is aimed at stopping the progress of the cirrhosis, reversing (to whatever extent possible) the damage that has already occurred, and treating complications that are disabling or life-threatening. Stopping or reversing the process requires removal of the cause, as in the following cases.

- **In alcoholic cirrhosis**: abstinence from alcohol and intake of an adequate wholesome diet.
- **In cirrhosis caused by viral hepatitis**: use drugs such as interferone to improve the immune responses of the infections or to help destroy the virus.
- Administer **corticosteroids** such as prednisolone in chronic hepatitis.
- Administersupplemental fat soluble vitamins such as vitamin A.D.E.K
- Vitamin B12 for anaemia
- Potassium sparing diuretics diuretics such as aldoctone or spironolactone 25mg qid to reduce oedema and ascites.
- In severe cirrhosis, a liver transplant may be life-saving

- Abdominal paracentesis in cases where patient has ascites (accumulation of fluids in the peritoneal cavity to relieve abdominal pressure but otherwise should be avoided because it removes protein.
- Vasopressin maybe indicated for oesophageal varices.
- Alcohol is prohibited and sedatives should be avoided.
- Liver transplant
- Avoid drugs which are metabolized by the liver e.g. sedatives, narcotics

Nursing care

Environment

The environment should be well ventilated and warm as patient tends to be feverish. It should also be clean to prevent infections.

Position

The patient is nursed in a semi sitting position to promote maximum respiratory function. This is so because the patient has severe dyspnoea due to pressure of the liver on the diaphragm and also due to Ascitis.

Rest and sleep

The patient finds it difficult to sleep due to discomfort. Ensure that the patient rests adequately to promote quick recovery of the liver and establish its function. Ensure that environment is quite and do similar nursing activities correctly such as bathing, pressure area care, nail acre and oral care.

Observations

Temperature, pulse and respirations are taken every four (4) hours in order to detect a rise or drop in temperature and to monitor if there is any improvement. A rapid pulse is a good indicator of haemorrhage, rapid respirations due to pressure on the diaphragm, Blood pressure is checked also in order to detect on set of shock or gastrointestinal bleeding.

- -monitor the degree of jaundice that is mild, moderate and severe.
- measure the abdominal girth as it is a good indicator of Ascitis.
- -measure weight daily in order to assess for improvement
- -monitor for vomiting; colour, amount, blood and its volume
- -observe the level of consciousness to detect early onset of hepatic coma.

Diet

The patient should be given a diet rich in calories, glucose drink, a high protein diet unless the patient is in coma or has a high level of blood urea. In late disease, proteins may have to be restricted as liver is unable to metabolise it. Small frequent meals should be given as the patient tends to have poor appetite. Give low salt diet because of Ascitis as there is a tendency of

accumulation of sodium. The patient should be advised to eat slowly as there is a tendency to have abdominal pains. Restrict sodium intake because of the ascites and oedema. Advise patient to stop alcohol consumption because it can cause more damage to liver cells.

Elimination

The patient should be given aperients or laxatives to allow them to have pain free bowel motions. If the patient has haemorrhoids give annusol suppositories to help minimise pain and oedema associated with haemorrhoids. Observe the stool for malaena and blood stain. The diet should have plenty of roughage and easily digestible food to prevent rectal bleeding.

Hygiene

The following hygienic needs should be met as the patient is usually weak; give daily baths, four hourly assisted oral care to promote comfort, blood circulation and infections.

Psychological care

The disease causes emotional and psychological distress to the patient. The patient must be assured that the condition can be controlled though it takes long. Ensure compliance from the patient and explain the condition to the relatives.

Advice on discharge

The patient should be advised on the following:

- Need to have adequate rest at home as the patient tends to have fatigue due to the inability of the liver to store glucose which is needed for energy.
- To change the occupation if it's demanding to avoid stress.
- Avoid un prescribed and over the counter drugs to avoid casing more damage to the liver which is already diseased
- Advise the patient to come for review but can come back to the hospital if symptoms persist before the review date such as: confusion, dyspepsia, drowsiness, increase in the level of Ascitis and jaundice so that he can be observed closely The normal review is after six (6) weeks.
- To stop drinking alcohol as this may cause more damage to the liver and hence retard recovery.

Complications

Portal hypertension

Portal hypertension is characterized by increased pressure in the portal circulation as well as splenomegaly, large collateral veins, Ascitis, systemic hypertension and oesophageal varices. Collateral circulation is as a result of an attempt to reduce the high portal pressure, reduce the increased plasma volume and lymphatic flow in the lower oesophagus, anterior abdominal wall, the parietal peritoneum and the rectum.

Oesophageal Varices

Oesophageal varices are due to the tortuous veins at the lower end of the oesophagus, enlarged and swollen due to portal hypertension. The varices rupture and bleed in response to ulceration and irritation due to alcohol ingestion, swallowing of poorly masticated food, ingestion of coarse food, acid regurgitation from the stomach, increased intraabdominal pressure caused by nausea, vomiting, straining at stool, coughing, sneezing or lifting heavy objects. The patient may have malaena stool and hematemesis. Excessive haemorrhage is an emergency

Peripheral Oedema and Ascitis

There is an impaired synthesis of albumin by the liver which leads to decreased oncotic pressure

Liver cancer - Hepatocellular carcinoma, a type of liver cancer commonly caused by cirrhosis, starts in the liver tissue itself. It has a high mortality rate.

Liver Failure leading to hepatic encephalopathy.

Renal Failure due to reduced blood flow to the kidneys.

Anaemia due to bleeding tendancies, loss of iron and hypoproteinaemia.

Severe generalized **infection**

2.9 Manangement Of A Patient With Billiary Disorders

You have now finished learning on conditions of the liver and now coming to the conditions of the biliary which is the gall bladder.

Cholelithiasis/Choledocholithiasis

Definitions

Cholelithiasis is the presence of gall stones in the gallbladder.

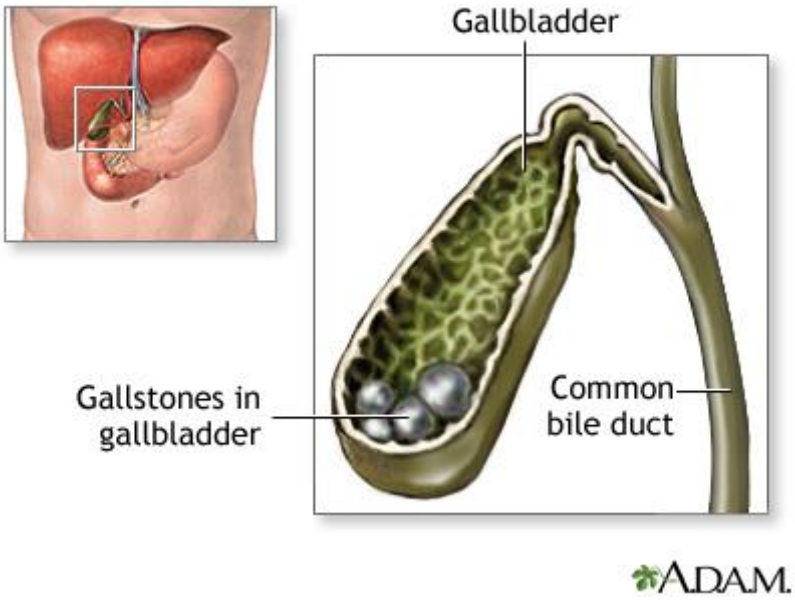
Choledocholithiasis is the presence of gallstones in the bile ducts.

Formation of Gallstones

Gallstones are hard, pebble-like deposits that form inside the gallbladder. Gallstones may be as small as a grain of sand or as large as a golf ball. Gallstones form when bile stored in the gallbladder hardens into pieces of stone-like material. Bile contains water, cholesterol, fats, bile salts, proteins, and bilirubin – a waste product. If the liquid bile contains too much cholesterol or bilirubin or reduced bile salts, it can harden into gallstones. The reason for the imbalances is not known.

There are 2 types of gallstones:

- Cholesterol stones: made out of hardened cholesterol. These are the most common type of gallstones. Cholesterol stones are usually yellow-green.
- Pigment stones: made from too much bilirubin in the bile. Pigment stones are small, dark stones.



FigureTableTableFigure 8:Gall stones

Incidence of Chloelithiasis/choledocolithiasis

The condition affects both sexes at any age but more females are affected than males, and incidence increases with age especially over the age of 40.

Cause/Aetiology

The cause of gallstones is not fully known. The stones tend to develop in people who have liver cirrhosis, biliary tract infections, or hereditary blood disorders such as sickle cell anaemia, in which the liver makes too much bilirubin.

Risk Factors

- **Sex**: women are twice as likely as men to develop gallstones. Excessive estrogen from pregnancy, hormone replacement therapy, and contraceptive pills appears to increase cholesterol levels in bile and decreases gallbladder movement, which can lead to gallstones.
- **Family history of gallstone**: gallstones often run in families.
- **Obesity:** being moderately overweight increases the risk for developing gallstones. The most likely reason is that the amount of bile salts in bile is reduced, resulting in more cholesterol. Increased cholesterol reduces gallbladder emptying.
- **Diet:** diets high in fat and cholesterol and low in fibre increase the risk of gallstones due to increased cholesterol in the bile and reduced gallbladder emptying.
- ***Rapid weight loss*:** as the body metabolizes fat during prolonged fasting and rapid weight loss, the liver secretes extra cholesterol into bile, which can cause gallstones. In addition, the gallbladder does not empty properly.

- ***Age:*** people older than 60 years are more likely to develop gallstones than younger people. As people age, the body tends to secrete more cholesterol into bile.
- ***Ethnicity*****:** some races have a genetic predisposition to secrete high levels of cholesterol in bile e.g. American Indians.
- ***Cholesterol-lowering drugs*****:** drugs that lower cholesterol levels in blood actually increase the amount of cholesterol secreted into bile. In turn, the risk of gallstones increases.
- ***Diabetes:*** people with diabetes generally have high levels of fatty acids called triglycerides. These fatty acids may increase the risk of gallstones.

Signs and Symptoms

As gallstones move into the bile ducts and create blockage, pressure increases in the gallbladder and one or more symptoms may occur. Symptoms of blocked bile ducts may have an abrupt onset. Gallbladder attacks often follow fatty meals, and they may occur during the night. The signs and symptoms include:

- Pain in the right upper or middle upper abdomen
  - ✓ May go away and come back
  - ✓ May be sharp, cramping, or dull
  - ✓ May spread to the back or below the right shoulder blade
  - ✓ Occurs within minutes of a meal
- Fever, even low grade or chills
- Yellowish discolouration of the skin and sclera
- Abdominal fullness
- Clay coloured stools
- Nausea and vomiting
  Fat intolerance (indigestion, pain bloating and belching)

Take Note: 2.4
Many people with gallstones are asymptomatic. The gallstones are often discovered when having a routine x-ray, abdominal surgery, or other medical procedures. These do not need treatment

.

Medical Management Of A Patient With Cholelithiasis

Investigations

- **Abdominal ultrasound**: will show the location and size of gallstones in the biliary tract. This is the most sensitive and specific test for gallstones.
- **Computerized Tomography (CT) Scan:** a non-invasive x-ray that produces cross-section images of the body. The test may show the gall stones or complications, such as infection and rupture of the gallbladder or bile ducts.

- **Cholecystography -** the patient is injected with a small amount of nonharmful radioactive material that is absorbed by the gallbladder the x-ray will show an opaque gall bladder with gall stones which will be seen as dark spots.

- **Endoscopic Retrograde Cholangiopancreatography (ERCP):** ERCP is used to locate and remove stones in the bile ducts. After lightly sedating the patient, an endoscope is inserted down in the oesophagus and through the stomach and into the small intestine. A special dye is injected so that the affected bile duct and the gallstone are located on a monitor. The stone is captured and removed with the endoscope.
- **Blood tests:** blood tests may be performed to look for signs of infection, obstruction, pancreatitis, or jaundice e.g. bilirubin, liver function tests and pancreatic enzymes.

Differential Diagnosis

Heart attack, appendicitis, peptic ulcers, irritable bowel syndrome, hiatus hernia, pancreatitis, and hepatitis.

Aims of Management

- To control pain
- To control possible infection with antibiotics
- To maintain fluid and electrolyte balance

Drug Therapy

Oral dissolution therapy

- Chenodeoxycholic acids (CDCA, Chenodiol): dissolution of cholesterol stones. 250mg P.O. b.i.d for the first 2 weeks, followed, as tolerated, by weekly increases of 250mg/day, up to 13 to 16 mg/kg/day for up to 24 months.
- Ursodeoxycholic acid (UDCA, Ursodiol, Actigall): dissolution of gallstones less than 20mm in diameter when surgery is not possible. 8 to 10mg/kg P.O. daily in two or three divided doses for 12 to 24 months.

Contact dissolution therapy

- Methyl tert-butyl ether: this drug is still being experimented. It is injected directly into the gallbladder to dissolve cholesterol stones.

Analgesics for pain (opiod analgesics)

Antispasmodics or anticholinergics to decrease secretions (which prevent biliary contraction) and counteract smooth muscle spasms e.g. dicyclomine

Antiemetics to control nausea and vomiting.

Antibiotics to eliminate infection.

If the above treatment fails, surgery is performed and this includes:

Cholecystectomy - removal of gallbladder.

Cholecystostomy (usually an emergency) - incision into the gallbladder to remove stones.

Complications of Cholelithiasis

- Infection of the gallbladder - the presence of the gall bladder may lead to infection.
- Rupture of the gallbladder
- Acute cholecystitis may develop if the gallstone becomes stuck in the neck of the gall bladder causing inflammation.
- Cancer of the gallbladder due to continued irritation.
- Pancreatitis which may be due to blockage of the pancreatic duct caused by gall stone.
- Small bowel obstruction and paralysis due to gallstone causing obstruction.
- Obstructive jaundice caused by presence of gall stone in the bile duct.

Cholecystitis

Definition: this is the inflammation of the gall bladder.

Cholecystitis can either be acute or chronic.

Aetiology (causes)

- Obstruction caused by gallstones or biliary sludge
- Following trauma, extensive burns or recent surgery
- Prolonged total parenteral nutrition and diabetes mellitus
- Bacteria reaching the gallbladder via the vascular or lymphatic route
- Chemical irritants in the bile
- Adhesions, neoplasm, anaesthesia and narcotics
- Inadequate blood supply

Causative Organisms (For bacterial causes of acute cholecystitis only)

- Escherichia coli (most common)
- Streptococci
- Salmonella

Clinical Features:

- Episodes or vague pain in the right upper quadrant of the abdomen that radiates to the right shoulder
- Pain is triggered by a high fat or high volume meal
- Anorexia
- Nausea and vomiting Dyspepsia
- Mild to moderate fever

- Acute abdominal tenderness and a positive Murphy's sigh (Palpation of abdomen causing severe increase in pain and temporary respiratory arrest)
- Patient may wake up at night due to pain
- Jaundice
- Clay coloured stool
    - **Diagnosis**
- lassical sign and symptom pain in the right upper quadrant of the abdomen that radiate to the shoulder usually precipitated by consumption of fat diet. Positive Murphy's sign
- Ultrasound will reveal inflamed gall bladder.
- Liver function tests
- MRI may also be very useful where available

Management:

1. Medical

- Anti-spasmodics (Atropine, Probanthine)
- IV fluids,
- Antibiotics for acute attack for example, ampicillin, or septrin
- Analgesia such as Pethidine 100 mg.

2. ***Surgical***- urgery is indicated where patient does not respond to medical treatment.

    Cholecystectomy--removal of gall-bladder

Nursing Care of a Patient with Cholelithiasis and cholecystitis

If the gallstones cause no symptoms, the patient should be left alone. No treatment should be given. However, hospitalization may be required if patient's pain lasts more than six hours.

Pain Relief: when patient has biliary colic, the patient should remain in bed and pethidine administered. If it is not possible to administer pethidine, antispasmodic drugs such as atropine, propantheline or nitroglycerin and morphine should be administered to relief painful reflex spasm that occurs in response to the stone in a duct. Local applications of heat to the upper abdomen may be done.

Diet: during an episode of biliary colic, the patient should not be given anything by mouth but administer the prescribed intravenous fluids. If vomiting and abdominal distention occur, a nasogastric tube is passed for suctioning in order to decompress the stomach. Local applications of heat to the upper abdomen may be ordered. After the acute episode and removal of the nasogastric tube, clear fluids are given and gradually increased to a light, low fat diet as

tolerated. Patient should be given replacement of fat soluble vitamins. Bile salts are also administered to facilitate digestion and vitamin absorption. If the patient is overweight, weight reduction should be considered. Provide low fat diet. Foods to be avoided include; dairy products such as whole milk, ice cream, cheese, fried foods, gravies, nuts.

Activity: there should be no restrictions of activity except during attacks of gallbladder colic when patient is advised to rest.

Observations: observe the patient's skin and sclera for signs of jaundice. Observe the colour of stool, if pale, there is an obstruction in the biliary duct and therefore a daily dose of vitamin K is given parenterally to maintain prothrombin formation and prevent bleeding. Also examine the stool for the presence of stones passed from the biliary tract into the intestine. Vital signs observations should be done twice daily unless the patient develops fever in which case observe four hourly.

Complications of Cholecystitis

Perforation

Gallstones

Cholangitis due to spread of infection along the bile ducts, causing jaundice

- Empyema (pus in the gallbladder)
- Gangrene (tissue death) of the gall bladder
- Pancreatitis
- Peritonitis (inflammation of the lining of the abdomen)

2.10Worm Infestations

Round Worms (Ascariasis)

Ascariasis is a human disease caused by the parasitic roundworm known as *Ascaris lumbricoides*. Perhaps as many as one quarter of the world's people are infected, and ascariasis is particularly prevalent in tropical regions and in areas of poor hygiene. Ascariasis is also called round worm infection**.**

and

Figure 9:Female male Ascaris

lumbricoides; the female measures approximately 16 inches (40 cm) in length.

Table

FigureTableFigure 10: A large mass of Ascaris lumbricoides that was passed from a person's intestinal tract

Find below is a picture of an Ascaris lumbricoides

Figure 11:Ascaris lumbricoides

TableTableSource: www.wikipedia.com 2013

Epidemiology

Roughly 1.5 billion individuals are infected with this worm, primarily in Africa and Asia.

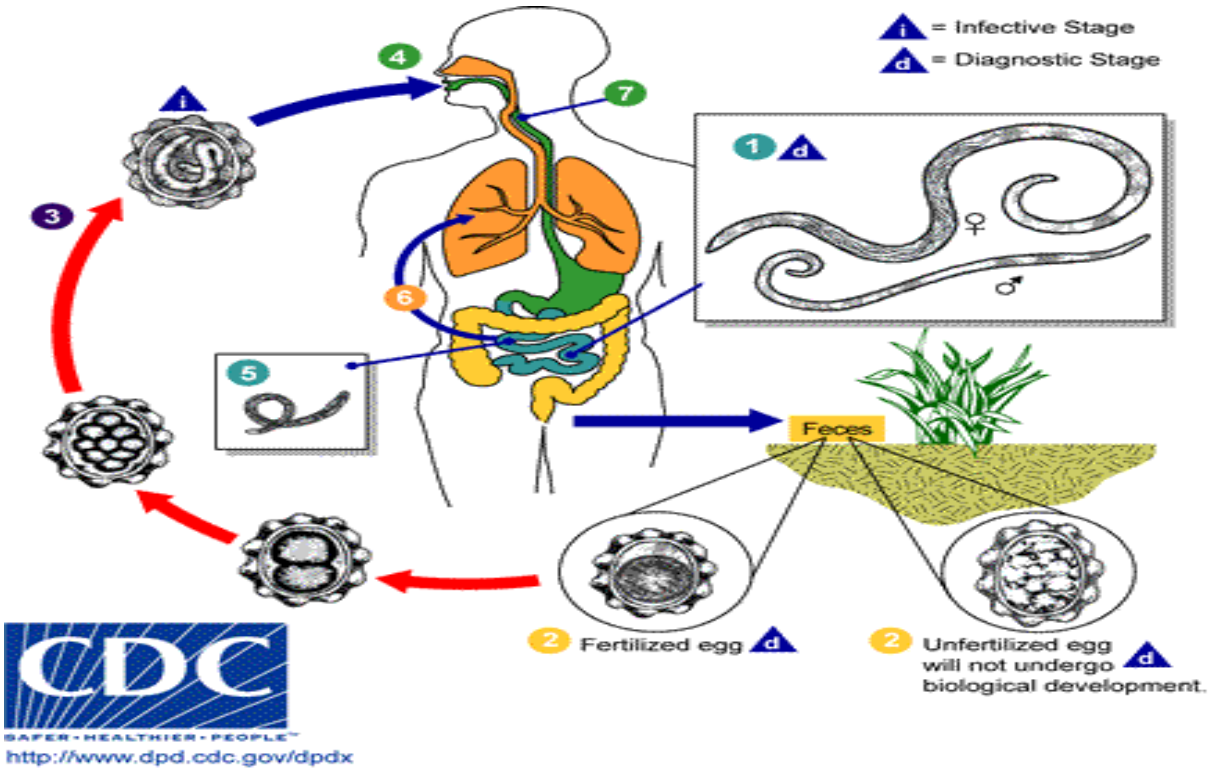
Ascariasis is endemic in the United States including Gulf Coast; in Nigeria and in Southeast Asia.

Causes

- Ascariasis is caused by *Ascaris lumbricoides.*
- It is transmitted to humans by ingestion of soil contaminated with human feces that harbours Ascaris lumbricoides ova.
- Ingestion may occur directly (by eating contaminated soil) or indirectly (by eating poorly washed raw vegetables grown in contaminated soil).

I hope you are moving on well and understanding the learning materials, now let's discuss the life cycle of Round worms (Ascariasis)

Life Cycle of Roundworms (Ascariasis)



FigureTableFigure 12 Life Cycle of Roundworms (Ascariasis)

Source: http://www.dpd.cdc.gov/dpdx

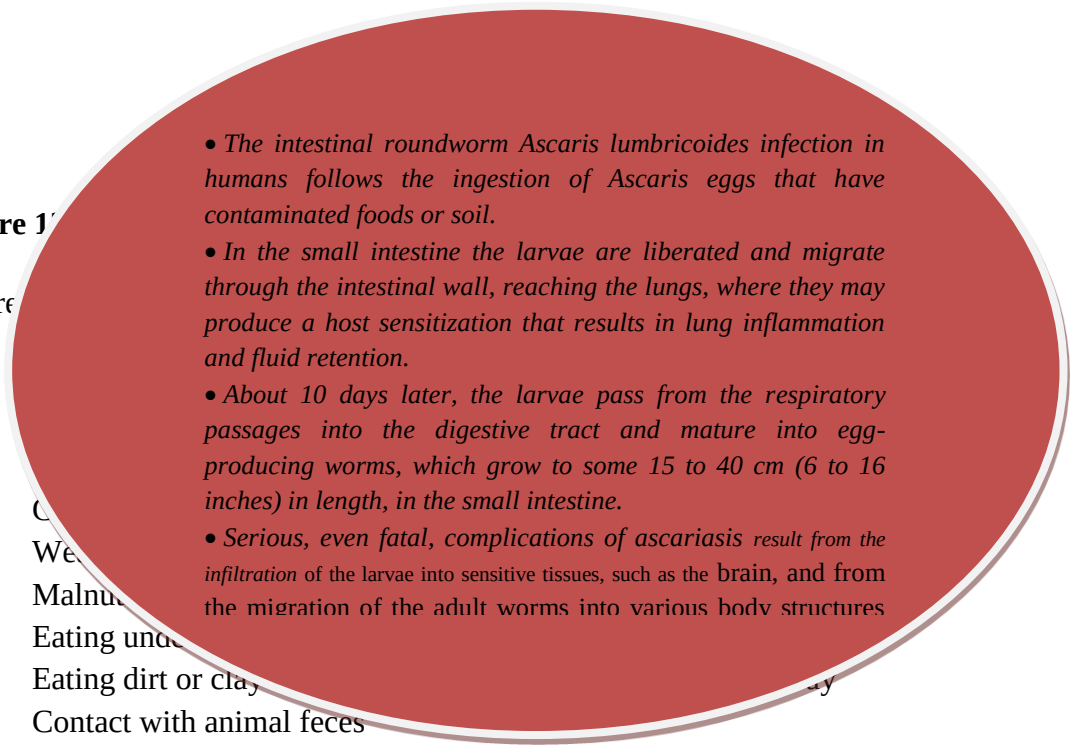
Life Cycle

- Ascariasis is never passed directly from person to person. After ingestion, A.lumbricoides ova hatch and release larvae, which penetrate the intestinal wall and reach the lungs through the bloodstream.
- After about ten (10) days in pulmonary capillaries and alveoli, the larvae migrate to the bronchioles, bronchi, trachea and epiglottis.
- There, they are swallowed and returned to the intestine to mature into worms.

Figure 1

Figure

- We
- Malnu
- Eating und
- Eating dirt or cla
- Contact with animal feces



Signs and Symptoms

- Vague stomach discomfort
- Vomiting a worm or passing a worm in stool
- Restlessness
- Disturbed sleep
- Signs of intestinal obstruction

- Weight loss
- Impaired growth
- Fever
- Abdominal distension

Diagnosis

- The diagnosis is usually incidental when the host passes a worm in the stool or vomit.
- Stool samples for ova and parasites will demonstrate *Ascaris* eggs.
- Larvae may be found in gastric or respiratory secretions in pulmonary disease.
- Blood counts may demonstrate peripheral eosinophilia.
- On X-ray, 15–35 cm long filling defects, sometimes with whirled appearance (bolus of worms).

Treatment

Pharmaceutical drugs that are used to kill roundworms are called *ascaricides* and include:

- ***Mebendazole***(Vermox) Causes slow immobilization and death of the worms by selectively and irreversibly blocking uptake of glucose. Oral dosage is 100 mg 12 hourly for 3 days.
- ***Piperazine:***A flaccid paralyzing agent that causes a blocking response of ascaris muscle to acetylcholine. The narcotizing effect immobilizes the worm, which prevents migration when treatment is accomplished with weak drugs such as thiabendazole. If used by itself it causes the worm to be passed out in the feces. Dosage is 75 mg/kg (max 3.5 g) as a single oral dose.
- ***Pyrantel pamoate;*** leads to depolarization of the ganglionic and block of nicotinic neuromuscular transmission, resulting in spastic paralysis of the worm. Spastic paralyzing agents, in particular pyrantel pamoate, may induce complete intestinal obstruction in a heavy worm load. Dosage is 11 mg/kg not to exceed **1 g** as a single dose.
- ***Albendazole****;* A broad-spectrum antihelminthic agent that decreases ATP production in the worm, causing energy depletion, immobilization, and finally death. Dosage is 400 mg given as single oral dose (contraindicated during pregnancy and children under 2 years).

Other Drugs

- ***Thiabendazole***. This may cause migration of the worm into the esophagus, so it is usually combined with piperazine.
- ***Hexylresorcinol***effective as a single dose,
- **Santonin**,

Nursing Diagnosis

- Altered growth and development
- Altered nutrition: less than body requirements
- Altered thought processes
- Colonic constipation
- High risk for fluid volume deficit
- High risk for infection
- Hyperthermia
- Ineffective breathing pattern
- Knowledge deficit
- Pain

Tapeworms (Cestodes)

Tapeworm infestation is the infection of the digestive tract by adult parasitic flatworms called cestodes or tapeworms. Live tapeworm larvae (coenuri) are sometimes ingested by consuming undercooked food. Once inside the digestive tract, a larva can grow into a very large adult tapeworm. Additionally, many tapeworm larvae cause symptoms in an intermediate host.

Common Types Of Tapeworm

Among the most common tapeworms in humans are:

- **Taenia Solium- the pork tapeworm**
- **Taenia saginata- the beef tapeworm**
- **Diphyllobothrium- the fish tapeworm**
- **Hymenolepis- the dwarf tapeworm**

Mode of transmission

•Ingestion of eggs-Tapeworm eggs are generally ingested through food, water or soil contaminated with human or animal faeces. For example, if a pig is infected with a tapeworm, it may pass eggs or segments (proglottids) of the adult tapeworm through its feces into soil. Each segment contains thousands of microscopic tapeworm eggs.

•Ingestion of larvae cysts in meat or muscle tissue

Tapeworm infection can also be caused by eating raw or undercooked meat from an animal or a fish that has the larval form of the tapeworm cysts in its muscle tissue. Once ingested, the larvae then develop into adult tapeworms in the intestines.

Life **Cycle Of A Beef And Pork Tape Worm**

The adult worms live in the human intestines and may grow 15 to 30 feet (beef tape worm) and or 8 to 10 feet (pork tape worm).egg bearing sections of the worm (proglottids) are passed in the stool and are eaten by the cattle. The eggs hatch in the cattle and invade the intestinal wall. They are then carried in the blood stream to the skeletal muscle, where they form cysts (cysticerci). People are infected by eating the cysts in the raw or undercooked beef. In pig tape worm the pig serve as the intermediate host. People also may act as intermediate host; The eggs reach the stomach either when a person swallows them or when proglottids are regurgitated from the intestines to the stomach. The embryos are released inside the stomach. They then penetrate the intestinal wall and travel to muscles, internal organs, the brain and tissue under the skin where they form cysts. Live cysts cause only a mild tissue reaction whereas dead ones invoke a vigorous reaction.

Signs and symptoms

Although tapeworms in the intestine usually cause no symptoms, some people experience:

- A feeling of motile proglottids emerging from the anus which causes distress.
- Upper abdominal discomfort
- Diarrhea and loss of appetite
- Loss of weight
- Nausea and vomiting
- Anorexia
- Anaemia may develop in people with the fish tapeworm.
- Rarely, worms may cause obstruction of the intestine.
- Occasionally segments of worm may be vomited.
- And very rarely, T. solium larvae can migrate to the brain causing severe headaches, seizures and other neurological problems. This condition is called neurocysticercosis. It can take years of development before the patient has those symptoms of the brain.

Diagnosis

- Infection is generally recognized when the infected person passes segments of proglottids in the stool (looks like white worms), especially if a segment is moving.

- Eggs may be seen around the anus or in stool in pork tape worm
- CT scan or MRI may reveal live cysts in such tissues as the brain in pork tape worm.
- Blood tests for antibodies against the parasites can be done

Treatment

Tapeworms are treated with medications taken by mouth, usually in a single dose.

- The drug of choice for tapeworm infections is praziquantel 10mg/kg is the drug of choice.
- Niclosamide can also be used.

Prevention of tape worms

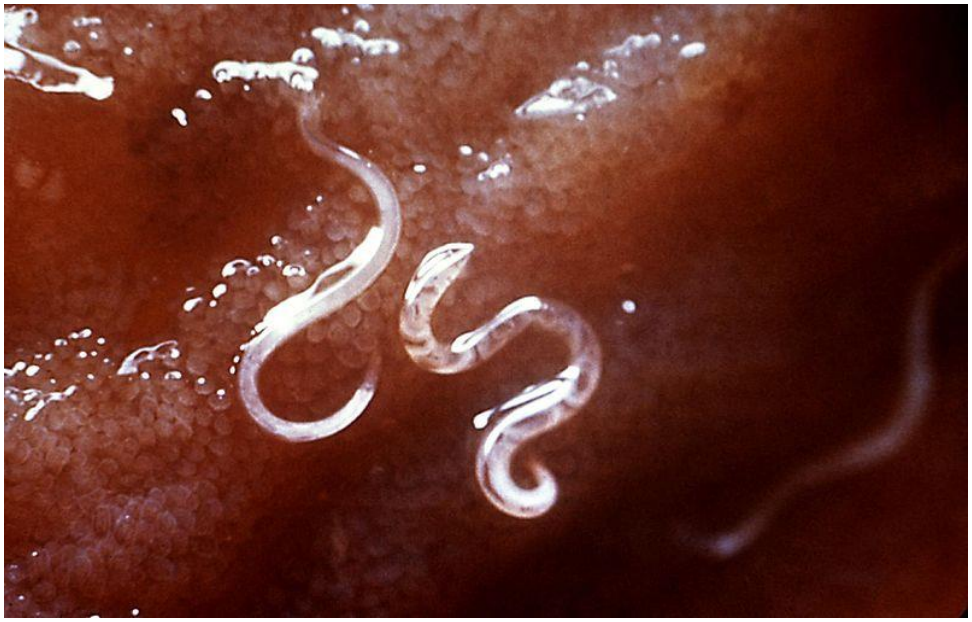
- Thorough cooking of pork, beef and fish.
- Infected persons should be treated with niclosamide or prasiquantel orally
- The stool of infected people should be re checked after 3 months and 6 months to ensure that the infection is cured.
- Infection can also be prevented by enforcing meat inspection and forbidding the sale of infected meat
- Good sanitary conditions will also help in preventing the infection

Hook Worm Disease

The **hookworm** is a parasitic nematode worm that lives in the small intestine of its host, human.

Hook worm disease is also called uncinariasis

Hookworm is a leading cause of maternal and child morbidity in the developing countries of the tropics and subtropics.



FigureTableFigure 12: hookworms

- In susceptible children hookworms cause intellectual, cognitive and growth retardation, intrauterine growth retardation, prematurity and low birth weight among new-borns born to infected mothers.
- Hookworm infection is rarely fatal, but anaemia can be significant in the heavily infected individual.

Epidemiology

It is estimated that between 576-740 million individuals are infected with hookworm today. Of these infected individuals, about 80 million are severely affected.

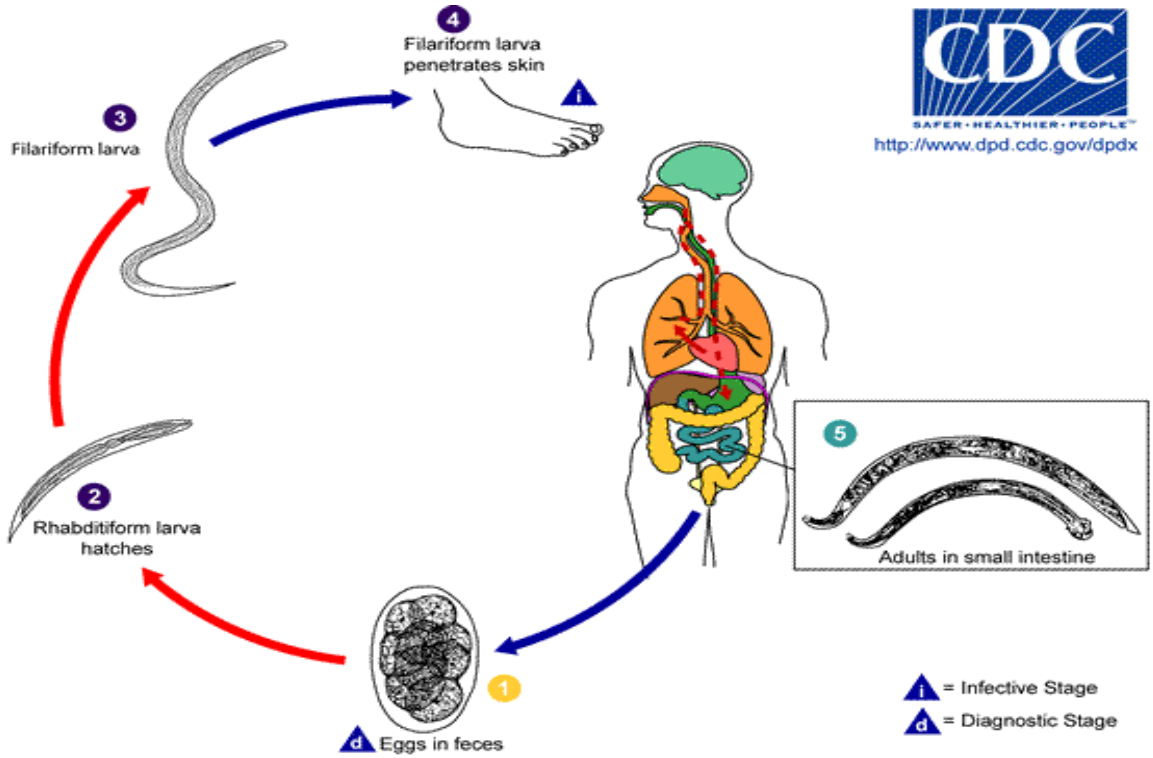
Causes

- Two species of hookworms commonly infect humans, *Ancylostoma duodenale*and *Necator americanus*.
- *Necator americanus* predominates in the America, Sub-Saharan Africa, Southeast Asia, China, and Indonesia.
- *Ancylostoma duodenale* predominates in the Middle East, North Africa, India and (formerly) in southern Europe.
- *Ankylostomiasis*, alternatively spelled *anchylostomiasis* and also called *helminthiasis,* is the disease caused by hookworms.
- It is caused when hookworms, present in large numbers, produce iron deficiency anaemia by voraciously sucking blood from the host's intestinal walls.

Morphology

- *A. duodenale* worms are greyish white or pinkish with the head slightly bent in relation to the rest of the body.
- They possess well developed mouths with two pairs of teeth.
- While males measure approximately one centimetre by 0.5 millimetres, the females are often longer and stouter.

Life Cycle of Hook worms



FigureTableFigure 13:Summary of biological life cycle

- *N. Americanus* and *A. duodenale* eggs can be found in warm, moist soil where they will eventually hatch into first stage larvae.
- The feeding non-infective rhabditoform stage, will feed on soil microbes and eventually molt into second stage larvae, which is also in the rhabditoform stage, will feed for approximately 7 days and then molt into the third stage larvae.
- This is the filariform stage of the parasite, that is, the non-feeding infective form of the larvae.

Life Cycle In Man

Figure 14: Life Cycle In Man

Summary of Biological Life Cycle

- TableEventually, the larvae will enter the lungs through the pulmonary capillaries and break out into the alveoli.
- They will then travel up the trachea to be coughed and swallowed by the host.
- After being swallowed, the larvae are then found in the small intestine where they molt into the adult worm stage.
- The entire process from skin penetration to adult development takes about 5–9 weeks.
- The female adult worms will release eggs (*N. Americanus* about 9,000-10,000 eggs/day and *A. duodenale* 25,000-30,000 eggs/day) which are passed in the feces of the human host.
- These eggs will hatch in the environment within several days and the cycle with start anew.

Incubation period

The incubation period can vary between a few weeks to many months and is largely dependent on the number of Hookworm parasites an individual is infected with.

Pathophysiology

Hookworm infection is generally considered to be asymptomatic, but is an extremely dangerous infection because its damage is “silent and insidious.”There are general symptoms that an individual may experience soon after infection.

- Ground-itch, which is an allergic reaction at the site of parasitic penetration and entry, is common in patients infected with *N. americanus*.
- Additionally, cough and pneumonitis may result as the larvae begin to break into the alveoli and travel up the trachea.
- Then once the larvae reach the small intestine of the host and begin to mature, the infected individual will suffer from diarrhea and other gastrointestinal discomfort.

However, the “silent and insidious” symptoms refer mainly to chronic, heavy-intensity hookworm infections.

Major morbidity associated with hookworm is caused by intestinal blood loss, iron deficiency anaemia, and protein malnutrition.

They result mainly from adult hookworms in the small intestine ingesting blood, rupturing erythrocytes, and degrading haemoglobin in the host.

This long-term blood loss can manifest itself physically through facial and peripheral oedema; eosinophilia and pica caused by iron deficiency anaemia.

It is widely accepted that children who suffer from chronic hookworm infection can suffer from growth retardation as well as intellectual and cognitive impairments.

Signs and Symptoms

- The symptoms can be linked to inflammation in the gut stimulated by feeding hookworms;
- Nausea,
- Abdominal pain and intermittent diarrhea
- Progressive anaemia in prolonged disease:
- Capricious appetite,
- Pica (or dirt-eating),
- Obstinate constipation followed by diarrhea,
- Palpitations, thready pulse, coldness of the skin, pallor of the mucous membranes, fatigue and weakness, shortness of breath
- Cases running a fatal course, dysentery, haemorrhages and oedema.

Diagnosis

- Diagnosis depends on finding characteristic worm eggs on microscopic examination of the stools.

Treatment

- *Albendazole* is effective both in the intestinal stage and during the stage the parasite is still migrating under the skin.
- In case of anaemia, iron supplementation can cause relief symptoms of iron deficiency anaemia.
- However, as red blood cell levels are restored, shortage of other essentials such as *folic acid*or *vitamin B12*may develop, so this might also be supplemented.
- The most common treatment for Hookworm are*Benzimidazoles (BZAs***),** specifically albendazole and mebendazole. BZAs kill adult worms by binding to the nematode's β-tubulin and subsequently inhibiting microtubule polymerization within the parasite.
- In certain circumstances, *levamisole*and *pyrantel pamoate*may be used.

Nursing Diagnosis

- Altered growth and development
- Altered nutrition; less than body requirements
- Diarrhoea
- Fatigue
- High risk for fluid volume deficit
- High risk for infection
- Knowledge deficit
- Ineffective breathing pattern

Prevention

The infective larvae develop and survive in an environment of damp dirt, particularly sandy and loamy soil. The main lines of precaution are those dictated by sanitary science:

- Do not defecate in places other than latrines, toilets etc.
- Do not use human excrement or raw sewage or untreated 'night soil' as manure/fertilizer in agriculture
- Deworm

- Hand washing

Pin Worms

The *pinworm* genus *Enterobius*, also known as *threadworm* or*seatworm*, is a nematode and a common intestinal parasite, especially in humans. The medical condition caused by pinworm infestation is known as *enterobiasis*.

Transmission

Pinworms spread through human-to-human transmission, by ingesting (i.e., swallowing) infectious pinworm eggs and/or by anal insertion. The eggs are hardy and can remain viable (i.e., infectious) in a moist environment for up to three weeks.

After the eggs have been initially deposited near the anus, they are readily transmitted to other surfaces through contamination. The surface of the eggs is sticky when laid, and the eggs are readily transmitted from their initial deposit near the anus to fingernails, hands, night-clothing and bed linen. From here, eggs are further transmitted to food, water, furniture, toys, bathroom fixtures and other objects. Dust containing eggs can become airborne and widely dispersed when dislodged from surfaces, for instance when shaking out bed clothes and linen. Consequently the eggs can enter the mouth and nose through inhalation, and be swallowed later. Although pinworms do not strictly multiply inside the body of their human host, some of the pinworm larvae may hatch on the anal mucosa, and migrate up the bowel and back into the gastrointestinal tract of the original host. This process is called *retroinfection*. When this retroinfection occurs, it leads to a heavy parasitic load and ensures that the pinworm infestation continues. Autoinfection (i.e., infection from the original host to itself), either through the anus-to-mouth route or through retroinfection, causes the pinworms to inhabit the same host indefinitely.

Epidemiology

The pinworm has a worldwide distribution, and is the most common helminth (i.e., parasitic worm) infection. Pinworms are particularly common in children. Finger sucking has been shown to increase both incidence and relapse rates, and nail biting has been similarly associated. Because it spreads from host to host through contamination, pinworms are common among people living in close contact theytend to occur in all people within a household. The prevalence of pinworms is not associated with gender, or with any particular social class, race, or culture.

Pinworm infections are more common within families with school-aged children, in primary caregivers of infected children, and in institutionalized children.

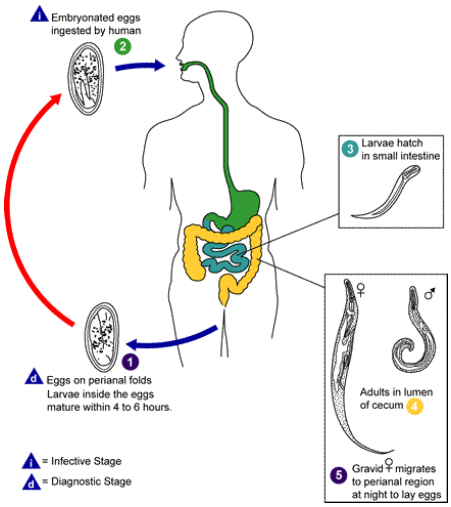
A person is infected with pinworms by ingesting pinworm eggs either directly or indirectly. These eggs are deposited around the anus by the worm and can be carried to common surfaces

such as hands, toys, bedding, clothing, and toilet seats. By putting anyone's contaminated hands (including one's own) around the mouth area or putting one's mouth on common contaminated surfaces, a person can ingest pinworm eggs and become infected with the pinworm parasite. Since pinworm eggs are so small, it is possible to ingest them while breathing.

Once someone has ingested pinworm eggs, there is an incubation period of 1 to 2 months or longer for the adult gravid female to mature in the small intestine. Once mature, the adult female worm migrates to the colon and lays eggs around the anus at night, when many of their hosts are asleep. People who are infected with pinworm can transfer the parasite to others for as long as there is a female pinworm depositing eggs on the perianal skin. A person can also re-infect themselves, or be re-infected by eggs from another person.

In humans, enterobius vermicularis causes the medical condition enterobiasis, whose primary symptom is itching in the anal area.

Life Cycle



The entire life cycle—from egg to adult—takes place in the human gastrointestinal tract of a single human host.

The life cycle begins with eggs being ingested. The eggs hatch in the duodenum. The emerging pinworm larvae grow rapidly to a size of 140 to 150 micrometers in size,and migrate through the small intestine towards the colon. During this migration they moult twice and become adults. Females survive for 5 to 13 weeks, and males about 7 weeks. The male and female pinworms mate in the ileum where after the male pinworms usually die, and are passed out with stool. The gravid female pinworms settle in the ileum, caecum (i.e., beginning of the large intestine), appendix and ascending colon, where they attach themselves to the mucosa and ingest colonic

contents. Almost the entire body of a gravid female becomes filled with eggs. The estimations of the number of eggs in a gravid female pinworm range from about 11,000 to 16,000. The egg-laying process begins approximately five weeks after initial ingestion of pinworm eggs by the human host. The gravid female pinworms migrate through the colon towards the rectum at a rate of 12 to 14 centimeters per hour. They emerge from the anus, and while moving on the skin near the anus, the female pinworms deposit eggs either through (1) contracting and expelling the eggs, (2) dying and then disintegrating, or (3) bodily rupture due to the host scratching the worm. After depositing the eggs, the female becomes opaque and dies. The reason the female emerges from the anus is to obtain the oxygen necessary for the maturation of the eggs.

Treatment

- The medications used for the treatment of pinworm are mebendazole, pyrantel pamoate, and albendazole.
- All three of these drugs are to be given in 1 dose at first and then another single dose 2 weeks later.
- The medication does not reliably kill pinworm eggs. Therefore, the second dose is to prevent re-infection by adult worms that hatch from any eggs not killed by the first treatment.
- Health practitioners and parents should weigh the health risks and benefits of these drugs for patients under 2 years of age.

Repeated infections should be treated by the same method as the first infection. In households where more than one member is infected or where repeated, symptomatic infections occur, it is recommended that all household members be treated at the same time. In institutions, mass and simultaneous treatment, repeated in 2 weeks, can be effective.

Prevention & Control

- Washing your hands with soap and warm water after using the toilet, changing diapers, and before handling food is the most successful way to prevent pinworm infection.
- In order to stop the spread of pinworm and possible re-infection, people who are infected should bathe every morning to help remove a large amount of the eggs on the skin. Showering is a better method than taking a bath, because showering avoids potentially contaminating the bath water with pinworm eggs. Infected people should not co-bathe with others during their time of infection.
- They should also cut fingernails regularly, and avoid biting the nails and scratching around the anus.
- Frequent changing of underclothes and bed linens first thing in the morning is a great way to prevent possible transmission of eggs in the environment and risk of reinfection. These items

should not be shaken and carefully placed into a washer and laundered in hot water followed by a hot dryer to kill any eggs that may be there.

- In institutions, day care centres, and schools, control of pinworm can be difficult, but mass drug administration during an outbreak can be successful. Teach children the importance of washing hands to prevent infection.

We have come to the end of the second unit; Let us now review what we discussed in this unit.

2.9 Summary

In this unit, you have learnt quite a number of things than in the previous unit. You started the unit by first reviewing the anatomy and physiology of the digestive system which is also known as the gastrointestinal tract.. Under this topic you learnt that the digestive system starts from the mouth to the anus. It also have some other structures which are called accessory organs and these are the liver, pancreas and the gall bladder. The digestive system carries out some functions which include ingestion of food, digestion, absorption and elimination of waste products. You also learnt the about the common procedures and investigations which are done in the digestive system disorders such as the endoscopies, liver biopsy, liver function test, barium meal, barium swallow, barium enema and many others. After looking at the investigations and procedures of the GIT, you proceeded on learning the disorders of the GIT. The GIT disorders included the stomatitis which is one of the disorders of the mouth and was defined as the inflammation of the oral mucosa. Other conditions of the mouth and oesophagus which you learnt are; parotitis, achalasia, gastresophageal reflux and hiccup. Disorders of the stomach and duodenum were discussed as well. Under this topic, you covered the 2 conditions which are gastritis and peptic ulcers. Gastritis was defined as inflammation of the gastric mucosa which can be acute or chronic. Peptic ulcer meanwhile was defined as an erosion or break in the lining of the GIT which comes in direct contact with the gastric juices. Type of peptic ulcer depends on the location such as the esophageal ulcer which is situated in the oesophagus, gastric ulcers in the stomach and duodenal ulcers in the duodenum. The other disoders which you learnt are those of the intestines which included the malabsorption, chrohn's disease, dysentery, cholera, typhoid and paratyphoid. Disorders of the liver and the gall bladder were also discussed and they included; hepatitis which is the inflammation of the liver, a liver cirrhosis, progressive condition of the liver which is characterised by destruction and degeneration of the liver paranchyma. Billiary disorders covered cholelitiasis, a condition where there is presence of stones in the gall bladder and cholecystitis which refers to inflammation of the bladder. Lastly you also learnt on worm infestations which included, round worm, tape worms, hook worms and pin worms. In worm infestations, you learnt that worms can be transmitted using different modes such as through ingestion of infective cysts and can penetrate through the skin.

Hope you have learnt something and achieved the unit objectives. In the next unit you will learn on the cardiovascular system. Before you start the new unit, let's see if the unit objectives have been achieved by answering the following

questions.

2.10Self-assessment test

1. List four procedures and investigations that can be done in digestive system disorders.
   A) ----------
   B) ----------
   C) ----------
   D) ----------
2. Stomatitis refers to inflammation of the:
   A) Stomach
   B) Liver
   C) Mouth
   D) Parotid gland
3. Gastritis refers to inflammation of the:
   A) Mouth
   B) Esophagus
   C) Stomach
   D) Gum
4. A condition where there is lack of peristaltic movement in the oesophagus and failure of relaxation of lower esophageal sphincter is referred to:

   A) Gastresophageal reflux

   B) Hiatus hernia

   C) Achalasia

   D) Peptic ulcer

5. The following are the common sites of peptic ulcers except:

   A) The lower part of the oesophagus

   B) First part of the duodenum

   C) The first part of the colon

   D) The stomach

6. What is the name of the common organism which is associated with peptic ulcers?
   A) Salmonella typhi
   B) Helicobacter pylori
   C) Hebacter Pyrolus

D) E. coli

7. Cholelithiasis refers to:

A) Inflammation of the gall bladder

B) Inflammation of the urinary bladder

C) Presence of stones in the gall bladder

D) Presence of stones sin the urinary bladder.

Match the following disorders in this column right column with causative organism in the left column.

8. Cholera	A) Shigella
9. Typhoid fever	B) Vibrio cholerae
10. Bacillary dysentery	C) Entamoeba hystolytica
11. Amoebic dysentery	**D) Salmonella typhi**

2.11Answers to self-assessment

1. Liver function test
   Endoscopy
   Barium meal
   Barium swallow
   Barium enema
   Liver biopsy
   Spleenography
   Stool examination
   (*Any of these*)
2. C
3. C
4. C
5. C
6. B
7. C
8. B
9. D
10. A
11. C

2.12 References

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UNIT 3 INTRODUCTION RESPIRATORY SYSTEM

3.0 Introduction

Welcome to our new unit on respiratory system. In the previous unit you learnt about the disorders of the digestive system. You first reviewed the anatomy and physiology of the digestive system and then went on to learn on the common investigations and procedures which are done on the disorders of the digestive system. Different disorders affecting the digestive system were discussed which included blood disorders such as anaemia, heart disorders like the congestive cardiac failure peripheral disorders such as venous thrombosis and just other disorders such as malaria.

In this unit you will again start with reviewing the anatomy and physiology of the respiratory system and the common investigations and procedures. Thereafter you will start learning the conditions affecting the respiratory system starting with upper respiratory tract disorders Followed by the Lower respiratory system disorders

3.1 Unit Objectives

By the end of this unit you should be able to:

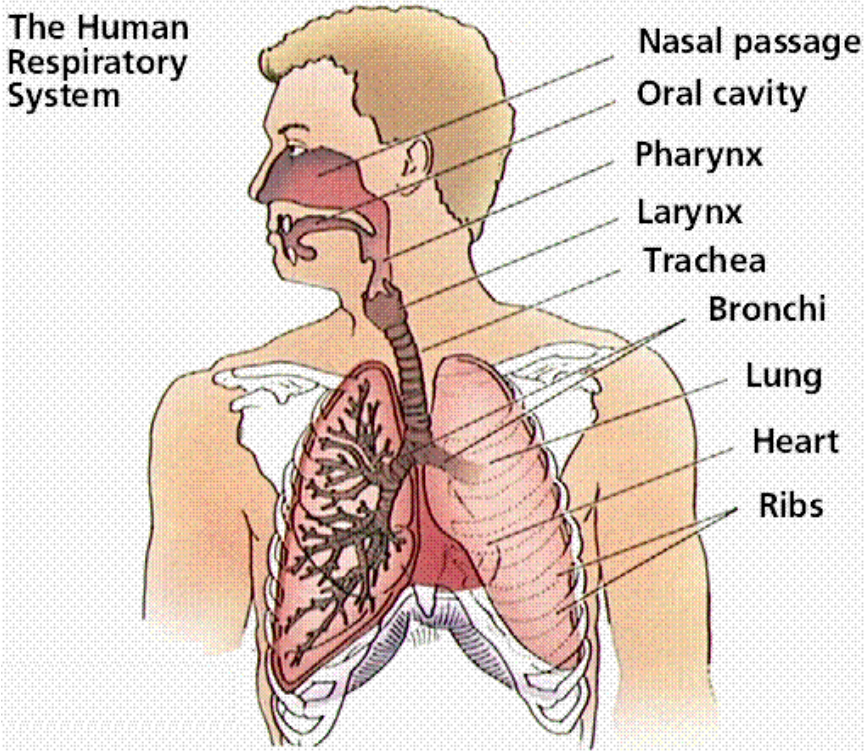
1. Discuss the anatomy and physiology of the respiratory system
2. Explain the role of the nurse in investigations and procedures of the respiratory disoders
3. Discuss the management of a patient with Upper Respiratory Tract disorders.
4. Discuss the Management of a patient with Lower Respiratory Tract disorders.

3.2 Anatomy and Physiology of the Respiratory System

This topic of the respiratory system is not new to you. You might have covered it at the primary school and secondary school though not in details. You also learnt about it in details in your foundation block under Anatomy and Physiology course. Disorders of the respiratory system are common and are encountered by nurses in every setting from the community to the intensive care unit. Expert assessment skills must be developedand used to provide the best care for patients with acute and chronic respiratory problems. In order to differentiate between normal and abnormal assessment findings, an understanding of respiratory function and the significance of abnormal diagnostic test results is essential. The understanding of Anatomy and Physiology in this system will help you relate the parts affected with the presenting signs and symptoms in relation to the pathophysiology.. The respiratory system is the biological system of a human being that introduces respiratory gases to the interior and performs gas exchange. The respiratory system provides the route by which the supply of oxygen present in the atmospheric air gains entry to the body and it provides the route of excretion of carbon dioxide.It comprises of the airway (nose, pharynx, larynx, bronchus and bronchioles) lungs and the respiratory muscles.

The organs of the respiratory system are:

- Nose
- Pharynx
- Larynx
- Trachea
- Two bronchi (one bronchus to each lung)
- Bronchioles and smaller air passages
- Two lungs and their coverings, the pleura, muscles of respiration — the intercostal muscles and the diaphragm.



FigureTableFigureTable 1: The respiratory organsFigure

The primary function of the respiratory system is to supply the blood with oxygen in order for the blood to deliver oxygen to all parts of the body. It also removes carbon dioxide through expiration.

The nose serves as the entry and exit point for oxygen and carbon dioxide respectively. The airways are passages where oxygen passes when inhaled to reach the lungs where exchange of gases takes place by the lungs. As the air breathed in moves through the air passages to reach the lungs, it is warmed or cooled to body temperature, moistened to become saturated with water vapour and 'cleaned' as particles of dust stick to the mucus which coats the lining membrane.Blood provides the transport system for these gases between the lungs and the cells of the body. Exchange of gases between the blood and the lungs is called external respiration and that between the blood and the cells internal respiration

Activity3.1
List the respiratory system organs and state the function of each.

3.3 The role of the nurse in Investigations and Procedures of Respiratory Disorders

The nurse has an important role to play in investigations and procedures that are done on the patients. The following are some of the duties which are carried out by the nurse in investigations and procedures:

Common investigations and procedures of the respiratory disorders include the following:

Endoscopy- Bronchoscopy

Bronchoscopy is a direct inspection and examination of the larynx, trachea, and bronchial using a flexible fiberoptic tube (bronchoscope). The procedure is done to diagnose and treat some conditions of the larynx, trachea and bronchial tree. The diagnostic purpose includes:

- To examine tissues or collect specimen
- To determine the location and extent of the pathological processes and obtain a tissue sample for diagnosis.
- To determine if tumour can be removed surgically.
- To diagnose bleeding sites (haemoptysis).

Therapeutic bronchoscopy can be used:

- To remove foreign bodies
- To remove secretions obstructing the tracheobronchial tree when patient can't manage to clear them.
- To treat post-operative atelectasis
- To destroy and excise lesions.

The procedure is done under local anaesthesia but can also be done under general anaesthesia.

X-rays

X-rays; uses electromagnetic radiation to make images. The image is recorded on a film – called radiograph.

X-rays can detect abnormal fluids, tumours, foreign bodies and other pathological conditions. A chest x-ray may reveal an extensive pathologic process in the lungs in the absence of symptoms. Chest x-ray is usually taken after full inspiration (a deep breath) because the lungs are best visualized when they are well aerated and also when the diaphragm is at its lowest level and the largest expanse of lung is visible**Blood gas analysis**

Blood gas analysis tests measure the concentration of oxygen and carbon dioxide within arterial blood. It also measures the acidity of the blood. These tests when done constantly can guide the therapy in respiratory failure and severe asthma (Kumar and Clark, 2005). Arterial blood gas analysis evaluates the effectiveness of the lungs in delivering oxygen and elimination of carbon dioxide from it (lungs). The test also show how well the lungs and kidneys interact to maintain normal blood pH (acid base balance).

Arterial oxygen saturation (SaO2) can be measured using an oxymeter. The oxymeter measures differential absorption of light by oxy- and deoxyhaemoglobin and measures the saturation to within 5% of that obtained blood gas analysis.

Respiratory function tests

Respiratory tests involve assessing for airflow limitation. The tests include:

Peak expiratory flow rate (PEFR)

This is done through the use of a peak flow meter. The patient is instructed to take full inspiration to total lung capacity and then blow out forcefully into the peak flow meter which is held horizontally.

Spirometry

The spirometer measures the FEV and the forced vital capacity (FVC). The technique involves a maximum inspiration followed by a forced expiration into the spirometer. The volume of air inhaled or exhaled and the length of each time breaths take place are recorded and analysed.

Sputum

Sputum is a material which is expelled from the airway passages through the mouth and consists of mucus, saliva and microorganisms. When the respiratory tract has a disorder the sputum can be purulent or can contain blood. Sputum examination is used for diagnosis. It is obtained for analysis to identify pathogenic organisms and to determine the presence of malignant cells. It may also be used to assess for hypersensitivity states in which there is an increase in eosinophils.

Sputum specimen can be obtained by asking the patient to

- Clear the nose and throat before expectoration.
- Rinse mouth before to decrease contamination of the sputum.
- Cough after taking a few breath rather than spitting.
- In cases where sputum cannot be raised spontaneously, the patient often can be induced to cough deeply by breathing an irritating aerosol of supersaturated saline, propylene glycol, or some other agents.
- Sputum can also be collected by endotracheal aspiration, bronchoscopic removal, bronchial brushing, transtracheal aspiration and gastric aspiration—usually for tuberculosis organisms.

Sputum specimen can also be inspected for colour such as green which indicates presence of infection or allergy and presence of blood may suggest neoplasm or pulmonary infarct.

Thoracentecis

This is a procedure which is performed to obtain a sample of fluid from the plural cavity. The sample obtained can be used for diagnostic or therapeutic purposes where fluid is drained to relieve shortness of breath.

Biopsy (Pleural And Lung)

This refers to excision of the small tissue from the lungs and pleural cavity for examination. Pleural biopsy is performed when there is presence of exudates to identify the cause and also in cases where culture is needed to identify tuberculosis or fungi (Brunner and Sadderth).
Lung biopsy is done to identify nature of the lesions in the lungs. The procedure can be done under local anaesthesia using bronchoscopy.

The role of a nurse in the above investigations and procedures are:

- To explain the procedure to the patients and what they are expected to do before the procedure(starving, bathing etc)
- To prepare the patient mentally and physically.
- To obtain signed consent for invasive procedures like thoracentesis and biopsy
- To instruct the patient on how to produce a good specimen, for instatnce the patient has to cough deeply to produce a quality sputum specimen.
- To prepare the equipment to be used during the procedure.
- To comfort the patient during the procedure.
- To assist the doctor during the procedure.
- To make the patient comfortable during and after the procedure and to make the appropriate observations of the patient following the investigation, to record them and report any irregularities to the senior staff e.g. ward in-charge or doctor.
- To remove the used equipment and prepare it for use again.
- To instruct patient to undress, put on the gown and remove any metal between neck and waist for radiological test.

To administer pre- medication before undergoing biopsy
To ensure that specimen is correctly labelled in suitable containers and are sent to the laboratory
You have now learnt the investigations and procedures of the respiratory disorders and now we are moving on to the disorders affecting the Respiratory System Let’s see how they can be applied in the conditions described below.

3.4 management of a patient with Upper Respiratory Tract Disorders

coryza

Coryza also known as common cold or viral rhinitis is a viral infection of the lining of the nose, throat, sinuses and airways which is self-limiting (Brunner and Suddarth)

Cause

Several viruses can cause coryza but the most common cause is the rhinovirus.

Mode of transmission

It can be transmitted by direct contact or by inhalation of airborne droplets; indirectly by hands and articles freshly soiled by discharges of nose and throat of infected persons.

Pathology

The mucous membranes of the nose, pharynx and tonsils become inflamed and swollen. A mucoid fluid is secreted and openings into sinuses may be blocked and patient presents with the following.

Signs and symptoms:

- Runny nose and lacrimation (excessive secretion of tears)
- Nasal discharge
- General body malaise lasting 2-7 days
- Nasal congestion
- Sneezing
- Sore throat
- Body chills
- Headache
- Coughing

Diagnosis

It is based on signs and symptoms. Tests are not necessary unless complications develop.

Treatment

There is no specific treatment. Management consists of symptomatic therapy and the following measures can help:

- Offer plenty of fluids
- Offer the patient salt gaggles (1 teaspoon in 1 L. warm water)
- Anti-inflammatory drugs such as aspirin 600mg tds for 3 daysAntihistamines piriton 4mg bd for 3 days

- Decongestants to dry up secretions.
- Nose drops eg. Ephedrine for adults--normal saline for children
- Increased intake of vitamin C
- Bed rest

Complications- may spread to the lower respiratory tree

- Sinusitis
- Otitis media (common, especially in children, due to blockage of eustachian tube and abscess formation in middle ear)
- Tonsillitis
- Bronchitis
- Pneumonia (more common in infants and the elderly)

Health Education

- A day or two at home helps limit the spread of infection to others
- Adise the patient to cover the mouth and nose when coughing or sneezing to prevent the spread of infectionProtect infants, immuno-deficient or debilitated patients from contact with URI's.
- Frequent hand washing is important to prevent the spread of infection.

Severe Acute Respiratory Syndrome

This is a serious respiratory syndrome which is characterized by headache, sneezing and fever.

Cause

It is caused by corona virus

Incubation Period

The incubation period is 2 to 10 days.

Mode of transmission

It is a droplet infection, which is spread by air travellers. The spread of the virus is promoted when people are in a closed environment.

Signs and symptoms

- Temperature of more than 38 degrees
- Sneezing
- Dry cough
- Loss of appetite and
- Loss of weight.
- General body malaise
- Headache
- Joint pains

Diagnosis

- History of contact
- Chest X-ray may show some patchy infiltrates or may have consolidation on one side.
- Full blood count will show raised white blood cells
- Throat or nasal swabs to isolate the causative organism

Treatment

- Treatment is symptomatic
- Analgesics such as panadol for pain
- Nasal drops
- Oxygen
- Tracheostomy if there are some spasms
- Intubation
- Broad – spectrum antibiotics is used to prevent secondary bacterial infection.
- Isolate patient
- Antivirals
-
- Swine Flu
- Swine flu is a highly contagious form of human influenza caused by a virus identical or related to a virus formerly isolated from infected swine.
-

- Cause

- Swine flu is caused by a mutated strain of influenza virus called ‘novel’H1N1 influenza virus. The virus evolved as a mix of genes from swine, bird and human viruses. It is easily transmissible among humans.
- Mode of Transmission
- Inhalation of infected droplets that have been coughed or sneezed out by an infected person
- Having direct contact with an infected person’s secretions - nasopharyngeal secretions.

- Risk factors
- People at risk of swine flu include:
- Those with lowered immunity - HIV/AIDS, malignancies, children, chronic diseases.
- Health care professionals
- Travellers or recent contact with those with swine flu.
- Signs and Symptoms
- Symptoms are similar to most influenza infections:
- Fever of 38˚C or more.
- Cough
- Sore throat
- Nasal secretions
- Fatigue and headache
- Diagnosis
- Presumptively clinically by the patient’s history of association with people known to have the disease and the clinical presentation.
- Laboratory test is the only confirmatory test i.e. a nasopharyngeal swab is collected and the particular antigens associated with the virus are identified.
- Management
- Treatment is supportive
- Bed rest and steam inhalation relieves the symptoms.
  Encourage patient to take a lot of oral fluids to maintain hydration
- Ephedrine 2 -3 drops instilled in each nostril or on nasal packIt is a nasal decongestant.
- Side effects include palpitations, insomnia, dizziness and euphoria.
- Nursing implications; patient not to drive or operate machines when on treatment.
- Amantadine 100mg BD. An antiviral which prevents or reduce the effects of swine flu if taken within 48 hours of onset of symptoms.
- S/E - depression, fatigue, peripheral oedema and orthostatic hypotension.
- Nursing implications – instruct patient to move slowly when changing position to avoid hypotension, administer drug several hours before bed time to avoid insomnia.

- Severe infections in some patients may require additional supportive measures such as ventilation support by use of oxygen machine and treatment of other infections like pneumonia that can occur.

- Prevention of swine flu
- Routine vaccination for all people aged more than 6 months is the best way to prevent novel H1N1 swine flu.
- Quarantine of virus infected people until they become non infectious (about 7-10 days after flu symptoms abate)
- Infected people can wear surgical face masks to reduce the amount of droplet spray from coughs and sneezes.
- Proper disposal of contaminated tissue.
- Kill or inactivate the virus before it reaches a human cell by hand washing, use of alcohol based hand sanitizers.
- Avoid crowds, parties and especially people who are coughing and sneezing

Hay Fever

Definition

Hay fever is an atopic allergic inflammation of the mucous membrane of the nose.

Causes

- MicroorganismsAllergens such as Dust, animal dander, foods such as onion, gallic, as well as use of sprays, perfumes etc.
- Pollens of flowers are mostly the causative agents and is usually seasonal.

Signs and Symptoms

- Sneezing
- Headache
- Rhinorrhoea
- Nasal congestion
- Nasal examination reviews pale edematous turbinates
- Fever/ Chills
- Coughing
- Wheezing
- Loss of appetite
- Red eyes

Treatment

- Antihistamines
- Antipyretics
- Decongestants

IEC

Avoid allergic agents
Advise adequate intake of fluids

Laryngitis

Laryngitis is the inflammation of the larynx.

Causes

- Virus (most common) eg. Measles, influenza, common cold. Bacterial invasion may be secondary.
- Polyps and cancer of the larynx
- Allergy
- Voice abuse
- Exposure to dust, chemicals and other pollutants.
- Smoking tobacco

Signs and symptoms

- Hoarseness of voice
- Loss of voice (aphonia)
- Sore throat
- Dry cough

Treatment

- Complete rest of the voice and no smoking.
- Cold steam or aerosol therapy
- Antibiotics only if bacterial origin

Complications

- Laryngeal edema with obstruction of airways, making endotracheal tube or tracheotomy necessary.
- Sepsis
- Meningitis
- Peritonsillar abscess
- Otitis media

- Sinusitis

Health education

- Adequate rest and good nutrition
- Avoid over-using the voice (shouting, loud singing)

Pharyngitis

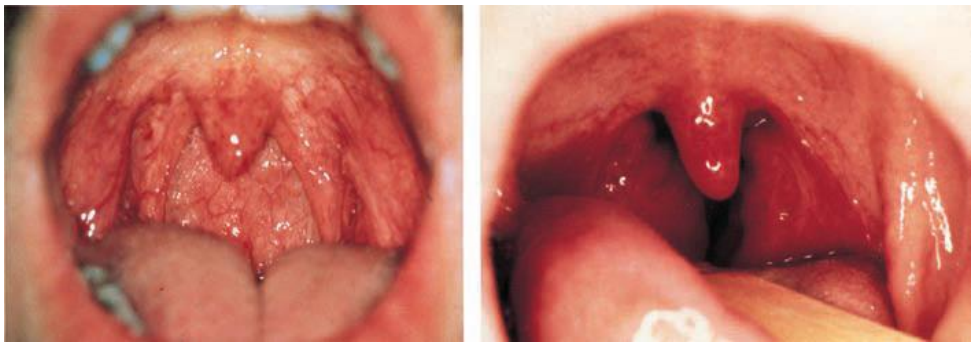
Pharyngitis is a sudden painful inflammation of the pharynx, the back portion of the throat that includes the
posterior third of the tongue, soft palate, and tonsils. It is commonly referred to as a sore (Smeltzer etal, 2010).

Cause

- Viruses adenovirus, influenza virus,, Epstein-Barr virus, and herpes simplex virus
- Bacteria - most common cause is group A beta-hemolytic streptococcus

Pathophysiology

The body responds by triggering an inflammatory response in the pharynx. This results in pain, fever, vasodilation, edema, and tissue damage, manifested by redness and swelling in the tonsillar pillars, uvula, and soft palate. A creamy exudate may be present in the tonsillar pillars.



(Smeltzer etal, 2010)

Signs and symptoms

- Enlarged cervical lymph nodes
- Fever
- General body malaise
- Pain
- Difficulty in swallowing

Diagnosis

Rapid screening tests for streptococcal antigens. The tests include:
Latex agglutination (LA) antigen test and

- solid-phase enzyme immunoassays
- (ELISA),
- optical immunoassay (OIA),
- streptolysin titers,
- Throat cultures are used to determine the causative organism,after which appropriate therapy is prescribed
- Nasal swabs
- Blood cultures may also be necessary to identify the causative organism.

Treatment

- If the cause is viral, supportive treatment is needed.
- Antibiotics such as penicillins, erythromycin and clarithromycin for 10 days if cause is bacterial.
- Ant inflammatory such as aspirin.
- Intravenous fluid
- Warm saline gaggles***Nutritional Therapy, A*** liquid or soft diet is provided during the acute stage of thedisease, depending on the patient's appetite and the degree of discomfort that occurs with swallowing. Cool beverages, warm liquids, and flavored frozen desserts such as Popsicles are often soothing.
- Bed rest
- Mouth care

Complications

- Nephritis
- Rheumatic fever
- Rheumatic heart

Tracheitis

Tracheitis is inflammation of the trachea causing pain in the chest, with coughing. It often occurs together with laryngitis and acute pharyngitis.

Cause

It is caused by viruses

Signs and symptoms

- Sore throat
- Pain on swallowing
- Hoarse voice

Irritating cough**.**

Treatment

- Symptomatic relief will include
- Steam inhalations,
- Gargles
- Simple linctus
- Rarely antibiotics
- **Nursing Care Of A Patient With Upper Respiratory Infections**

Maintaining a Patent Airway

An accumulation of secretions can block the airway in patients with an upper airway infection. As a result, changes in the respiratory pattern occur, and the work of breathing increases to compensate for the blockage. The nurse can implement several measures to loosen thick secretions or to keep the secretions moist so that they can be easily expectorated.Increasing fluid intake helps thin the mucus. Use of room vaporizers or steam inhalation also loosens secretions and reduces inflammation of the mucous membranes. To enhance drainage from the sinuses, the nurse instructs the patient about positioning; this depends on the location of the infection or inflammation. For example, drainage for sinusitis or rhinitis is achieved in the upright position. In some conditions, topical or systemic medications, when prescribed, help relieve nasal or throat congestion

Promoting Communication

Upper airway infections may result in hoarseness or loss of speech. The nurse should instruct the patient to refrain from speaking as much as possible and, if possible, to communicate in writing instead. Additional strain on the vocal cords may delay full return of the voice. The nurse should encourage the patient and family to use alternative forms of communication,such as a memo pad or a bell to signal for assistance.

Encouraging Fluid Intake

Upper airway infections lead to fluid loss. Sore throat, malaise, and fever may interfere with a patient's willingness to eat and drink. The nurse provides a list of easily ingested foods to increase caloric intake during the acute phase of illness.These include soups, pudding, yogurt, cottage cheese,high protein drinks, and Popsicles. The nurse encourages the patient to drink 2 to 3 L of fluid per day during the acutestage of airway infection, unless contraindicated, to thin thesecretions and promote drainage

Promoting Comfort

URIs usually produces localized discomfort. In sinusitis, pain may occur in the area of the sinuses, or a general headache may be produced. In pharyngitis, laryngitis, or tonsillitis, a sore throat occurs. The nurse encourages the patient to take analgesics, such as acetaminophen with codeine, as prescribed, to relieve this discomfort.

Take Note 4.1

Patients with persistent hoarseness or Stridor must be thoroughly investigated and this will include laryngoscopy to exclude laryngeal cancer, underlying lung or Tuberculosis should also be excluded.

You are now moving on to the lower part of the respiratory system.

3.5 5 Management of a patient with Lower Respiratory Tract Disorders

Asthma

INTRODUCTION

1. Asthma is a chronic inflammatory disease of the airways that causes hyperresponseveness, mucosal edema and mucus production. This inflammation ultimately leads to recurrent episodes of asthma symptoms such as cough, chest tightness, wheezing and dyspnoes. Asthma can occur at any age and it is the most common chronic disease of childhood Asthma differs from other obstructive lung diseases in that it is largely reversible either spontaneously or with treatment. Allergy is the strongest predisposing factor for asthma. Chronic exposure to airway irritants or allergens also increases the risk for developing asthma.

DEFINITIONS

2. Asthma is a chronic inflammatory disorder of the airways that is characterized by an exaggerated bronco constrictors response to a wide variety of stimuli (Monahan, 2007).

3. **Classifications Of Asthma**
   - **Extrinsic asthma: -** This is the type of asthma with a definite external cause. It occurs mostly in atopic individuals who show positive skin-prick reaction to common inhalant allergens. It starts in childhood and is caused by allergens like pollen, dust, animal dander, feathers, foods etc. patients usually have a history of asthma or allergies in the family, past medical history of eczema or allergic rhinitis is also common.

- **Intrinsic asthma**: - This occurs where no causative agent can be identified. It's non allergic and occurs secondary to respiratory tract infections. It develops in adulthood with no history of asthma in the family.

Common Factors That May Trigger An Asthmatic Attack

1. ***Environmental factors***
   - Change in temperature especially cold air
   - Change in humidity dry air
2. ***Atmospheric pollutants***
   - Cigarette and industrial fumes
   - Ozone sulphur dioxide
   - Formaldehyde
3. ***Strong odors***
   - Perfume
4. ***Allergens***
   - Feathers
   - Animal dander
   - Dust mites
   - Mold
   - Salads shellfish
   - Fresh and dried fruits
5. ***Exercise***
6. ***Stress and emotional upset***

7. ***Medication***
   - Nosteroidal anti-inflammatory Drugs(NSAIDS) Aspirin
   - Beta blockers
   - Cholinergic drugs
8. ***Chemicals***
   - Toluene
   - Paints
   - Rubber
   - Plastics

Pathophysiology Of Asthma

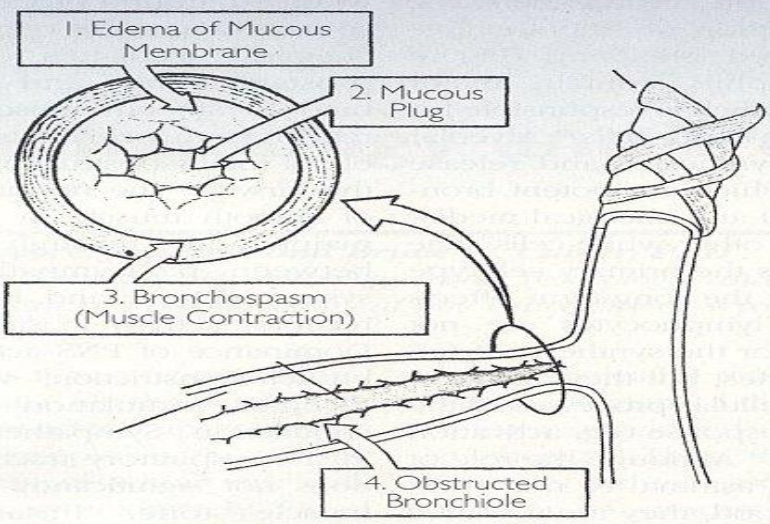
Bronchoconstriction- When the patient inhales a substance to which he/she is hypersensitive, allergens interact with the Immune Globuline IgE on the mast cells. This causes degranulation of the mast cells in the bronchial walls leading to rupture of mast cells releasing chemical mediators such as histamine, bradykinin, leukotrienes and prostaglandin. These mediators of inflammation lead to bronchoconstriction, increased vascular permeability and leakage contributing to oedema

and mucous secretion. Consequently mucosal thickening and airway swelling, thus the airway becomes more rigid and interferes with air flow. Dyspnoea results as well as wheezing due to mucus secretion and bronchospams.

Airway oedema- In persistent asthma a chronic and complex response ensues, which is characterized by an influx of numerous inflammatory cells, the transformation and participation of airway structural cells and the secretion of an array of cytokins, chemokines and growth factor. These results in edema, inflammation, mucus hypersecretion and the formation of mucus plugs, as well as structural changes such as hypertrophy and hyperplasia of the airway smooth muscle stimulating the mucous membrane to secrete excessive mucus, further narrowing the bronchial lumen.

Airway remodeling-With increasing severity and chronicity of disease, permanent structural changes can occur in the airway these are associated with a progressive loss of lung function that is not prevented by or fully reversible by current therapy. These structural changes can include thickening of the sub-basement membrane, subepithelial fibrosis, airway smooth muscle hypertrophy and hyperplasia, blood vessel proliferation and dilation, and mucous gland hyperplasia and hypersecretion. Goblet cells secrete viscous mucus that is difficult to cough up. Mucus fills the lung bases, inhibiting alveolar ventilation. Blood, shunted to alveoli in other lung parts still can't compensate for diminished ventilation leading to respiratory acidosis. Inflammation of the bronchial walls may also injure the epithelium, thereby stimulating nerve endings and initiate neural reflexes that further aggravates and propagate the broncho spasms. This leads to fixed narrowing of the airway and a reduced response to broncho dilators.

Figure 1. Diagram showing pathophysiologic processes that cause obstruction in the airways in patients with asthma



(Gould B. *Pathophysiology for the Health-Related Professions.* Philadelphia, WB Saunders, 1997)

Clinical Manifestations

- Wheezing respirations which is whistling sound made during expiration when the airways are blocked or compressed.
- Expiration is forced and prolonged due to broncho spasms, hyper inflated lungs and trapped alveolar air.
- Dry cough or cough which may produce thick, clear or yellow sputum.
- Chest tightness due to bronchial constriction
- Extreme anxiety due to breathlessness.
- Sweating(diaphoresis)- results from labored respiration
- Dyspnoea due to thick mucus, mucosal oedema and smooth muscle spasm causing obstruction of small airways resulting in labored respiration
- Orthopnoea – dyspnoea that is relieved in the upright position. Patient fails to lie flat but rather maintain a three point position.
- Peripheral cyanosis which is due to reduced oxygen in the blood and this may indicate the onset of life- threatening status asthmaticus and respiratory failure.
- Hypoxia due to reduced oxygen circulating in the blood as result of reduced gaseous exchange.
- Hypercapnoea – greater than normal carbon dioxide in the blood which results from difficulty in expirations as a result of broncho spasm.
- Tarchycardia in attempt to compensate for hypoxia and hypercapnoea
- If there is no response to treatment there is exhaustion

Management

Aims

1. To prevent chronic symptoms
2. To maintain near normal pulmonary function
3. To prevent complications.

Investigations

- Typical clinical presentation and past history
- History of exposure to specific allergens
- Lung function test to establish a degree of impairment, degree of obstruction and its reversibility and also to establish baseline ventilator function.
- Chest x-ray will show possible hyperinflation with areas of focal atelectasis.
- Skin sensitivity test done to identify allergen or other triggers responsible for onset of asthma symptoms
- Arterial blood gas analysis- obtained to identify presence of mild to severe hypoxemia and mild to severe respiratory acidosis.

Primary Survey / Care During An Acute Asthmatic Attack

An asthmatic attack should be treated as quickly as possible to open airways.

A – Airway- in an asthmatic attack secretions tend to become viscous and can plug airway.

Intervention – the nursing role in improving breathing patterns and gas exchange, is to help patient assume a position of comfort, administer medications as ordered and monitor for both therapeutic and adverse effects of medications.

Patient should be nursed in an upright position well supported with pillows or lean forward on the cardiac table. Administer nebulised salbutamol 5gm or a short acting medication such as aminophyline 250mg IV bolus over 10minutes or 750mg in 1 liter 10% dextrose over 8hours, and 50% dextrose to prevent hypoglycaemia as these patients lose a lot of energy due to exhaustion caused by labored breathing. Administer steroids such as hydrocortisone 200mg intravenously to reduce local oedema.

B – Breathing – assess for ventilation by looking at the chest movements associated with breathing and also listen or feel for air being expired through the nose and mouth. Patient may present with slow laborious wheezing sound on expiration, there will be use of accessory muscles such as abdominal muscles for breathing. Expiration is always more strenuous and prolonged than inspiration which forces the patient to sit upright and use every accessory muscle.

Intervention –administer humidified oxygen by nasal cannular at 2 liters /minute to ease breathing, later adjust oxygen according to the patient's vital functions and ABG measurements.

C – Circulation will not be altered but the partial oxygen pressure due to altered gaseous exchange in the lungs. The patient will have tachycardia due to impaired gaseous exchange in the lungs. The pulse will be fast and thread.

Intervention – commence intravenous fluids to rehydrate the patient thus improving circulation continue with oxygen therapy.

Treatment

- **Beta-adrenergic receptor agonist –**are the best drugs for relieving sudden attacks of asthma and prevents attacks that might be triggered by exercise.
- Drugs used include rapid acting bronchodilators such as ;
  - *Salbutamol* - this is the fastest and most effective, hence remain the drug of choice. It is given through a nebulizer or orally 2gm TDS. Action – selectively stimulate beta receptors producing bronchodilatation. Side effects – tachycardia, Bp changes, nervousness, palpitation, muscle tremors, nausea, vomiting, insomnia, dry mouth, headache. Nursing implication – should not be used in patients with angina or cardiac disorders. Encourage the patient to take the drug even when feeling well.
  - *Metaproterenol* – stimulates beta adrenergic receptors, producing bronchodilatation. Increases mucocilliary clearance. The rest as above.

- Aminophyline given intravenously. Action – relaxation of bronchial smooth muscle and improve contractility of fatigued diaphragm. Side effects – tachycardia, Bp changes, arrhythmias, anorexia, nausea and vomiting, nervousness, irritability, headache, muscle twitching, epigastric pain, diarrhea, palpitation, insomnia. Nursing implication- instruct patient to lie down if they experience dizziness.

- Corticosteroids
  - *Predinisolone* given orally. Action- it has anti-inflammatory and immunosuppressive effects. Decrease edema in bronchial airway thus decreasing mucus secretion.
    Side effects- skin changes, osteoporosis, increased appetite, obesity, immunosupression, catabolism, muscle weakness. Nursing implication – Advise patient to take drug on alternative days as it reduces side effects; drug should be taken with food or milk in the morning as it causes peptic ulcers.
- Long acting bronchodilators- epinephrine given subcutaneously, usually in emergency treatment of an acute reaction. Dose 0.2- 0.5 as a single dose. Side effects- headache, dizziness, palpitation, tremors, restlessness, hypertension and tachycardia.
- Oxygen therapy 4-6 liters
- Fluid therapy
- Antibiotics such as Amoxicillin 500mg TDS for 5 to 7 days.

Subsequent Care

Environment – Nurse patient in a quiet, clean environment near to Nurse's bay for ease observation. The room should be well ventilated and free from dust. It should contain all rescustative equipment such as oxygen cylinder, suctioning machine etc.

Position – Place the patient in the semi fowler's position and encourage diaphragmatic breathing to allow enough air intakes.

Psychological care- Create a therapeutic relationship with the patient so that the patient can have confidence in you. Reassure the patient during an asthmatic attack. Provide comfort by being with the patient. Explain the disease process, the cause of the wheezing and labored respiration to allay anxiety. Allow the patient to verbalize his fears to allay anxiety. Explain every procedure and machines that are being used to promote cooperation. Explain the use the oxygen machine to alley fear and anxiety. Involve the relative in the care and explain what is happening to the patient.

Hygiene – Wipe the patient frequently to remove sweet as the patient sweets a lot due to labored breathing. Change linen whenever soiled to make patient comfortable.

Fluids and nutrition- Give plenty of fluids to combat dehydration and loosen secretions. **Elimination –** Monitor the urine output to ascertain the renal function.

Exercises – Help patient and the family to perform diaphragmatic breathing. Encourage the patient to perform relaxation exercises as needed. Plan activity and rest to minimize patient's energy expenditure as activity increases metabolic rate and oxygen requirements.

Medication – Administer drugs as prescribed and observe for any adverse effects.

Information Education And Communication

- Teach the patient how to use an oral inhaler and caution him about the possible adverse reactions associated with the medications he is receiving.
- Show patient how to breathe deeply. Instruct him how to cough secretions accumulated overnight.
- Teach the patient and the family to avoid known allergens and irritants such as smocking, dust perfumes, fur and cold weather etc.
- Emphasize the importance of taking only prescribed drugs as certain drugs such as aspirin may precipitate an asthmatic attack.
- Urge the patient to be drinking plenty of fluids at least 3 liters in 24hrs to help loosen secretions and maintain hydration. Encourage the patient to eat a well balanced diet to prevent respiratory infection and fatigue.
- Explain the importance of review dates
- Teach the patient signs and symptoms of an impending asthmatic attack and encourage them to seek medical attention as soon as possible.

Complication

1. ***Status asthmaticus***- This is a severe asthmatic attack which cannot be controlled with usual medications. This arises when impaired gas exchange and heightened airway resistance increase the work of breathing. Symptoms of acute asthmatic attack continue despite measures to relieve them.
2. ***Respiratory*** failure- This is the impairment of the lung's ability to maintain balance between oxygen and carbon dioxide.
3. ***Tention pnemothorax*** -This occurs due to rupture of the sub pleural bleb
4. ***Cardiac arrest***- Occurs secondary to respiratory failure
5. ***Emphysema*** **–**Iirreversible accumulation of air in the alveolar spaces due to repeated asthmatic attacks which results in decrease in total breathing capacity.
6. ***Atelectasis***- lung collapse due to accumulation of air in the alveoli.

Bronchitis

Bronchitis is the inflammation of the bronchi in the lower respiratory tract usually due to infection (Lewis *et al 2004*).

Causes

- Microorganisms – viruses and bacteria.
- Extension of infection from the trachea

- Traumatic injuries
- Excessive cold air
- Smoking
- Over exposure to industrial fumes

Types

There are 2 types of bronchitis and these are:

- Acute bronchitis
- Chronic bronchitis

Acute bronchitis

This is the inflammation of the bronchial tree with minimal changes in the parts affected. Acute bronchitis is characterized by the development of a cough or small sensation in the back of the throat, with or without the production of sputum.Acute bronchitis is most often caused by viruses that infect the epithelium of the bronchi, resulting in inflammation and increased mucus

Causes

Viruses such as:

- *Rhinoviruses*
- *Coronaviruses*
- *Adenoviruses*
- *Metapneumovirus*
- *Parainfluenza virus*
- *Respiratory syncytial virus*
- *Influenza.*

Bacteria such as

- *Mycoplasma pneumoniae,*
- *Chlamydophila pneumoniae,*
- *Bordetella pertussis, Streptococcus pneumoniae,*

Signs and symptoms

- Dry cough which later becomes productive
- Chest pains
- Haemoptysis (coughing up blood stained sputum)
- General body malaise
- Dyspnoea

- Fatigue
- Sore throat
- Cyanosis
- Nasal congestion

Chronic Bronchitis

Chronic bronchitis, a type of chronic obstructive pulmonary disease (COPD), is defined by a productive cough that lasts greater than three months each year for at least two years in the absence of other underlying disease. Chronic bronchitis usually develops due to recurrent injury to the airways caused by inhaled irritants. Cigarette is the most common cause, followed by exposure to air pollutants such as sulfur dioxide or nitrogen dioxide, and occupational exposure to respiratory irritants. Individuals exposed to cigarette smoke, chemical lung irritants, or who are immunocompromised have an increased risk of developing bronchitis (Stanley, loeb, etal, 1992).**Causes of chronic bronchitis Causes**

Most cases of chronic bronchitis are caused by:

- Smoking cigarettes or other forms of tobacco.
- Chronic inhalation of air pollution or irritating fumes or dust from hazardous exposures in occupations such as coal mining
- Grain handling
- Textile manufacturing
- Livestock farming
- Metal molding may also be a risk factor for the development of chronic bronchitis.

Pathology

Chronic bronchitis results in hypertrophy and hyperplasia of the bronchial mucous glands following the exposure. This leads to increased mucus production, cilliary damage, squamous metaplasia of the columnar epithelium, and chronic leukocystic and lymphocystic infiltration of bronchial walls.Hyper secretion from the goblet cells blocks the free movement of cilia, which normally sweep dust irritants and mucus from the airway. This results in the disappearance of cilia and consequently alters the function of the alveolar macrophages, leading to increased bronchial infections. As a result, the airway stays blocked, and mucus and debris accumulate in the respiratory tract thus narrowing the airway lumen leading to diminished airflow. Greater resistance to airflow increases the work of breathing. The epithelial layer becomes ulcerated and when the ulcer heals the walls thicken leading to further narrowing of the airways (Stanley, loeb, etal, 1992).

Signs and symptoms

- Chronic cough due to repeated irritation
- Copious sputum which is thick and brown in colour resulting from increased production of mucus from the goblet cells.
- Haemoptysis due to bleeding which is taking place in certain parts.
- Dyspnoea due to constriction of the bronchiole tree resulting from inflammation and presence of mucus.
- Chest pains due to reduced air spaces.
- Cyanosis due to hypoxia
- Fever due to fever.
- Wheezing due to accumulation of mucus in the airway

Investigation for bronchitis

- ✓ History and physical examinations
- ✓ Chest x – ray may show hyperinfiltration and increased bronchovascular markings.
- ✓ Pulmonary function test demonstrate increased residual volume, decreased vital capacity and forced expiratory flow and normal static compliance and diffusing capacity.
- ✓ Arterial blood gas analysis displays decreased PaO_2 and normal or increased $PaCO_2$
- ✓ Sputum culture may reveal many microorganisms and neutrophils
- ✓ Eletrocardiogram may detect atrial arrhythsmias

Treatment

- Antibiotics if the cause is bacterial e.g. Ampicillin
- Anti-inflammatory drugs such a prednisolone
- Cough expectorants e.g. Amonium chloride
- Steam inhalations to liquefy secretions
- Warm, well ventilated room
- Light diet with plenty of fluids

Nursing Care Of A Patient With Bronchitis

Aims:

- To maintain clear air way
- To relieve dyspnoea
- To prevent complications.

Environment

Nurse patient in a well ventilated room near to the nurse's bay for ease observation. Ensure that the room has all the necessary equipment and bed accessories such as oxygen cylinder, suctioning machine, drip stand, backrest etc.The environment should be quiet to promote rest. Humidify the room using the steam from boiling kettle to liquefy the secretions.

Position

Nurse patient in the semi fowler's position with the help of a backrest and pillows to aid in lung expansion and relieve dyspnea.

Psychological care

Explain the disease process to the patient and the family to allay anxiety .Answer the patient's questions and encourage him and the family to express their concerns about the illness. Involve the patient and the family in the care decisions. Refer them to other support services as appropriate.Explain to the patient the purpose of all the equipment especially the oxygen to allay anxiety and gain patient's cooperation. Explain every procedure done on the patient to enhance cooperation.

Observation

Observe the vital signs of life that is temperature, pulse, respiration and blood pressure. Observe the sputum for the consistence, quality, amount and colour. Observe the breathing pattern. Monitor the patient's response to treatment. Observe the side effects of drugs. Evaluate the changes in the respiratory function. Weigh the patient three times weekly and assess for oedema to rule complications like cor-pulmonale.

Nutrition

Provide the patient with a high calorie, protein rich diet to enhance healing**.** Offer small frequent meals to conserve the patient's energy and prevent fatigue**.** Ensure that the patient receives adequate fluids at least 3 liters a day to loosen secretions**.** Schedule respiratory therapy at least 1 hour before or after a meal**.**Provide mouth care after bronchodilator inhalation therapy

Elimination

Monitor and record intake and output to ascertain the renal functioning**..**Offer diet containing roughage to prevent constipation so that the patient does not put more pressure on the heart.

Hygiene

In the acute phase, bath the patient in bed to promote blood circulation and promote self esteem. Provide oral care to promote salivation. Bed should be well made and linen changed whenever soiled to promote comfort. Provide a clean sputum mug with the lid to prevent spread of infection. Provide the bin where the tissue should be disposed off to prevent spread of infection. Hand washing should be practiced every after handling secretions

Rest/ exercises

Create a quiet environment that will allow the patient to rest to promote recovery**.** Perform chest physiotherapy including postural drainage and chest percussion **.** Help the patient to alternate periods of rest and activity to conserve energy and prevent fatigue**.** Encourage daily activities and provide diversional activities as appropriate

Medication

Administer medications as ordered and note the patient's response to the drugs

Information education and communication

- Advise the patient to avoid crowds and people with known infections, and to obtain influenza and pneumococuss immunization.
- Teach the patient and family how to perform postural drainage and chest percussion. Instruct the patient to maintain each position for 10 minutes before a caregiver performs percussion and the patient coughs. Also teach the patient coughing and deep-breathing techniques to promote good ventilation and to remove secretions.
- Review all medications, including dosage, adverse effects, and purposes for the prescriptions. Advise him to report any adverse reactions to the doctor immediately.
- Encourage the patient to eat high-calorie, protein-rich meals and to drink plenty of fluids to prevent dehydration and help loosen secretions
- If the patient smokes, advise him to stop. Provide him with smoking-cessations lessons of counseling if necessary.
- Urge the patient to avoid irritatants such as automobile exhausts fumes, aerosol sprays, and industrial pollutants.
- Warn the patient that exposure to blasts of cold air may precipitate bronchospasm. Suggest that he avoids cold, windy weather or that he covers his mouth and nose with a scarf or mask if he must go outside.

If the patient takes theophylline (broncho dilator), warn him cigarettes or marijuana smoking significantly increases plasma clearance of theophylline. Also, patients who quit smoking should notify the doctor because they may

experience the onset of the adverse effects of higher blood levels of theophylline. **Complications**

- Bronchopneumonia
- With repeated infections of the lower respiratory tract
- Cor –pulmonale comes as a result of obstruction in the lungs
- Acute respiratory failure
- Pneumonia due to spread of infection
- Right sided heart failure due to increased pressure on the right side of the heart in order to push blood into the lungs.
- Atelectasis
  Bronciectasis due to chronic dilatation of the bronchi and destruction of the bronchial walls.

-

Bronhiectasis

This refers to chronic abnormal dilatation of the bronchi and destruction of bronchial walls.

Cause

- Microorganisms-viruses, bacteria
- Cystic fibrosis
- Obstruction (foreign body, tumor).
- Inhalation of corrosive gas

Predisposing conditions

- Measles
- Broncho-pneumonia,
- Chronic bronchitis
- Tuberculosis

Pathology- a chronic dilation of the bronchi often due to a blockage from past infection which is characterized by inflammation and leukocytic accumulation.

Signs and Symptoms

- Chronic cough with large amounts of purulent sputum
- Haemoptysis
- Clubbing of the fingers
- Loss of energy, appetite, and weight
- Fever and sweating at times

Diagnosis

- Bronchoscopy
- Chest x-ray
- Full blood count
- Pulmonary function test

Treatment

- Physiotherapy--breathing exercises; postural drainage
- High fluid intake - 3000 mls a day - to help liquefy secretions
- Antibiotics for specific infection
- Surgical removal of infected area - lobe (lobectomy) or lung (pneumonectomy)

Complications

- Bronchopneumonia
- Cor pumornale
- Right sided heart failure

Prevention

Avoid upper respiratory infections

Pneumonia

Definition

Pneumonia is an acute inflammation of the lung parenchyma.

Causative organisms

Bacteria - Streptococcus pneumonia, Haemophilus influenza, Staphylococcus aureus, Mycoplasma, and Pseudomonas aureginosa

Viruses – Cytomegalovirus, Respiratory syncytial virus
Fungi - Pneumocystis Jirovicii Pneumonia (PJP).
Protozoa: mycoplasma pneumonia

Predisposingfactors

Existing chest infection e.g. bronchitis

- Aspiration of gastric secretions
- Aspiration of infected mucus following URT
- Unconsciousness- impaired cough reflex
- Smoking - damage of the epithelial lining of the respiratory tract.
- Old age and children- due to lowered immune system.
- Inhalation of foreign material into the respiratory tract which later descends into the lower respiratory tract causing pneumonia
- Obstruction of the respiratory tract by growths e.g. cancer which leads to retention of fluids and secretions in the lung tissue this fluid eventually becomes infected
- Prolonged to cold which irritate the respiratory tract and reduces the ability of cilia to expel the foreign bodies including micro organisms. This raises the risk of micro organisms invading the respiratory tract

Classification

May be classified according to cause or anatomical structure affected

According To Anatomical Structure Involved

- Broncho pneumonia (Involving the bronchi)
- Lobar pneumonia (Involving the lobe)

According To Cause

Aspiration pneumonia (Due to inhaling fluids, food or vomitus into the airway)

- Hypostatic pneumonia (Due to immobility)
- Atypical pneumonia (caused by less common organisms)
- Typical pneumonia (caused by common such as Bacterial / viral / fungal)
- Respiratory tract

Types of pneumonia

Aspiration pneumonia

Results from entry of endogenous or exogenous substances into the lower respiratory tract:
Bacteria that normally resides in the upper respiratory tract.
Gastric contents, irritating gases or exogenous chemicals - Impair lung defenses causing inflammatory changes leading to growth of bacteria.

Hypostatic Pneumonia

Portions of the lungs not well ventilated develop the infection.
Occurs primarily in the aged or those debilitated by disease who lie in the same position.

Lobar pneumonia

Is the type of pneumonia that may be confined to a lobe or part of the lobe of the lung.

There is inflammation of the interstitial and the alveoli does not contain significant fluid but only protein rich hyaline membrane

It usually affects fit young healthy males.

Untreated, acute inflammation undergoes 4 stages which are:

Congestion - the affected lobe is heavy, red and oedematous. This is due to the outpouring of exudates in the alveolar.

Red hepatisation - Within a few days the lung assumes a liver like appearance. The alveolar spaces are filled with neutrophils, red cells and fibrin.

Grey hepatisation - the lung is now dry, gray and firm because the red blood cells are lysed and accumulation of fibrin.

Resolution- this follows in uncomplicated cases as the exudates are enzymatically digested and reabsorbed by the macrophages or coughed up.

Bronchopneumonia

It is the commonest form of pneumonia affecting mainly the very young, the elderly and the immunosupressed. This is the inflammation of the lung occurring in patches around the infected terminal bronchiole it may be confined to a small segment of the lung or it may wide spread to the whole lung or both lungs.Results from secondary to other infections e.g. measles, and may also result from aspiration of oral pharyngeal material into the airway.Patchy consolidation involving one or several lobe.

Pathophysiology

The invading organism provokes an overly exuberant immune response in the lungs. The small blood vessel (capillaries) becomes leaky, and protein-rich fluid seeps into the alveoli. This results in a less functional area for oxygen-carbon dioxide exchange in the alveoli. The patient becomes relatively oxygen deprived, while retaining potentially damaging carbon dioxide. The patient breathes faster and faster, in an effort to bring in more oxygen and blow off more carbon dioxide.Mucus production is increased, and the leaky capillaries may tinge the mucus with blood. Mucus plugs actually further decrease the efficiency of gas exchange in the lung. The alveoli fill further with fluid and debris from the large number of white blood cells being produced to fight the infection.

Consolidation, a feature of bacterial pneumonias, occurs when the alveoli, which are normally hollow air spaces within the lung, instead become solid, due to quantities of fluid and debris.

Pneumocystis JIROVECII pneumonia

Now called Jerovecii (PJP)

This type of pneumonia occurs in people with compromised immunity.

Major opportunistic infections in HIV/AIDS.

Fever, Shortness of breath and Dry cough are the common manifestations.

Pathophysiology

The causative organism is inhaled or spread from upper respiratory tract infection. It lodges in the alveoli where it multiplies.The organisms trigger the inflammatory process and aggregation of white blood cells.

This will cause local capillary leak, oedema and watery exudates .First 12-48 hours the alveoli appears reddish and lungs consolidate due to wide spread dilatation of pulmonary blood vessels.

The fluids which collect in and around the alveoli causing the walls to thicken and the patient will start coughing trying to clear the airway. The capillary leak and watery exudates will overflow and spread the infection to other areas of the lung. If the organisms get into the blood stream, then sepsis results causing fever. When organism enters the pleural cavity, empyema comes in leading to pleurisy.

Red blood cells and fibrin also move into the alveoli. These events reduce gas exchange; reduce the vital capacity and alveoli collapse leading to hypoxemia

1. Signs and symptoms
   Fever up to 39 to 40˚C accompanied with rigors and delirium.
2. **Severe chest pains** – over the affected lobe due to the inflammatory process and rubbing of the pleura against each other.
3. **Rapid respirations** of 25-45 per minute as a compensatory mechanism to the impaired gaseous exchange.
4. **Dry cough** which becomes productive after 24-48 hours with typical rusty sputum especially in lobar pneumonia. This is due to massive exudation and erosion of alveolar blood vessels. The sputum is thick and tenacious and often adheres to the side of the sputum mug
5. **Chest in drawing** commonly seen in children resulting from pleural rub
6. **Tachycardia** - As a compensatory mechanism by the body to increase oxygen supply in the blood and tissues.
7. **Restless** - Due to reduced oxygen perfusion to the tissues.
8. **Dyspnoea** due to impaired gas exchange related to consolidation of the lung.
9. **Cyanosis** in severe cases due to impaired tissue perfusion.
10. **Crepitations** with suppressed breath sounds are heard over the affected area due to consolidation

Investigations

1. X-ray - The area affected on X-Ray appears as an area of increased density. Pleural effusion can also be detected.
2. Sputum for M/C/S
3. Blood culture show the causative agent if bactraemia is present
4. Blood for FBC shows high neutrophil count.
5. Blood gas analysis to rule out any reduction in PO2 in arterial blood
6. Investigations
7. On auscultation there will be reduced are entry and crackling sound (rales) in the affected lung.
8. On percussion there will be dullness on the affected side due to consolidation of the underlying lung.

Medical management

Antibiotics based on culture e.g.

Benzyl penicillin 8-12 MIU in 4 divided doses IV/IM minimum of 5 days.

It is antibacterial in nature. S/E – allergic reactions, GIT disturbances. Implications – don't administer to penicillin allergic patients.

Ceftriaxone-Dose –Adult; 1-2g as a single dose or in 2 divided doses.

It is bactericidal.

Side effects; headache, leucopoenia tenderness at injection site.

Nursing implications; use cautiously in patients allergic to penicillin

Antipyretics - Acetylsalicylic acid 300 - 600mg tds x 3/7.

Oxygen therapy is also administered in cases of dyspnoea that is 2-5 litres per minute.

If the infection was fungal anti fungal have to be used e.g. Fluconazole 100mg bd 7-14 days

Broncho dilators e.g. ventolin 2-4mg tid

Rest to promote recovery

Balanced diet to boost the immunity thereby promote quick recovery

Give a lot of oral fluid to loosen the secretions and bring sputum up

Nursing Care

Aims

1. To relieve dyspnea
2. To promote quick recovery
3. To educate the patient on his condition
4. To promote a good nutritional status

5. To relieve the pain
6. To prevent complications

Environment

Patient should be nursed in a warm well-ventilated area. A fan may be provided when the patient is very febrile and perspiring a lot to provide comfort. Clean environment to prevent secondary infection.

Have the oxygen giving apparatus for use because patient is dyspnoeic. The drip stand in the room for IVI when need arises

Position

Nurse the patient in a semi fowler's position to promote lung expansion and increase gaseous exchange.

Encourage frequent change of position to promote drainage of secretions. Change the patient's position two hourly to prevent development of pressure sores .As the condition improves allow the patient adopt any position of comfort to promote rest.

Observations

Acute phase 4 hourly observations of vital signs progressing to 12 hourly as the condition stabilizes.

Check for Respirations for depth, rate and rhythm so as to rule out dyspnoea and tachypnoea. Temperature for fever, pulse for tachycardia. Vitals will help check if patient is improving. Signs of confusion indicative of hypoxia

Rest

Nurse the patient in a quiet room to promote rest. Play the radio at low volume to promote rest. Answer all phone calls promptly to prevent disturbing the patient there by promote rest. Do related procedures in blocks to promote rest. Administer prescribed analgesics in order to promote rest. Ensure that squeaking trolleys a oiled to prevent noise and there by promote rest

Observations

Do vital sign and BP to act as the base line data in order to know if the condition is improving or deteriorating, bserve for cyanosis if improving or getting worse and give oxygen therapy when necessary

Observe Dyspnea if present will prop up the patient to promote lung expansion and there by relieve dyspnea, Observe the pressure area to detect on set of pressure sore development.Observe the sputum for colour amount and consistency to detect Haemoptysis and report the physician

Observe the patient's facial expressions to detect pain and administer prescribed analgesics like panadol

Observe the feeding pattern of my patient and take measures like giving small frequent meals to promote appetite. Observe the respirations to detect tachycardia and report accordingly

Psychological care

Explain the disease process in order to raise the knowledge levels and thereby alley anxiety

Encourage the patient to ask question and I will answer accordingly those I cant answer I will refer to the physician. Explain all procedures to my patient in order to allay anxiety. Involve a successfully managed case to come and talk to my patient in order to allow the patient ask pressing question and get answer this will improve the patients out look on his condition, Involve the loved ones in his care in order for him not to feel neglected. Provide diversional therapy in order to shift the patient's mind from the hospital routine and his condition; involve him in planning his own care in order for him not to feel left out

Explain to him that as the health care team we are doing everything possible to ensure that he get better in order to promote co-operation.Allow patient and relatives to verbalise their concerns then allay them appropriately

Medication

Give oxygen via a correct mask or nasal cannulae so as to prevent dyspnoea and improve the respirations.

Administer prescribed antibiotics as well as prescribed analgesics.

Nutrition

Encourage hourly oral fluids according to patient's preference this will help liquefy the secretions aiding their expectoration.

IV fluids could be given to promote hydration.

Serve small frequent meals to promote appetite

I will allow visitors to bring food preferred by the patient in order to promote appetite.

Give protein and calorie to promote healing and provide energy.

Do regular mouth washes in order to promote appetite

Elimination

Provide a lot of fluids and roughage to prevent constipation

Prove copious fluids in order to promote renal wash out and there by prevent renal problems

Offer a bed pan if he is confined to bed to promote bowel movement

Hygiene

Hygiene is important to promote comfort of the patient. Bed bath if the patient is very sick and a big bath if the patient is stable.Oral toilet is encouraged to prevent halitosis.Change linen whenever soiled especially that the patient will be having fevers which cause increased perspiration. Do nail care to prevent auto infection

Exercise

Exercises are encouraged if the condition of the patient allows to promote blood circulation and recovery.

Passive exercises in the acute stage such as sitting in bed. Patient is taught and encouraged to be doing breathing exercises to promote lung expansion.encourage early ambulation as soon as the condition permits in order to prevent deep vein thrombosis and other complications of immobility

Information Education And Communication n

Minimizing factors that can cause reinfection, including close living conditions, poor nutrition and poorly ventilated living houses or working environment.

Patient is taught and encouraged to continue doing breathing exercises at home.

Adherence to treatment – it has been observed that the highest cause of readmissions to hospital is poor adherence to treatment after discharge. The patient and his family will be told to continue with the prescribed medication even as they will be at home. This will help with completely clearing the infection, hence promoting complete recovery and avoid recurrences of the illness.

Nutrition - An emphasis will be placed on the importance of continued good nutrition with the locally available foods such as nshima with vegetables and kapenta.

This will enhance quick recovery and boost the immunity, henceforth, preventing other illnesses from setting in.

Review dates - Importance of review dates is explained to both the patient and his family.

They are told that they need to follow these appointments with the doctor as it serves as follow up on his condition.

This will also help the doctor assess his discharge plan and see if it will need adjustments or not.

He is also informed on significant changes in signs such as fever, increasing chest pains and haemoptysis, and that he should seek quick medical advice should these signs appear.

The client and his family are taught on the importance of rest and gradual resumption of activities

Complications

Atelectasis - decreased or absent air in the entire or part of the lung. This is due to growth of fibroblasts from the alveolar septa resulting in fibrosed, tough, airless leathery lung tissue.

Pleural effusion- increased fluid in the pleural space. This is usually as a result of the exudation following the inflammation of the lung parenchyma. It can cause shortness of breath by compressing the lung.

Empyema - presence of pus within the pleural cavity. This usually arises from bacterial spread from a severe pneumonia.

Lung abscess – is a localized area of suppuration within a lung tissue that leads to parenchymal destruction.

Metastatic infection - occasionally infection in the lungs and pleural cavity in lobar pneumonia may extend into pericardium and the heart causing pericarditits, endocarditis and myocarditis.

Haematogenous spread to cause; Meningitis, Acute Otitis media and Arthritis

AteleCTACTasis

Atelectasis is a condition in which there is incomplete expansion of lobules (clusters of alveoli) or lung segments, which may result in partial or complete lung collapse. It can be acute or chronic (Scott and Lenker, 2005).

Causes

- Obstruction of the bronchus by mucus plug, bronchiectasis or cystic fibrosis.
- Occlusion of foreign bodies
- Chronic obstructive pulmonary disease like asthma
- Idiopathic respiratory distress of the newborn (hyaline membrane disease).
- Oxygen toxicity
- Pulmonary edema.
- Excessive smoking which leads to damage of cilia
- Inflammatory lung disease
- Bronchogenic carcinoma
- External compression such as those resulting from upper surgical incisions, rib fractures, pleuritic chest pain, tight chest dressings and obesity. .bronchial occlusion by mucus plugs
    - Penetrating chest injury
    - Prolonged immobility
    - Central nervous system depression-as in over dose of anaesthetic drugs

Pathophysiology

Atelectasis may occur in adults as a result of reduced ventilation or any blockage that obstructs passage of air to and from the alveoli, thus reducing alveolar ventilation. After the trapped alveolar air is absorbed into the bloodstream, no additional air can enter into the alveoli because of the blockage. As a result, the affected portion of the lung becomesairless and the alveoli collapse.**Signs and Symptoms**

- Cough, but not prominent
- Sputum production
- Chest pain
- Dyspnoea
- Pleural effusion (transudate type)
- Cyanosis (late sign)Tachycardia
- Low-grade fever

Diagnosis

- History taking where patient will report several symptoms such as dyspnea, pleuritic chest pains.
- Physical examination will reveal decreased chest wall movements, cyanosis substernal or intercoastal retractionsand crackles may also be heard during auscultations.
- Chest x-ray will reveal segmental or lobar collapse.
- Bronchoscopy may rule out neoplasm or foreign body
- Arterial blood analysis may detect respiratory acidosis and hypoxaemia
- Pulse oxymetry may show deterioting SaO2 levels.

Treatment

- Postural drainage
- Frequent coughing and deep breathing exercises may improve oxygenation.
- Bronchodilators such as ventolin can improve mucocilliary clearance and dilate airways.
- Continuous positive airway pressure breathing therapy (CPAP) – artificial ventilation used to supply oxygen to the patient to correct hypoxia and dyspneoa.
- If the cause is neoplasm, surgery or radiotherapy may be needed.
- Analgesics to relieve pain
- Aspiration with sterile tracheal catheters may facilitate removal of mucous plugs.

Lung Abscess

Lung abscess is an infection accompanied by pis accumulation and tissues destruction; it's a localised lung infection.Destruction (Stanley loeb etal,1992)

- **Causes**
- Lung abscess can be caused by locased area of pneumonia or from necrotic area from neoplasm
- Pyogenic bacteria; mycobacteria, fungi and parasites
- Cavitary infarction from embolism
- Necrotic lesions from silicosis or coal workers
- Cavitary cancer from primary bronchogenic carcinoma or metastatic cancer.
- Septic embolism
- Infected cysts

Signs and symptoms

- Coughing which may be blood stained or purulent

- Foul smelling sputum
- Pleuritic chest pains
- Dyspnea
- General body malaise
- Headache
- Anorexia
- Weight loss
- Diaphoresis (excessive sweating)
- Weight loss

Diagnosis

- History taking where patient will reveal the symptoms such as pleuritic chest pain, diaphoresis and foul smelling sputuum
- Physical examination- on percussion areas of dullness over affected tissue will be noted. On auscultation, crackles will be heard.
- Chest x-ray often reveals an abscess which will look like a solid mass.
- Percutaneous aspiration of an abscess provide a specimen which can be used for culture to identify causative organism
- Sputum examination to dertemine the causative organism
- Full blood count willshow raised levels of white blood cells.
- Computed tomography scan (CT scan) will help to differentiate the type of lesion.

Treatment

- Prolonged use of antibiotics therapy
- Postural drainage may help drain the necrotic material of the upper airway where the patient can cough it up.
- Oxygen therapy to relieve hypoxia
- Lesion resection or removal of diseased lung section

Empyema

Empyema refers to accumulation of thick, purulent fluid (pus) within the pleural space, often with fibrin development and a loculated (walled-off) area where infection is located (Smeltzer etal, 2010)).

Causes

- Bacterial pneumonia
- Lung abscess

- Penetrating chest trauma
- Infection of the pleural space
- Esophageal rapture
-

Signs and symptoms

- Fever and chills
- Night sweats
- Chest pains which worsens during inspiration due to inflammation
- Dry cough
- Dyspnea
- Anorexia
- Weight loss

Diagnosis

- Physical examination – on auscultation, there will be decreased or absent breath sounds over the affected area and percussion will reveal dullness on as well as decreased fremitus.
- Chest x-ray to rule out pneumonia and lung abscessBlood culture to identify causative organism
-
- Thoracentecis- sample of fluid is withdrawn to identify causative organism.

Treatment

- Intravenous antibiotics such as septrin, ampicillin
- Intravenous fluids
- Under seal water drainage where pus is drained from the pleural cavity
- Analgesics for pain

Antipyretics such as aspirin

Pulmonary Tuberculosis

Pulmonary tuberculosis is a chronic infectious respiratory condition that primarily affects the lung paranchyma. It may also be transmitted to other parts of the body such as the meninges, bones, lymph nodes and kidneys (Smeltzer etal, 2010)).

Incidence

Pulmonary Tuberculosis is higher in people who are crowded e.g prisons.

Cause

- It is caused by mycobacterium tuberculosis, an acid-fast aerobic rod shaped organism that grows slowly and is sensitive to and sensitive to heat. It can also be caused by mycobacterium bovis which causes disease in cattle. Mycobacterium bovis is a rare cause of tuberculosis.

Mode of transmission

It is transmitted through droplets. The bacillus is expelled from those with active disease during coughing, sneezing or talking. It is airborne, and remains alive for a long time in dust. The organism is inhaled into the lungs, and may spread to other parts of the body through blood. Bovine tuberculosis is contracted by drinking unpasteurised milk from infected cattle.

Predisposing factors

- Poor housing; small houses with poor ventilation play a major role in TB transmission.
- Poor nutritional status; undernourished are predisposed to TB as resistance to infection is reduced.
- Overcrowding in places like markets, schools, and these make it easier to contract TB.
- Age; reduced immunity in the young and old make them prone to TB
- Smoking; the smoke destroys the lung resulting in parenchymal dysfunction leading to diseases like TB.
- Alcoholism; alcohol reduces the immunity of an individual making him or her prone to TB
- Drugs; patients who take immuno-suppressive drugs like steroids are prone to TB due to reduced immunity.
- Immuno suppression caused by diseases like HIV/AIDS, cancer, age the youngest and oldest being at risk.

Pathophysiology

. Tuberculosis begins when a susceptible person inhales mycobacteria and becomes infected. The bacteria are transmitted through the airways to the alveoli, where they are deposited and begin to multiply. The bacilli also are transported via the lymph system and bloodstream to other parts of the body (kidneys, bones, cerebral cortex) and other areas of the lungs (upper lobes). The body's immune system responds by

initiating an inflammatory reaction. Phagocytes (neutrophils and macrophages) engulf many of the bacteria, and TB-specific lymphocytes lyse (destroy) the bacilli and normal tissue. This tissue reaction results in the accumulation of exudate in the alveoli, causing bronchopneumonia. The initial infection usually occurs 2 to 10 weeks after exposure. Granulomas, new tissue masses of live and dead bacilli,are surrounded by macrophages, which form a protective wall. They are then transformed to a fibrous tissue mass, the

Central portion of which is called a Ghon tubercle. This is primary TB. The patient will have no signs or symptoms of the disease, but will have a calcified spot seen on X-ray, a positive TB skin test, and some immunity to the disease. The material becomes necrotic, forming a cheesy mass. This mass may become calcified and form a collagenous scar. At this point, the bacteria become dormant, and there is no further progression of active disease. After initial exposure and

infection, active disease may develop because of a compromised or inadequate immune system response .Active disease also may occur with re-infection and activation of dormant bacteria. In this case, the Ghon tubercle ulcerates, releasing the cheesy material into the bronchi. The bacteria then become airborne, resulting in further spread of the disease. Then the ulcerated tubercle heals and forms scar tissue. This causes the infected lung to become more inflamed, resulting in further development of bronchopneumonia and tubercle formation.

Signs and symptoms

- ***Cough*** – The cough is initially dry due to the irritation by the bacilli that is foreign to the system, later becomes productive when the cheesy material spills into the bronchus and cough up, usually purulent sputum. Later there is hemoptysis due to erosion of blood vessels.
- ***Chest pain*** - is due to the destruction of tissue by disease process exposing the nerve endings and also due to a reduction of oxygen causing ischemia leading to pain. Pain is dull or pleuritic in nature, chest tightness may be present.
- ***Dyspnoea*** – is due to a reduction in lung surface area, the destruction of blood vessels, infection and inflammation of alveoli. If this persists and becomes severe, there may be cyanosis.
- ***Fever*** – is due to the stimulation of the temperature-regulating center in the hypothalamus by the toxins of the bacilli. Fever is cyclic, present around midday and in the night. Fever is long term and low grade.
- ***Chills and sweats*** – are due to fever, there may be sweats around midday and at night when there is fever.
- ***Fatigue and anorexia***. Due to massive destruction of tissue by bacilli and a high ATP requirement. Prolonged anorexia leads to weight loss.

Diagnosis

- History of exposure to T.B. and presenting symptoms.

1. Physical examination will reveal swollen or tender lymph nodes in the neck or other areas, fluid around a lung (pleural effusion) and unusual breath sounds (crackles).
2. Skin testing with either tuberculin-purified protein derivative (PPD) or old tuberculin (OT) is most common. It reveals that the patient has been infected with tuberculosis but does not indicate active disease.

-

- Chest x-ray – posteroanterior (PA) and lateral are the standard views. They will show cavities in the lungs.
- Sputum smear – Acid Fast bacilli – determines the presence of mycobacterium tuberculosis, which after taking up the dye is not de colourized by acid alcohol. This this confirmatory test.
- Sputum culture and sensitivity – culture identifies the specific organism to enable making a specific diagnosis. It should be collected before initiation of antibiotic therapy and thereafter to monitor effectiveness of antibiotic therapy. Sensitivity serves as a guide to anti-microbials therapy by identifying antibiotics that prevent the growth of the organism present in the sputum.
- Gastric washing – most patients swallow sputum when coughing in the morning or during sleep, an examination of gastric content can reveal causative organism.
- Cerebral Spinal Fluid or aspirates from abscess analysis shows the TB BacillBlood – FBC, there is raised white cell count and ESR will be above 20mm.
- Bronchoscopy can be of help if the patient can't produce adequate sputum specimen.
- Thoracentesis

Medical treatment

Drugs are the most important part of the treatment. They must always be given in combination because this is more effective in killing the bacteria and decreases the chance of developing resistance. The drugs which commonly used in Zambia are:

- Rifampicin
- Isoniazide
- Pyrazinamide
- Ethambutol
- Streptomycin

The treatment is divided into three categories

Category 1

- Very infectious group
- New patients who are smear positive (sputum positive)
- Patients with TB meningitis ,TB spine , miliary TB
- They are put on short course therapy of 2 months intensive supervise treatment
- Then 6 months continuation phase.

Drugs *2EHRZ/6EH*

- Ethambutol 800mg OD
- Rifina 450/300 mg OD

- Pyrazinamide 1.5 gm OD
- In TB meningitis, continuation phase is up to 10 months. After initial intensive phase, sputum is tested, if it is positive again, intensive phase continues for 1 more month.

Category 2

- Relapses
- Patients who were initially smear positive and remained positive after 5 -8 months of treatment (usually due to drug resistance).
- They are put on treatment therapy of 3 months intensive therapy and 5 months continuation phase

Drugs ***2HRZS/1HRZE/5HRE***

Category 3

- Children below 12 years
- Pulmonary smear negative but extra pulmonary smear positive
- Pleural effusion

Drugs

- Children ***2RHZ/4HR*** (Ethambutol not given as it may cause blindness)
- Adult ***2RHZ/6HE***
- Pregnant women ***8HRZE***

Most anti-TB drugs are now manufactured in fixed-dose combinations (FDCs) instead of only separate tablets. Fixed-dose combinations are drugs combined in tablet or capsule form, in specific dosages, to facilitate correct drug intake. The following FDCs are becoming widely available:

- isoniazid–rifampicin–pyrazinamide–ethambutol (HRZE), (4FDCs))
- isoniazid–rifampicin–pyrazinamide (HRZ)3
- isoniazid–rifampicin (HR)2
- isoniazid–ethambutol (HE)2

The table below shows the anti- TB drugs, action and the side effects

Figure **Figure**

DRUG	**ACTION**	**SIDE EFFECTS**	**NURSING INTERVENTION**
Isoniazid (INH)	Is bactericidal. Penetrates all body tissues and fluids, including cerebrospinal fluid.	peripheral neuritis, optic neuritis, psychotic episodes, vertigo and steven-	Increases the risk of hepatic toxicity and so it is important to educate the care giver to report any

		johnson syndrome	dis colouration of the skin, sclera and mucous membranes
Rifampicin (R)	Is bactericidal. Penetrates all body fluids and tissues including cerebrospinal fluid.	- Nausea vomiting diarrhea, orange –red discoloration of urine, saliva and other body fluids.	Tell patient that urine and sweat may appear orange temporarily; it may decrease effectiveness of oral contraceptives, anticoagulants, corticosteroids and other drugs. Instruct the patient to consult with health care providers prescribing other medications and alert them to this therapy.
Ethambutol (E)	Is bacteriostatic. Penetrates body tissues and fluids except cerebrospinal fluid.	Optic neuritis, red green colour blindness, peripheral neuritis, pruritus and urticaria.	Tell patient to take with food and have vision checked frequently. Assess lung sounds and character and amount of sputum periodically throughout therapy. Advise patient to report blurring of vision, constriction of visual fields, or changes in colour perception immediately. Tell patient that renal and hepatic functions, CBC, and uric acid levels should be monitored routinely throughout therapy.
Pyrazinamide (Z)	Prevents or reverses neuropathy	Hepatotoxicity, jaundice, anorexia	Instruct patient to take medication as directed. If a dose is missed, it may be

	associated with INH therapy; used in transport of amino acids, formation of neurotransmitters, and synthesis of heme.	flushing, dysuria and arthlagia.	omitted because an extended time is required to become deficient in vitamin B6. The source of vitamin is a well-balanced diet with foods high in vitamin B6 (bananas, whole-grain cereals, potatoes, lima beans and meats). Tell patients taking vitamin supplements not to exceed the RDA.
Streptomycin (S)	Is bactericidal. Inhibits protein synthesis in bacteria; has poor penetration of body tissues.	Hypersensitivity reaction, paraethesia	Monitor kidney function of patient monthly and check vestibular and general hearing monthly

Supportive treatment includes the following:

- Corticosteroids' in complicated forms such TB meningitis, TB lymph node glands and pericardial TB e.g. prednisolone 2mg/kg body weight daily, increased to 4mg/kg daily in the case of most seriously ill children with maximum dosage of 60mg/kg body weight for 4 weeks. The dose should be gradually reduced over 12 weeks before stopping.
- Analgesics/antipyretic- such as paracetamol 100mg/kg
- Appetite boosters- Multivitamins
- Haematinics- folic acid and ferrous sulphate (MoH, 2007).

Nursing care

Aims

To eliminate the causative organism

To prevent spread of infection

Environment

Isolate patient in a quiet and well ventilated room until patient is no longer contagious to prevent spread of infection.

Position

Nurse patient in any comfortable position but if dyspnoeic put in upright position to facilitate full lung expansion. Frequent turning is necessary to prevent accumulation of sputum on one side and also pressure sores.

Rest

Provide periods of rest to conserve energy and promote recovery. This can be achieved by doing related procedures collectively, providing a quite environment and administering analgesics in cases where patient experiences chest pains.

Observations

Observe general condition to see whether condition is improving or not. Do vital sign observations 4 hourly which are temperature, pulse, respirations and pulse to determine the progression of disease and response to therapy. Watch for the side effects of the drugs such as neuropathy. Observe for colour and consistency of the sputum. Observe also the degree of dyspnea, restlessness and cyanosis to determine the need for therapy.

Psychological care

Explain disease process to allay anxiety. Explain also the reason for isolation to gain cooperation. Allow patient and significant others to ask questions and air out their views. Include patient in care and let relatives take part whenever possible. Provide diversional therapy to divert patient's mind from the condition.

Nutrition

Provide patient with nutritious food reach in proteins, vitamins and carbohydrates to build up worn out tissues, boast immunity and provide energy respectively. Serve food which is appetizing in small frequent amounts to promote appetite. Weigh patient daily. Ensure good hydration to loosen secretions and prevent dehydration. Do oral care as well to promote appetite.

Hygiene

Do bed bath or assisted bath to promote patient's comfort and blood circulation. Frequent oral care should be done to promote appetite and mouth infections. Change linen whenever necessary to promote comfort. Keep sputum mugs clean and covered.

Elimination

Give food rich in roughage to prevent constipation. Increase fluid intake to prevent constipation as well as flush out toxins. Sputum should also be observed for consistency and colour Observe the bowel pattern.

Health education/ advice on discharge

- Educate the patient and the family members on the disease. Teach signs and symptoms of TB and the need to seek medical advice early.
- Drug compliance – advice patient to take the medication as prescribed to combat the infection and to prevent drug resistance.
- Emphasize the need of following the review dates
- Explain the importance of screening all contacts in order to ensure early diagnosis and treatment.
- Stress the importance of eating nutritious diet
- Teach patient on specific precautions to prevent spread of infection
- Advise patient to avoid overcrowded places

Prevention of infection

- Isolate patient by nursing him/her away from other patients
- Advise patient to cover mouth when coughing.
- Use masks, gowns and gloves when nursing and handling patient's secretions.
- Disinfect sputum before disposing off.

Restrict visitors**Nursing care plan of TB**

Problem identified

1. Altered gas exchange
2. Knowledge deficit
3. Altered nutrition.

Figure

PROBLEM	**NURSING DIAGNOSIS**	**GOAL/ OBJECTIVES**	**INTERVENTION**	**EVALUATION**
Impaired gaseous exchange	Impaired gas exchange related to reduced lung expansion evidenced by dyspnoea.	Patient will have improved gas exchange within the first 48 hours of admission.	-patient will be nursed in a propped up position to allow full lung expansion. - administ	- patient has improved gaseous exchange within 48hrs.of admission evidenced by absence of dyspnoea and normal respirations.

			er oxygen 100% through the nasal catheter to promote free air entry in to the lungs. -Nasal care will be performed to clear all debris and secretions which may block free entry of air through the respiratory tract. -give prescribed anti TB drugs	
Knowledge deficit	Knowledge deficit about	ppatient will be provided with knowledge on the	-provision of education on the spread and	Patient had acquired Knowledge on

	spread and treatment of TB related to lack of informatio n evidenced by anxiety and inquisitive ness.	spread and treatment of tuberculosis within the first week of commencement of treatment	treatment of TB in order to reduce the anxiety and inquisitiveness to allow them to ask questions and answers will be provided appropriately.	spread and treatment of TB within first week of commencement of treatmentevidenc ed by verbalization,cal mness.
Altered nutrition	Altered nutrition anorexia due to disease process evidenced by weight loss and general body weakness.	Patient's nutritional stutus will be improved during the period of hospitaliza tion.	- serve nutritiou s food in small frequent and well balanced meals to encoura ge patient to eat and hence promote healing. -Do oral care to promote free flow of saliva and encoura ge good breaths which cause	Patient's nutritional status has been impoved duriring the period of hospiatalization evidenced by weight gain andpatient looking strong.,

			nausea. Administer prescribed vitamins like multivitamin to boost the immune recovery.	

Complications Of Pulmonary Tb

- Miliary TB – this may come about if a necrotic ghon complex erodes through a blood vessel, large amounts of organisms invade the bloodstream and spread to all body organs
- Respiratory failure due to extensive scaring of the lung tissue and accumulation of secretions in the airway
- Pleurisy with Effusion- this result from the release of caseous material into the pleural space the bacterial containing material triggers an inflammatory reaction and pleural exudates of protein rich fluid.Broncho Pleural fistula
- Empyema
- Atelectasis

- Pneumothorax- results from the rupture of a peripheral cavity. Can lead to the formation of a bronchopleural fistula.

- ***Bronchiectasis,*** bronchial obstruction, and airway stenosis may result from endobronchial disease, though this is much less common in the post-chemotherapy era. It is more common in the presence of extensive parenchymal disease, and is associated with lymph node enlargement with compromise of airway size.
- ***Tuberculosis pneumonia,*** this may result when large amounts of tubercle bacilli are discharged from the liquefied necrotic lesions into the lung or lymph nodes.

Co-infections of TB with HIV (Refer to Medicine III on opportunistic infection)

Occupational Health And Safety Diseases

These are diseases that are caused by harmful particles, mists, vapors or gases inhaled while a person works. Exposure to such factors can cause different types of lung diseases such as asbestosis, silicosis and coal workers pneumoconiosis among others.

Pulmonary Embolism

Pulmonary embolism (**PE**) is a blockage of the main artery of the lung or one of its branches by a substance that has travelled from elsewhere in the body through the bloodstream (embolism). PE most commonly results from deep vein thrombosis (a blood clot in the deep veins of the legs or pelvis) that breaks off and migrates to the lung, a process termed *venous thromboembolism* (VTE). A small proportion of cases are caused by the embolization of air, fat, or talc in drugs of intravenous drug abusers or amniotic fluid. The obstruction of the blood flow through the lungs and the resultant pressure on the right ventricle of the heart lead to the symptoms and signs of PE. The risk of PE is increased in various situations, such as cancer or prolonged bed rest.

Definition; pulmonary embolism is the occlusion of one or more pulmonary arteries by thrombi that originate elsewhere, typically in the large veins of the lower extremities(Beer etal, 2006).

Risk factors

The risks are condition that impair venous return and that cause endothelia injury or dysfunction especially in patients with underlying hypercoagulable state.

- Atrial fibrillation
- Cigarette smoking
- Extremity trauma

- Hypercoagulability disorders such as anti-Prothrombin III disorder, increase in factor VIII and XI.
- Immobilization
- Indwelling venous cathers
- Malignancy
- Oral contraceptives
- Nephrotic syndrome
- **Signs and symptoms of pulmonary embolism**
    - Difficulty breathing
    - Chest pain on inspiration, and palpitations.
    - Low blood oxygen saturation and
    - Cough and hemoptysis
    - Cyanosis, rapid breathing, and a rapid heart rate.
    - Severe cases of PE can lead to collapse, abnormally low blood pressure, syncope and cardiac arrest.
    - In the presence of right cardiac failure distended jugular vein may be evident.

Diagnosis

- Pulse oximetry to assess oxygenation
- **ECG** shows tachycardia and T- wave abnormalities.
- **Chest X-ray** will show focal infiltrate, atelectasis and pleural effusion, often done on people with shortness of breath to help rule-out other causes, such as congestive heart failure and rib fracture .
- **CT** pulmonary angiography
- **Ultrasonography** of the legs, also known as leg doppler, in search of deep venous thrombosis (DVT).
- **Echocardiography-** In massive and submissive PE, dysfunction of the right side of the heart may be seen on echocardiography, an indication that the pulmonary artery is severely obstructed and the right ventricle, a low pressure pump, is unable to match the pressure.

Treatment

1. The initial treatment of pulmonary embolism is oxygen therapy to correct hypoxemia
2. Intravenous fluid 0.9% normal saline
3. Vasopressors for hypotension
4. Patient should be admitted in then acute bay for continued monitoring for life threatening cardiovascular conditions.
5. Subsequent treatment involves anticoagulants such as heparin bolus 80units/kg or heparin infusion 18gm/k. warfarin.
6. Clot elimination ,

Pneumoconiosis

Pneumoconiosis refers to a group of lung diseases caused by inhalation and retention of dust particles, toxic fumes and chemicals (Lewis et al, and 2011).

Types

Depending upon the type of dust, the disease is given different names:

- Coalworker's pneumoconiosis (also known as "black lung" or anthracosis) - coal, carbon
- Asbestosis - asbestos
- Silicosis (also known as "grinder's disease") - silica
- Bauxite fibrosis - bauxite
- Berylliosis - beryllium
- Siderosis - iron
- Byssinosis - cotton
- Silicosiderosis - mixed dust containing silica and iron
- Labrador Lung (found in miners in Labrador, Canada) - mixed dust containing iron, silica and anthophyllite, a type of asbestos

Diagnosis

- History taking will reveal manifestations such as shortness of breath and exposure to causative agents.
- Chest X-ray may show a characteristic patchy, subpleural, bibasilar interstitial infiltrates or small cystic called honey combing.

-

Pathology

Following the exposure to chemicals, dust or gases, there is diffuse parenchyma infiltration with phagocytic cells. This results in diffuse pulmonary fibrosis due to tissue repair after inflammation.

Silicosis

Silicosis is a permanent scarring of the lungs caused by inhalation of silica dust (Berkow, 1997

Pathophysiology

When small silica dust particles are inhaled, they can embed themselves deeply into the tiny alveolar sacs and ducts in the lungs, where oxygen and carbon dioxide gases are exchanged. There, the lungs cannot clear out the dust by mucous or coughing.

When fine particles of silica dust are deposited in the lungs, macrophages that ingest the dust particles will set off an inflammation response by releasing tumour necrosis factors, interleukin-1, leukotriene B4 and other cytokines. In turn, these stimulate fibroblasts to proliferate and produce collagen around the silica particle, thus resulting in fibrosis and the formation of the nodular lesions.

Classification

Classification of silicosis is made according to the disease's severity), onset, and rapidity of progression. These include:

- Acute silicosis
- Chronic simple silicosis
- Accelarated silicosis
- Complicated silicosis

Acute silicosis

The onset is sudden, and occurs within few weeks to 5 years after exposure to high concentrations of silica dust.

Signs and symptoms

- Rapid onset of severe shortness of breath
- cough
- weakness

- weight loss

Chronic simple silicosis

The onset is gradual usually results from long-term exposure to relatively low concentrations of silica dust and usually appearing 10–30 years after first exposure. It is the most common type of silicosis.

Signs and symptoms

- Initially patient is asymptomatic
- Dyspnea (shortness of breath) exacerbated by exertion
- Cough, often persistent and sometimes severe
- Fatigue
- Tachypnea (rapid breathing) which is often labored
- Loss of appetite and weight loss
- Chest pain
- Fever
- Gradual dark shallow rifts in nails eventually leading to cracks as protein fibers within nail beds are destroyed.

Accelerated silicosis

This type of Silicosis develops over 5–10 years after first exposure to higher concentrations of silica dust. Symptoms and x-ray findings are similar to chronic simple silicosis, but occur earlier and tend to progress more rapidly. Accelerated silicosis can easily complicate to complicated silicosis.

Complicated silicosis

Silicosis can become "complicated" by the development of severe scarring (progressive massive fibrosis). This type is associated with more severe symptoms and respiratory impairment than simple disease. Other lung conditions such as tuberculosis, non-tuberculous mycobacterial infection, and fungal infection, certain autoimmune diseases, and lung cancer can worsen the condition.

Diagnosis

- History taking and physical examination
- Chest x-ray will show distinctive patterns of scarring and nodules
- Arterial blood gas analysis reveals low blood oxygen concentration.

- Pulmonary function test shows reduced vital capacity in complicated silicosis.

Treatment

Silicosis is an irreversible condition with no cure. Treatment options currently focus on alleviating the symptoms and preventing complications. These include:

- Stopping further exposure to silica and other lung irritants, including tobacco smoking.
- Cough suppressants.
- Antibiotics for bacterial lung infection.
- TB prophylaxis for those with positive tuberculin skin test or IGRA blood test.
- Prolonged anti-tuberculosis (multi-drug regimen) for those with active TB.
- Chest physiotherapy to help the bronchial drainage of mucus.
- Oxygen administration to treat hypoxemia, if present.
- Bronchodilators to facilitate breathing.
- Lung transplantation to replace the damaged lung tissue is the most effective treatment, but is associated with severe risks of its own.
- For acute silicosis, Whole-lung lavage may alleviate symptoms, but does not decrease overall mortality.
- Corticosteroids therapy
- Adequate fluid intake

Provide high calorie, high protein diet

3.7Self-assessment test

1. The following are the common investigations and procedures of the respiratory system disorders except:
   A) Bronchoscopy
   B) Gastroscopy
   C) Sputum examination
   D) Chest x-ray
2. The condition which can come due to blockage of the Eustachian tube in rhinitis is:
   A) Bronchitis
   B) Sinusitis
   C) Otitis media

Pneumonia

3) The commonest mode transmission for respiratory disorders is

A) Ingestion of infective organism

B) Inhalation of the infective organismC) Fecal oral route

D) Sexual contact

4. A medical name for loss of voice is dysphagia T/F

5. The management of laryngitis includes resting of the voice. T/F

6. Bronchitis is one of the disorders of the upper respiratory tract. T/F

7. Medical term for lung collapse is atelectasis T/F

8. Accumulation of thick and purulent fluid in the pleural cavity is referred to as empyema T/F

9. The causative organism of pulmonary tuberculosis is

A) Tuberculosis bacteria

B) Mycobacterium tuberculosis

C) Staphylococcal aureus

D) Streptococcal organism

10. Tuberculosis can also be transmitted to other body parts via blood. T/F

11. Coughing up blood is referred to as haemoptysis T/F

12. The common type of tuberculosis is

A) Milliary tuberculosis

B) Extra pulmonary tuberculosis

C) Pulmonary tuberculosis

D) TB adenitis

13. The following are the specific antibiotics which are used in Zambia to treat tuberculosis exept:

A) Rifampicin

B) Streptomycin

C) Gentamycin

D) Ethambutol

14. An adult who is sputum positive and is treated for TB for the first time qualifies to be under:

A) Category 1 regime

B) Category 2 regime

C) Category 3 regime

D) Category 4 regime

15. One of the side effects of isoniazide is hepatotoxicity. T/F

16. A condition which is caused by silica and causes permanent scarring of the lungs is reffered to a Silicosis. T/F

3.8Answers to the self-assessment questions

1. B
2. Otitis media
3. B
4. F
5. T
6. F
7. T
8. T
9. B
10. T
11. T
12. C
13. C
14. A
15. T
16. T

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UNIT 4: CARDIOVASCULAR SYSTEM

4.0 Introduction

Dear Learner, congratulations for coming this far. You are now in the last unit of our course on medical and medical nursing part 1.You will learn about cardiovascular system. As you have learnt in high school and foundation in nursing, the heart is a key to life. It pumps blood through blood vessels and circulates it throughout the body organs keeping us alive. In this unit you will review the anatomy and physiology of the cardiovascular system and the roles of the nurse in investigations and procedures in cardiovascular disorders. This will be followed by management of the patient with blood and heart disorders, hypertension and peripheral vascular disorders .You will also learn malaria and filariasis.

4.1Unit objectives

At the end of the session you should be able to:

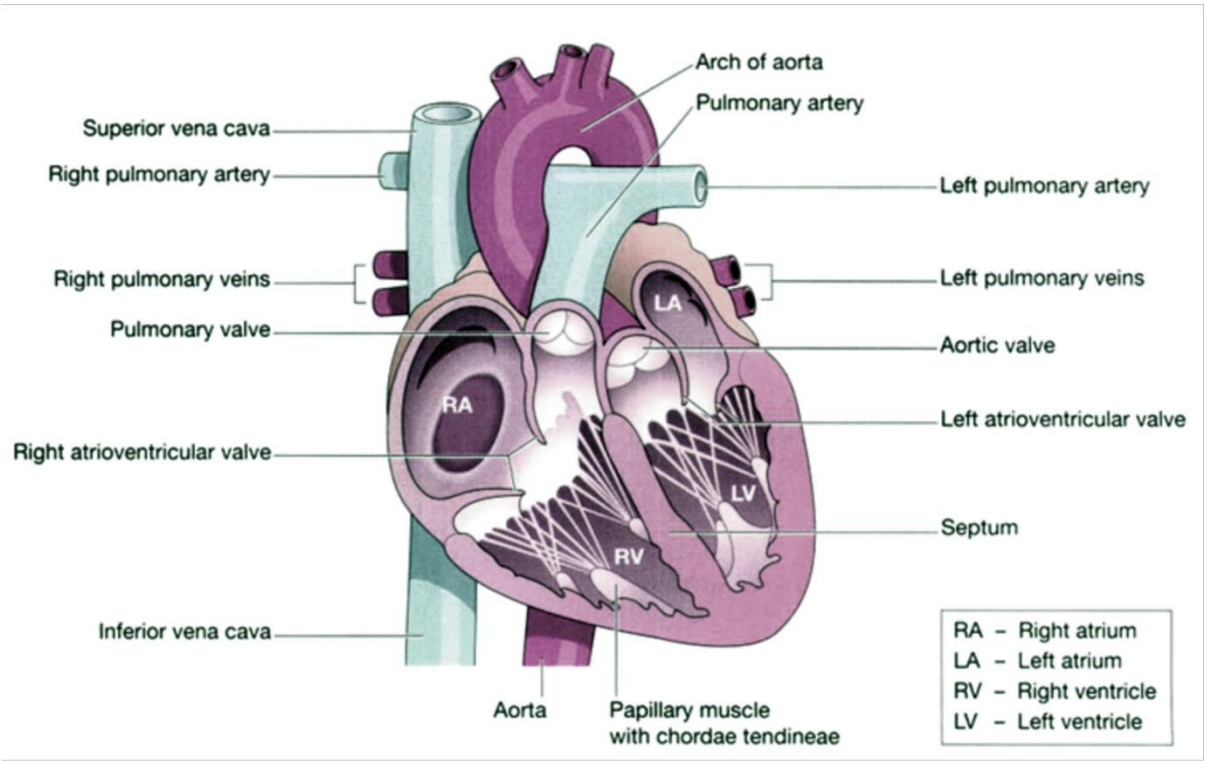
4.2 Anatomy And Physiology Of The Cardiovascular System

The cardiovascular system consists of the heart, arteries, veins and the lymphatic's. The main function is to ensure blood circulation throughout the body ensuring oxygen and nutrient delivery to the tissues and organs. This system brings life supporting nutrients to cells, removes metabolic wastes products and carries hormones from one part of the body to the other. The cardiovascular system is divided into two main parts for descriptive purposes;

- **Circulatory system** – the heart which acts as a pumps operating side by side and the blood vessels through which the blood circulates.
- **The lymphatic system-** consisting of lymphnodes and lymph vessels through which colorless lymph flows.

The Heart

The heart has three layers; *Endocardium*- the smooth inner layer, the *myocardium*- the thick muscular middle layer that contracts in rhythmic beats and the *epicardium*- the thin serous membrane or outer surface of the heart. Covering the entire heart is the saclike membrane called the pericardium which has two layers; a visceral layer that's in contact with the heart and a parietal or outer layer.



FigureTable

The Blood Vessels

The heart pumps blood into vessels that vary in structure,size and function, and there are several types: arteries,arterioles, capillaries, venules and veins.

Arteries and arterioles

These are the blood vessels that transport blood away from the heart.

Capillaries and sinusoids

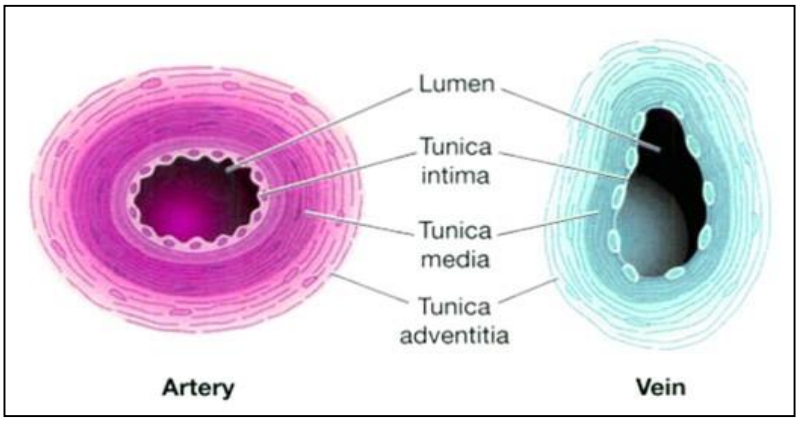
The smallest arterioles break up into a number of minutevessels called *capillaries.* Capillary walls consist of a single layer of endothelial cells through which water and other small-molecule substances can pass.

Veins and venules

The veins are the blood vessels that return blood at low pressure to the heart. The walls of the veins are thinner than those of arteries but have the same three layers of tissue. They are thinner because there is less muscleand elastic tissue in the tunica media.

The structures of the artery and the veins

- ***tunica adventitia*** or outer layer of fibrous tissue
- ***tunica media*** or middle layer of smooth muscle elastic tissue
- ***tunica intima*** or inner lining of squamous epithelium called *endothelium*



FigureTable

The amount of muscular and elastic tissue varies in the arteries depending upon their size. In the large arteries, sometimes called elastic arteries, the tunica media consists of more elastic tissue and less smooth muscle. These proportions gradually change as the arteries branch many times and become smaller until in the *arterioles* (the smallest arteries). Here the tunica media consists almost entirely of smooth muscle. Arteries have thicker walls than veins and this enables them to withst and the

high pressure of arterial blood. When an artery is cut blood spurts at high pressure while a slower, steady flow of blood escapes from a vein.Some veins possess *valves*, which prevent backflow of blood, ensuring that it flows towards the heart. Valves are abundant in the veins ofthe limbs, especiallythe lower limbs where blood must travel a considerable distance against gravity when the individual is standing. Valves are absent in very small and very large veins in thethorax and abdomen. They are formed by a fold of tunicaintima strengthened by connective tissue. The cusps are *semilunar* in shape with the concavity towards the heart. The smallest veins are called *venules.*

The heart pumps blood into two anatomically separate systems of blood vessels namely:

- Pulmonary circulation
- Systemic circulation

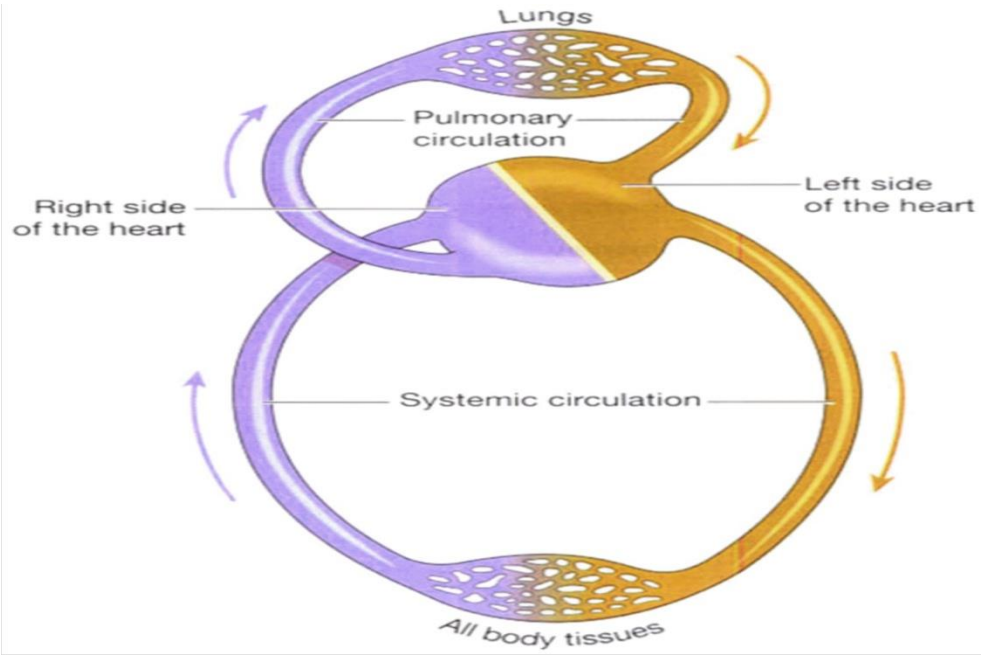
Pulmonary circulation

The right side of the heart generates the circulation to the lungs where gas exchange occurs e.g., CO2 leaves the blood and enters the lungs and O2 leaves the lungs and enters the blood and the left heart feeds the rest of the body. The right atrium drains the deoxygenated blood from the superior and inferior vena cavae and discharges blood to the right ventricle which in turn pumps it into the pulmonary artery.

Systemic circulation

The left side of the heart pumps blood into the systemic circulation which supplies the rest of the body, there, tissue wastes is passed into the blood for excretion and body cells extract nutrients and oxygen. The left atrium drains oxygenated blood from the lungs through four pulmonary veins and discharges blood into the left ventricle, which in turn pumps it into the aorta. During ventricular contraction (systole) the tricuspid valve in the right heart and the mitral valve in the heart close, and the pulmonary and aortic valves open. In diastole, the pulmonary and aortic valves close and the two atrioventricular valves open.

The systolic pressure in the LV is normally at least four times greater than that in the right, and the wall of the LV is usually at least 1cm thick compared with the RV. The atria lie within mediastinum, anterior to the oesophagus and the descending aorta. The ventricles lie anterior to the atria and narrow towards the apex of the heart, which lies immediately behind the sternum and is not only to the right of but also anterior to the LV.



The relationship between the pulmonary and the systemic circulation.

FigureTable

4 .3The Roles Of The Nurse In Investigations

Review the roles of the nurse in investigations that you leant in unit 2 by answering the following question.

- Explain the roles of the nurse in investigations as you leant in unit 2?

Well done, now continue to learn on common cardiovascular investigations. They are many cardiovascular investigations that can be done. You will learn others as you proceed in nursing.

When the heart cells are injured they release cardiac- specific troponin. Usually the level in the blood is low. An Increase indicates myocardium injury (MI).

- Following are the common investigations in cardiovascular disorders showing amount of levels in the blood and their implications.
- Blood creatine kinase (CK-MB)
- Creative Kinase (CK) is released in the presence of MI. Concentration > 4% -6% of total CK are highly indicative of MI . Serum level increase within 4- 6 hr specific isozyme after MI.
- ***Troponin(cardiac)***
- Troponin (T) and troponin 1(cTnl) are contractile proteins that are released following an MI on cardiac tissue.

- If cTnl are Negative < o.5 ng /ml (0.5 mcg/L) then there is no MI.
- If there is Indeterminate or suspicious for injury to myocardium injury, it increases from 0.5-2.3 ng/ml. or (0.5-2.3 mcg/L).
  If MI is positive cTnl is >2.3 ng/ml. (2.3 mcg/L)

- Myoglobin
- It is a low-molecular weight protein that is 99% -100 % sensitive for MI. A serum concentration of 30 -60 min indicates MI. The levels vary with sex.
  - Male -15.2 – 91 .2 mcg/L.

  Female; 11.1 – 57.5mcg/l

C-reactive protein (CRP)

CRP when present in blood marks inflammation. This predicts patients to cardiac disease and events, even with normal lipid values. One is said to be at the following levels of MI

Lowest risk: 1mg/L

Moderate risk: 1-3 mg

High risk : >3mg/L

Serum lipids Cholesterol.

Cholesterol is a blood lipid. Elevated cholesterol indicates atherosclerotic heart disease.

Normal values range :< 200 mg/dL (, 5.18 mmol/L) and

varies with age and gender.

Triglycerides

They are a mixture of fatty acids. When elevated they are associated with cardiovascular disease or diabetes.

The normal intervals are :< 150 mg/dL (1.7 mmol /L) and it

varies with age

Echocardiogram

The following are examples

A. Contrast
B. M-mode
C. colour-flow imaging (duplex)
D .Real-time three dimensional

A transducer that emits and receives ultrasound waves is placed in four positions on the chest above the heart .It records sound waves of the heart, direction and flow of blood through the heart. It also measures valvular abnormalities, congenital cardiac defeats, wall motion, ejection fraction, and cardiac function. The image is enhanced by IV contrast agent.

This is very interesting you have seen that the new technology is used to diagnose heart disorders. And then it makes it easy for one to be treated on time. For example, an echocardiogram is a test that uses sound waves to create pictures of the heart. The picture is more detailed than a standard x-ray image. An echovardiogram is safe and does not expose the client to radiation. Now continue to learn on these investigations on the cardia:

disorders

Pharmacisological echocardiogram

It substitutes exercises stress test in individuals an able to exercise. A positive inotropic agent (dobutamine or dipyridamode) is infused. The dosage is increased by 5 min intervals while cardiogram being performed detecting heart wall abnormalities at each intervals.

Transesophageal echocardiogram (TEE)

A probe with ultra sound transducer at the tip is swallowed with controlled angle and depth by the doctor. As it passes the esophagus clear images of the heart size, wall motion, valvular abnormalities, endocarditis, vegetation are sent back. Source of thrombi if present sent back. IV Contrast medium is injected to evaluate direction of blood flow in case of an atrial or ventricular septal defect.

Nuclear Cardiology

IV injection of radioactive isotopes (technetium-sestamibi) is used. The radioactive uptake by the heart is viewed by a camera. It is done to rule out myocardial contractility information, myocardial perfusion, and acute cell injure.

Cardiac Computer Tomography (CT). They are two types CT angiography (CTA) and Ultrafast CT/Electron beam computed tomography(EBCT)

It is done with or without IV contrast to visualize heart anatomy, coronary circulation, and blood vessels.

CT angiography (CTA)

An IV contract used to obtain images of blood vessels and diagnose cardiac diseases.

Ultrafast CT.

Uses scanning electron beam to quantify calcification in coronary arteries and heart valves.

Cardiac Catheterization

A catheter is inserted in to a right vein and or artery on left side of the heart. This is done to obtain oxygen levels and pressure readings within the heart chambers. Contrast medium is used in this investigation.

Coronary angiography

During Cardiac catheterization, contact medium is injected directly into coronary arteries to assess coronary arteries and collateral circulation.

Well done, you have just covered some common investigations that are done to rule out cardiovascular disorders .Now let us continue by revising common procedures done to rule out cardiovascular disorders. You covered them in fundamental nursing. To revise I will ask you the following questions;

- How do you take vital signs (Temperature, pulse, and respirations)
- Explain four major techniques that are used in performing the physical examination of patients as learnt in unit 1? (inspection, palpation, percussion, and auscultation)

You are proceeding to learn on blood disorders. Remember in cardiac investigations you had learnt that as the specific cardiac troponin circulate in the blood they indicate MI.

4.4 Management Of The Patient With Blood Disorders

How are blood disorders divided?

Blood disorders are divided into;

- Anaemia
- Leukemia
- Hemorrhagic disorder

Thrombocytopenia

What is blood?

Bloodis a connective tissue composed of plasma and blood cells.

What are the three types of blood cells?

The three types of cell are;

- Erythrocytes (red cells)
- Platelets (thrombocytes)
- Leukocytes (white cells)

What is the original of blood cells?

All blood cells originate from pluripotent stem cells and go through several developmental stages before entering the blood. Different types of blood cells follow separate lines of development. Blood cell formation takes place in the red bone marrow. In adults, haemopoiesis in the skeleton is confined to flat bones, irregular bones and the ends (epiphyses) of long bones. The main sites are the sternum, ribs, pelvis and skull. In addition, some lymphocytes (white blood cells) are produced in lymphoid tissue.

Functions Of Blood Cells

What are the functions of erythrocytes, leukocytes, and platelets cells?

The following are the functions of blood cells;

- **Erythrocytes:** transport oxygen from the lungs to the body tissues and carbon dioxide from the body tissues to the lungs. Oxygen binds to hemoglobin in order to be transported.
- **Leukocytes:** protect the body against microbes and other foreign materials. There are two main types of leukocytes:
    1. Granulocytes (polymorphonuclear leukocytes) – neutrophils, eosinophils and basophils
    2. Agranulocytes – monocytes and lymphocytes

- **Platelets:** contain substances that promote blood clotting, which causes hemostasis (cessation of bleeding)

The Anaemias

How do you define anaemia?

- **Definition:** Anaemia is a condition in which there is a reduction in the oxygen carrying capacity of the blood as a result of fewer circulating erythrocytes than normal or a decrease in the concentration of haemoglobin or haematocrits. A person is said to have anaemia when the haemoglobin concentration is below 13.5g/dl in adult males and 11.5g/dl in females.

Types of Anaemia

Discuss the types of anaemia?

The different types of anaemia are;

- Haemorrhagic anaemia this is due to blood loss. Blood loss can either be acute or chronic
- Haemolytic anaemia is due to destruction of red blood cells.
- Deficiency anaemia is due to deficiency of certain nutrients such as iron, vitamin B_{12}, folic acid and vitamin .These vitamins are essential for production of red blood cells
- Aplastic anaemia is due to defective bone marrow function

General Manifestations of Anaemia

What are the signs and symptoms of anaemia?

The signs and symptoms of anaemia are;

- Shortness of breath on exertion
- Pallor of the mucus membrane, palms or conjunctiva
- Headache
- Dizziness
- Faintness
- Tingling or “pins and needles” in the extremities
- Person feels cold in temperatures comfortable to others
- Tachycardia/heart palpitations
- In severe cases – oedema, cardiomegaly, signs of angina pectoris

Deficiency Anaemia

What are the causes of iron deficiency anaemia?

Iron deficiency Anaemia is most commonly caused by:

- Increased demand during pregnancy, lactation, and menstruation
- Chronic blood loss from the GIT e.g. worm infestation, peptic ulceration, carcinoma of any part of the GIT
- Dietary deprivation of iron
- Impaired intestinal absorption

Clinical Manifestations

What are the s***Igns And Symptoms Of Deficiency Anaemia?***

The signs and symptoms of deficiency anaemia are;

- Soreness and inflammation of the mouth and tongue
- Changes in finger nails – brittle, discoloured, concave or spoon shaped
- Dysphagia due to oesophageal atrophy
- Pica – eating of strange things, such as charcoal, earth, or foods in great excess e.g. tomatoes, greens

Vitamin B_{12} Deficiency Anaemia:

What are the causes of vitamin B_{12} deficiency anamia?

Vitamin B_{12} deficiency is caused by:

- The diet may supply inadequate amounts of vitamin B_{12},
- There may be a deficiency of intrinsic factor, as in pernicious anaemia, following total gastrectomy,
- The terminal ileum from which the vitamin is absorbed may be absent following surgery, or it may be diseased as in Crohn's disease.
- Vitamin B_{12} may be removed from the bowel by intestinal parasites such as fish tapeworms

Clinical Manifestations

What are the signs and symptoms of vitamin deficiency?

The signs and symptoms are;

- Glossitis and angular stomatitis. The tongue will appear smooth
- Loss of appetite and some intolerance of food
- Symmetrical tingling or "pins and needles" or coldness and numbness in the extremities.
- Muscular weakness
- Ataxia (loss of coordination and staggering)
- Paralysis
- Impairment of vision due to degeneration of the optic nerves

- Pale yellow tint due to increased haemolysis
- Infertility in females
- Dementia may occur in either sex

APLASTIC ANAEMIA

How does aplastic anaemia occur?

Aplastic anaemia occurs when there is a marked reduction of haemopoietic stem cells in the bone marrow. There is no infiltration of malignant tissue and the patient's nutritional status is normal.

CAUSES

What is the cause of aplastic anaemia?

The causes of aplastic anaemia are:

- Suppression of the bone marrow from substances such as chloramphenicol, the sulphonamides, DDT, benzene, glue solvents etc
- Congenital aplastic anaemia

Clinical Manifestations

What are the signs and symptom of aplastic anaemia?

The signs and symptom of aplastic anaemia are;

- Pallor
- Lethargic
- Prone to minor infections like sore throat, tonsillitis, and pharyngitis and skin infections
- Thrombocytopenia
- Bruising

HEMOLYTIC ANAEMIA

What are the causes of hemolytic anaemia?

Hemolytic anaemia occurs when the erythrocytes are prematurely destroyed (i.e. before their normal 100-120 days life span).

How is hemolytic anaemia divided?

Hemolytic anaemia can be broadly divided into two groups:

- Those where the defect is in the erythrocytes, such as hereditary spherocytosis, thalassaemia and sickle cell anaemia
- Those where the abnormality is in the plasma such as incompatible blood transfusion, haemolytic disease of the newborn, and infections such as malaria.

  Clinical Manifestations

  What are the signs and symptoms of hemolytic anaemia?

  The signs and symptoms of hemolytic anaemia are;

- Pallor
- Haemoglobinuria
- Ulceration of the legs,
- Pruritic skin
- Spleenomegally
- Skeletal changes due to enlargement of marrow spaces
- Mild jaundice
- May have gallstones if anaemia is long standing, due to excessive production of bilirubin

Congratulations you have learnt a lot on the types and manifestation of different types of anaemia.

Investigations

What investigation can be done to rule out anaemia?

The investigations that can be conducted to rule out anaemia are;

- Full blood count, will show decreased number of circulating erythrocytes (hypochromic and microcytic).The reticulocyte count is also low, and the thrombocyte count may be moderately raised in iron deficiency anaemia.
- Serum iron and total iron binding capacity (TIBC). Serum iron will be low whilst TIBC rises
- Blood film shows megaloblastic changes in Vitamin B_{12} deficiency and folic acid deficiency
- Haemoglobin levels will be low
- Stool for microscopy and occult blood to rule out worm infestation and GIT bleeding
- Blood smear for malaria parasite

Now that you know the investigations, you should be keen to learn on treatment/ therapy of anaemia**.**

Therapy

How can anaemia be treated?

- If iron deficiency, administer Ferrous Sulphate ($FeSO_4$) 200mg three times a day (tid) for 1 month
- Folic acid 5 mg once per day (od) orally for 1 month
- Blood transfusion i.e. packed cells when Hb is very low or whole blood if the cause of anaemia is hemorrhage
- Bone marrow transplant for aplastic anaemia
- Oxygen therapy if patient is hypoxic

Ok that was good do not lose hope at least there is treatment for anaemia. You should find it interesting to learn on how to manage a patient with anaemia were need arises.

Management Of A Patient With Anaemia

Discuss the management of a patient with anaemia?

The care required by the anemic patient varies with the severity and cause of his disease. The anaemia may not be severe enough to necessitate bed rest or hospitalization. It may be chronic, as in pernicious and sickle cell anaemia resulting. Here the patients need modification in his way of life. With some patients, anaemia may be entirely cured by correction of the cause. Depending on the etiologic factor, there may be symptoms and problems in addition to those attributable to anaemia. For example, in hemolytic anaemia there may be the problem of jaundice. In the sickle cell type there is the serious problem of vascular obstruction. Regardless of the type of anaemia, the patients have one common difficulty of decreased capacity of their blood to transport oxygen.

Aims Of Nursing Management

- To eliminate the cause
- To increase the oxygen carrying capacity of blood
- To reduce demand for oxygen
- To alleviate the discomforts experienced by the patient
- To prevent complications
  To help the patient to live a life that is as useful and satisfying as his condition will permit when the disease is chronic
- **Rest:** the patient is encouraged to reduce his daily activities and rest at intervals to decrease the demand for oxygen. In severe anaemia, complete bed rest is necessary until the red blood cells and hemoglobin increase and no hypoxia. An explanation of the basis of the fatigue and weakness and the importance of rest may help the patient. Nursing care should be planned to conserve the patient's energy. There should be uninterrupted periods of rest, assistance in turning and feeding. Visits should be encouraged for brief periods only in order to ensure rest. Non exhaustive activities are encouraged.

- **Observations:** the patient with pernicious anaemia is observed for signs of degenerative changes in his nervous system. For example, tingling, numbness and sensations of "pins and needles" in distal portions of the extremities loss of finer movements. Also difficulty in holding small objects, weakness of limbs, ataxia and impaired vision should be reported to the doctor immediately. The patient's tolerance for activity in hemolytic anaemia, the colour of sclera and skin is noted daily for pallor to rule out yellowish dis colouration (jaundice).

- If the patient has sickle cell anaemia, frequent close observation is made for swollen, tender and painful areas, and changes in body functions or the patient's mental and physical abilities that may indicate areas of thrombotic disease.

Reports of laboratory studies should be followed and nursing measures adapted to indicate changes. For example, a decrease in the haemoglobin, hematocrit or erythrocyte count may be such that the patient's activity should be further reduced.

Nutrition: the diet should be light, easily digestible and selected to provide the protein, iron, vitamins and other elements necessary for the production of red blood cells and haemoglobin. Discuss with the patient the importance of foods in increasing the red cells and haemoglobin and to determine his food preferences. Offer small but frequent meals to encourage patient to eat. If the patient's mouth, tongue and oesophagus are sore, roughage and hot and spiced foods are avoided. Mouth care just before food is served, remaining with the patient; provide aid to of weak patients. A well-ventilated, neat environment that is free of commotion and disturbing sights is conducive for promoting appetite.

Extra fluids are important in haemolytic anaemia especially sickle cell, to counteract the increased blood viscosity and circulatory stagnation. The daily fluid intake is recorded. If an adequate oral intake is not tolerated, an intravenous infusion is given.

Respiratory Support: severe anaemia may cause shortness of breath or dyspnoea. The patient may be more comfortable with the head of the bed elevated and the room well ventilated. If the dyspnoea is present with the patient at rest, oxygen administration may be necessary. Sufficient assistance is given to the patient to avoid unnecessary expenditure of energy and increased oxygen demand. A blood transfusion of packed cells may be given to increase the oxygen-carrying capacity.

Mouth Care: in pernicious and iron deficiency anaemia, frequent cool, mildly alkaline mouthwashes are given to sooth the mouth. A soft bristled toothbrush or an absorbent applicator is used to clean the teeth. The mouth is cleansed before and after taking nourishment.

Skin Care: if the patient is confined to bed, his position is changed every 1 to 2 hours. Frequent pressure area care and massage is done to prevent breakage of skin. If the anaemia is hemolytic, there may be jaundice and pruritus (itching of the skin). Slightly warm or tepid water is used for bathing. Sodium bicarbonate added to the bath water may relieve irritation. The use of soap is avoided to reduce itching. The patient's finger nails are kept short and clean to prevent excoriation and infection of the skin should the patient scratch the irritated areas.

Warmth: As a result of the reduced amount of oxygen available for metabolism, the anemic patient produces less body heat. He may require extra clothing and bedding and a warm, ventilated room.

Pain and Headache: patients with anaemia frequently experience severe headache as a result of the cerebral hypoxia. Relieve symptoms by encouraging the patient to remain quiet and inactive. Environmental stimuli such as bright light and noise should be reduced to a minimum. Cold compress and analgesics such as aspirin or acetaminophen is prescribed and administered.

- Hyperplasia of the red bone marrow is associated with anaemia. The increased production of marrow cells in response hypoxia results in pressure from the volume of tissue, which causes pain. A bed cradle is used to protect the site form the weight of the bedclothes. Local heat application may provide some relief and analgesic is administered.

- **Prevention of Infection:** the patient with severe anaemia is more susceptible to infections. Reverse isolation should be considered to prevent patient from acquiring infections. Visitors and personnel with any infection, such as a cold or sore throat, should not be permitted contact with patient.

Well done you have learnt a lot, you know that blood transfusion may be required in severe anaemia. Learn and revise the nursing care in blood transfusion that you leant in detail in surgical nursing.

- **Blood Transfusion:** in severe anaemia the patient may require a blood transfusion of packed cells .But unless the anaemia is due to blood loss in which case, whole blood will be transfused in. Quarter hourly observation of the pulse and respirations should be done. Temperature and blood pressure should also be monitored. This is done to avoid dangers of circulatory overload and blood transfusion reactions.
- **Psychological Support:** the patient may be depressed and concerned about his future; the fatigue and inability to achieve prove discouraging. The nurse should listen to the patient. Provide reassurance that much is being done to restore his blood cells. Reassure the patient that everything possible will be done. Patients with chronic anaemia usually regain confidence and enthusiasm readily as they improve in the amount of energy they have and wellbeing.
- Information, Education and Communication

Nature of the condition: Give an explanation of the nature of the disorder to the family and patient. Point out that the symptoms such as weakness, fatigue, and shortness of breath are related to his anaemia. This is a resulting of deficiency of oxygen. This is done to enhance understanding and cooperation.

- **Nutrition:** The patient and family should be told on the importance of a mixed nutritious diet. Help them plan meals that help in blood formation from the local affordable but nutritious food.
- **Medication:** stress the importance of taking the prescribed medication for the length of period that they have been advised. The necessity of maintenance doses of vitamin B_{12} should be explained for patients with pernicious anaemia. Even if he feels good, he should not omit the prescribed vitamin B_{12}. In severe anaemia were a weekly injection is prescribed, referral is made to local clinic to continue getting the drug.

Follow up: if the anaemia is chronic, regular visits to the clinic is advised. The patient is advised to return to the clinic or hospital if need arises.

Prevention Of Anaemia (And Predisposing Factors): in sickle cell anaemia, the patient/parents give an explanation of the predisposing factors to crises, and how to care for the patient.

Congratulations for completing the anaemia's

It is now important to learn on nursing care of other blood disorders that can also lead to abnormal bleeding resulting into anaemia.

Nursing care of patient with haemorrhagic disorders

Discuss the nursing care of a patient with hemorrhagic disorders?

Objectives

1. To promptly identify bleeding and prevent blood loss
2. Control bleeding when it occurs.
3. Prevent complications.

Position

You have to learn control of pain as you position the patient to gain cooperation. The following would be done;

- If joint pain is present, support area with pillows. Joint immobilization and local chilling (such as packing ice around the joint) may bring relief.

  If pain is severe, inform the doctor who would be assisted to aspirate blood from the joint.

- Control topical bleeding quickly by applying pressure or ice to the injured site. Packing the area with fibrin foam, and applying topical hemostatic agents such as thrombin will control bleeding.
- Place body in good body alignment.
- Lift off linen by using a bed cradle because of painful joint.

Psychological care

You have to give psychological care to gain cooperation, so here are some ideas on what to say;

- Explain the nature, the causes, signs and symptoms of the disorder to both patient and relatives
- Inform patient about all procedures and what is expected of him.
- Inform patient the safe activities he can be involved in.
- Explain precautions to take when doing activities to minimize injury.
- Emphasize the need of the factor replacement.
- Emphasis on the abrupt action to take when bleeding occurs.
- If patient is of school going age, teachers should be informed of the individual's condition, the necessary restriction and precautions and the action to take if patient sustains injury or starts bleeding.

Observations

In order for the bleeding to be attended to on time and treatment given you have to be very observant. So the following will assist you to rule out bleeding;

- Check vital signs TPR and BP to detect any deviation from normal.
- Check for signs of cerebral dysfunction include confusion disorientation and restlessness due to bleeding into the brain.
- Look for signs of internal bleeding include pallor, weakness, air hunger, tachycardia, low BP.
- Observe open lesions, incisions, intravenous and intramuscular sites for bleeding or hematoma
- Examine and test excreta include urine, stool and vomit for blood
- Examine the joints for edema, limitation of movement and tenderness
- Inspect skin and mouth for the appearance of petechial and purple patches
- Record size and colour and location of petechial
- If on blood transfusion, carry out observations

Diet

You should encourage the patient on the following;

- To take adequate fluid and use a stool softener it necessary to prevent infection and injury to the anal mucosa.
- To eat light and well balanced diet available affordable meals.
- To avoid rough and highly seasoned foods to prevent damage to the oral and esophageal mucosa

Hygiene

You well know that hygiene is very important in our life if we want to remain health. So encourage the patient to;

- To have a daily bed baths or big baths or shower depending on the condition of the patient
- Keep a good dental hygiene to avoid tooth extraction
- Use a very soft tooth brush to prevent trauma and bleeding of gums
- Keep nails clean and short
- Handle patient gently
- Be turned 2 hourly and do pressure area care to avoid pressure sores

Drugs

You have done well so far, continue to learn on drugs that can be prescribed in hemorrhagic disorders. You must drugs give as prescribed, to the right patient, right route, right dose and at the right time .Stated are the drugs;

- Prednisolone 20-40mg BD OD effect anti inflammatory
- Vitamin K 10mg stat

- Ferrous sulphate 200mg TDSFolic acid 5mg OD
- If any infection antibiotics e.g Ampicillin
- Salicylates e.g Aspirin are contraindicated

Exercises

You should encourage the patient to exercises but the following should be considered;

- Exercises should be mild and non-traumatic
- Deep breathing and coughing should be done if patient is confined to be
- Limb exercises are essential if joints are affected.
- Once bleeding stops and swelling subsides, the client should perform active range-of-motion exercises without weight-bearing to prevent complications such as deformity and muscle atrophy.

Elimination

- You must ensure patient is opening bowel and emptying the urinary bladder. Record the amount and the colour. If patient vomits record amount and colour

Health education

You have to give health education to the patient in order for them to get prompt assistance and good care. What heath education can you give in haemolytic disorders?

- To always carry identification band and the blood type
- To seek prompt treatment following trauma and earliest symptom of bleeding
- Avoid forceful sneezing and coughing and to maintain adequate fluid to keep nasal and bronchial mucosa moist
- Need to prevent injury
- Avoid sport and occupation that present the risk of trauma
- Provide information on the availability of the community health nurse
- Need for frequent medical check up
- Importance of taking drugs
- Avoid wearing tight clothing
- Provide genetic counselling especially haemophilia

Heamorrhagic Disorders

These disorders have an abnormal tendency to bleed. Hemorrhagic disorders can be classified into 3 major groups:

1. ***Disorders of the thrombocytes*:** conditions where there are reduced thrombocytes e.g. thrombocytopenia, thrombocythaemia and thrombosthenia
2. ***Disorders due to defects in clotting mechanism*:** conditions such as hemophilia, prothrombin deficiency, oral anticoagulant therapy and advanced hepatic failure.
3. ***Disorders due to defects of the blood vessels*:** this type of disorders can be caused by rage of other conditions.

the following haemorrhagic disorders:

-
-
- Haemophilia (Rare Condition)
- Ebola Virus

THROMBOCYTOPENIA

Thrombocytopenia is defined as a lower than normal number of circulating platelets (Ranges of 150 000 to 400 000).

Thrombocytopenia is present when there is a significant decrease in the number of thrombocytes. They are destroyed as soon as they are produced or because they are not being produced.

Disorders due to defects of the blood vessels: this type of disorders can be caused by:

a. A wide range of infections, e.g. measles, typhoid fever and septicaemia
b. Chemical agents may also be responsible for damaging vessels e.g. aspirin, frusemide, indomethacin, phenytoin and snake venom.
c. Anaphylactic reactions (Henochschonlein purpura)
d. Conditions such as hepatitis and renal failure

The normal lifespan of platelets is approximately 8 to 10 days. In thrombocytopenia it is only 1 to 3 days.

Aetiology

- Decreased Platelet production
- *inherited*
- *Acquired*
- Aplastic anaemia
- Haematologic malignant disorder
- Myelosuppressive drugs
- Chronic alcoholism
- Exposure to ionizing radiation
- Viral infections

Deficiencies of cobalamin and folic acid

1. Increased Platelet Distribution
   a. *Non immune*

- Thrombotic thrombocytopenia purpura
- Pregnancy
- Infection
- Drug induced
- Severe burns

b. *Immune*
- Immune thrombocytopenic purpura
- Human immunodeficiency virus infection
- Drug induced

c. *Splenomegally*

2. Drugs, spices and vitamin causing abnormalities in platelet function
   a. *Suppression of platelet production*
      - Thiazide diuretics, alcohol, oestrogen and chemotherapeutic drugs

   b. Abnormal platelet aggregation
      - NSAIDS: Ibuprofen, indomethacin naproxen
      - Antibiotics: Penicillin and cephalosperins
      - Analgesics: Aspirin and aspirin containing drugs
      - Spices: Ginger, cumin, turmeric, cloves and garlic
      - Vitamins: Vitamin C and vitamin E.
      - Heparin
      - Other drugs, chloroquine, digitoxin, methyldopa, oral hypogycaemic agents, phenorbital, quinidone, quinine, refampicin

PROTHROMBIN DEFICIENCY

Also refers hypothrombinaemia or vitamin K deficiency.

Vitamin K is essential for the production of prothrombin by the liver. The deficiency is due to the following:

- Inadequate intake
- Impaired absorption
- Defective utilization of vitamin K

If the diet does not supple the adequate amount of vitamin K, it is synthesized by the normal flora of the intestinal tract.Sterilisation of bowel by drugs e.g Neomycin may produce vitamin K deficiency.

Bile salts are essential in the intestines for the absorption of vitamin K.A commonest cause of prothrombin is the obstruction of flow of bile into the intestine. Liver disease may cause reduction of prothrombin.

Treatment

1. Give vitamin K supplements

2. Blood transfusion maybe indicated especially in liver disease.
3. Complete best rest
4. Protect from injury.

Nursing Care of a Patient With Haemorrhagic Disorders

Objectives

The objectives of nursing care are to:

4. Promptly identify bleeding and prevent blood loss
5. Control bleeding when it occurs.
6. Prevent complications.

Position

You should control the pain as you position the patient in order to gain their cooperation. In addition, you should do the following:

- If joint pain is present, support the area with pillows. Joint immobilization and local chilling (such as packing ice around the joint) may bring relief.

  If pain is severe, inform the doctor who may aspirate blood from the joint to relieve the pain;

- Control topical bleeding quickly by applying pressure or ice to the injured site. Packing the area with fibrin foam and applying topical hemostatic agents such as thrombin will control bleeding.
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- Explain precautions to take when doing activities to minimize injury
- Emphasize the need of the factor replacement
- Emphasis on the abrupt action to take when bleeding occurs

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- Examine the joints for oedema, limitation of movement and tenderness
- Inspect skin and mouth for the appearance of petechial and purple patches
- Record size and colour and location of petechial

Diet

You should encourage the patient to:

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- Eat light and well balanced diet available affordable meals.
- Avoid rough and highly seasoned foods to prevent damage to the oral and esophageal mucosa

Hygiene

You well know that hygiene is very important in our life. Therefore you should encourage the patient to:

- Have daily bed baths or big baths or showers depending on the condition of the patient
- Keep a good dental hygiene to avoid tooth extraction
- Use a very soft tooth brush to prevent trauma and bleeding of gums

Keep nails clean and short

In addition, you should handle patient gently and turn them 2 hourly to avoid pressure sores.

Drugs

You must give drugs as prescribed, to the right patient, right route, right dose and at the right time. The main drugs include:

- Prednisolone 20-40mg BD OD effect anti inflammatory
- Vitamin K 10mg stat
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- If any infection antibiotics e.g Ampicillin
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You should encourage the patient to exercises but the following should be considered:

- Exercises should be mild and non-traumatic
- Deep breathing and coughing should be done if patient is confined in bed
- Limb exercises are essential if joints are affected.
- Once bleeding stops and swelling subsides, the client should perform active range-of-motion exercises without weight-bearing to prevent complications such as deformity and muscle atrophy.

Elimination

You must ensure that the patient is opening bowel and emptying the urinary bladder. Record the amount and colour. If the patient vomits you should record the amount and colour

Health Education

Give health education to the patient in order for them to get prompt assistance and good care. In cases of haemolytic disorders, you should give the following information:

To always carry an identification band and their blood type

- To seek prompt treatment following trauma and earliest symptom of bleeding
- To avoid forceful sneezing and coughing and to maintain adequate fluid to keep nasal and bronchial mucosa moist
- Need to prevent injury
- To avoid sport and occupation that present the risk of trauma
- Need for frequent medical checkups
- Importance of taking drugs
- To avoid wearing tight clothing

- Provide genetic counselling especially in cases of haemophilia

What is haemophilia, let us look at that next.

HAEMOPHILIA (rare condition)

What is haemophilia?

Haemophilia is a coagulation conditions that occurs as a result of deficiency of factor VIII (anti haemophilic factor) or factor IX (Christmas factor).

It is a hereditary blood disease characterized by greatly prolonged coagulation time.

Transmission

Haemophilia is transmitted through:

- The female has xx chromosomes
- The male has xy chromosomes

If a girl inherits an x chromosome bearing the haemophilic gene from the father she becomes a carrier. The disorder is not manifested in her because the X chromosome with the abnormal gene is not from the mother.

If a carrier marries a known haemophilic then:

- There will be 25% chance that her daughters will be normal
- There will be 25% chance that her daughters will be carriers
- There will be 25% chance that her sons will be normal
- There will be 25% chance that her sons will be carriers

The sons of a haemophilic father are all normal because they receive a y chromosome from him.

Clinical Features

What are the signs and symptoms of haemophilia? Think about it for a minute and then complete the following activity.

Activity

Write down two signs and symptoms of haemophilia in your notebook.

We hope your list included the following clinical features of haemophilia:

- Excessive bleeding after a slight injury or after tooth extraction or even brushing of the teeth with a hard tooth brush. The characteristic of bleeding is persistent rather than the amount.
- Bleeding into the joints especially knees, ankles and elbow.
- Pain in the affected joints.
- Defect in the affected joints
- Recurrent hematoma formation in deep subcutaneous tissue of intramuscular muscle (muscular atrophy sometimes result) and around the peripheral nerves. The hematoma may cause compression that can result in severe pain, anaesthesia of the innervated part, nerve damage, and paralysis.
- Haematuria (blood in urine) from genitourinary trauma.
- Splenic rupture from falls or abdominal trauma
- Intracranial bleeding, which is the leading cause of haemorrhagic death
- Delayed haemorrhage that follows minor injuries, bleeding may not start on the site until hours or even days after the traumatic event.
- Severe, sometimes fatal, epistaxis (nose bleeding) after injury to the nose.
- Overwhelming gastric haemorrhage, this may be linked to gastric disorders such as ulcers and gastritis.

Investigations

The following investigations can be conducted to rule out haemophilia:

- Prolonged bleeding time
- Prolonged clotting time
- Platelet count is normal.

Treatment

The following is the treatment for haemophilia:

- If factor VIII is low or factor IX, it's replaced by factor VIII or factor IX by infusion. Fibrinogen can also be used. Vasopressin may be administered as it causes constriction of smooth muscles.
- During an episode of bleeding the patient should be on complete bed rest. Immobilize the joints if there is bleeding into the joints. The proper positioning of limbs in good alignment to reduce the possibility of deformity should be done.

Complications

The following are the complications of haemophilia:

- Antibodies to the artificial factor concentrate. Ankylosis-stiff joint
- Abnormal immobility and fixation of a joint due to its pathological changes in the joint or its surrounding tissue. This occurs mainly due to bleeding into joints(haemothroses)

You now know about haemophilia, next let us discuss the dreaded ebola virus

EBOLA VIRUS

You have heard about this rare condition on the news. Now is your chance to learn what it is. You will begin to learn on the structure of the ebolavirions that cause ebola

Ebolavirion Structure

Like all filoviruses, ebolavirions are filamentous particles that may appear in the shape of a shepherd's crook or in the shape of a "U" or a "6". They may be coiled, toroid, or branched. Ebolavirions are generally 80 nm in width, but vary somewhat in length.The median particle length of ebolaviruses ranges from 974–1,086 nm (in contrast to marburgvirions, whose median particle length was measured to be 795–828 nm).In some cases particles as long as 14,000 nm have been detected in tissue culture. Ebolavirions consist of seven structural proteins. The ribonucleoprotein is embedded in a matrix, formed by the major (VP40) and minor (VP24) matrix proteins. These particles are surrounded by a lipid membrane derived from the host cell membrane. The membrane anchors a glycoprotein (GP1,2) that projects 7 to 10 nm spikes away from its surface. While nearly identical to marburgvirions in structure, ebolavirions are antigenically distinct.

Replication

The ebolavirus life cycle begins with virion attachment to specific cell-surface receptors. Then fusion of the virion envelope with cellular membranes and the concomitant release of the virus nucleocapsid into the cytosol.Viral RdRp partially uncoats the nucleocapsid and transcribes the genes into positive-stranded mRNAs.Then it is translated into structural and nonstructural proteins. Ebolavirus L binds to a single promoter located at the 3' end of the genome. Transcription either terminate after a gene or continues to the next gene downstream. Replication results in full-length, positive-stranded antigenomes that are in turn transcribed into negative-stranded virus progeny genome copy. Newly synthesized structural proteins and genomes self-assemble and accumulate near the inside of the cell membrane. Virions bud off from the cell, gaining their envelopes from the cellular membrane they bud from. The mature progeny particles then infect other cells to repeat the cycle.

Epidemiology

What is epidemiology of EDV

Outbreaks of EVD have mainly been restricted to Africa. Governments and individuals quickly respond to quarantine the area while the lack of roads and transportation helps to contain outbreak. EVD was first described after almost simultaneous viral haemorrhagic fever outbreaks occurred in Democratic Republic of Congo and Sudan 1976. EVD is believed to occur after an ebolavirus is transmitted human index case via contact with an infected animal host. Human-to-human transmission occurs via direct contact with blood or bodily fluids from an infected person (including embalming of a deceased victim).It can also occur by contact with contaminated medical equipment such as needles. Aerosol transmission has not been observed during natural EVD outbreaks. The potential for widespread EVD epidemics is considered low due to the high case-fatality rate. The rapidity of demise of patients, and the often remote areas where infections occur also contributes.

Pathophysiology

You must be happy to learn the advantage of being in a third well. The spread EVD is controlled .What is the pathophysiology of Ebola?

Endothelial cells, mononuclear phagocytes, and hepatocytes are the main targets of infection. After infection, in a secreted glycoprotein (sGP) the Ebola virus glycoprotein (GP) is synthesized. Ebola replication overwhelms protein synthesis of infected cells and host immune defenses. The GP forms a trimeric complex, which binds the virus to the endothelial cells lining the interior surface of blood vessels. The sGP forms a dimeric protein which interferes with the signalling of neutrophils, a type of white blood cell. This allows the virus to evade the immune system by inhibiting early steps of neutrophil activation. These white blood cells also serve as carriers to transport the virus throughout the entire body to places such as the lymph nodes, liver, lungs, and spleen. The presence of viral particles and cell damage resulting from budding the release of cytokines result into fever and inflammation. The cytopathic effect, from infection in the endothelial cells, results in a loss of vascular integrity. This loss in vascular integrity is furthered with synthesis of GP, which reduces specific integrins .This is responsible for cell adhesion to the inter-cellular structure, and damage to the liver, which leads to coagulopathy.

Investigations/Diagnosis

What are some investigations that can lead to diagnosis EVD?.

EVD is clinically indistinguishable from Marburg virus disease (MVD).It can also easily be confused with many other diseases prevalent in Equatorial Africa. Following are examples of these disease; viral hemorrhagic fevers, falciparum malaria, typhoid fever and shigellosis. Others include rickettsial diseases such as typhus, cholera, gram-negative septicemia, borreliosis such as relapsing fever or EHEC enteritis.

The most important indicator that may lead to the suspicion of EVD at clinical examination is the medical history of the patient, in particular the travel and occupational history (which countries were visited?). Also the patient's exposure to wildlife (exposure to bats, bat excrement, nonhuan primates?). EVD can be confirmed by isolation of ebolaviruses from or by detection of

ebolavirus antigen or genomic or subgenomic RNAs in patient blood. Also it would be through serum samples during the acute phase of EVD. Ebolavirus isolation is usually performed by inoculation of grivet kidney epithelial Vero E6 or MA-104 cell cultures. But also by inoculation of human adrenal carcinoma SW-13 cells, all of which react to infection with characteristic cytopathic effects. Filovirions can easily be visualized and identified in cell culture by electron microscopy due to their unique filamentous shapes. The electron microscopy cannot differentiate the various filoviruses alone despite some overall length differences.

Immunofluorescence assays are used to confirm ebolavirus presence in cell cultures. During an outbreak, virus isolation and electron microscopy are most often not feasible options.

Non-infectious diseases that can be confused with EVD are acute promyelocytic leukemia, hemolytic uremic syndrome, snake envenomation. Others are clotting factor deficiencies/platelet disorders, thrombotic thrombocytopenic purpura, hereditary hemorrhagic telangiectasia, Kawasaki disease, and even warfarin intoxication.

Treatment

What is the treatment of EVD?.

There is currently no-approved ebolavirus-specific therapy for EVD. Treatment is primarily supportive in nature and includes;

- Minimizing invasive procedures.
- Balancing fluids and electrolytes to counter dehydration.
- Administration of anticoagulants early in infection to prevent or control disseminated intravascular coagulation.
- Administration of procoagulants late in infection to control hemorrhaging.
- Maintaining oxygen levels
- Pain management

Administration of antibiotics or antimycotics to treat secondary infections.

Prognosis

What is the prognosis of EVD?

Prognosis is generally poor (average case-fatality rate of all EVD outbreaks to date = 68%). If a patient survives, recovery may be prompt and complete. Also protracted with sequelae, such as orchitis, arthralgia, myalgia, desquamation or alopecia. Ocular manifestations, such as photophobia, hyperlacrimation, iritis, iridocyclitis, choroiditis and blindness have also been described. EBOV and SUDV can persist in the sperm of some survivors giving rise to secondary infections and disease via sexual intercourse.

Prevention

- Ebola viruses are highly infectious as well as contagious so prevention is said to be better than cure. What is the prevention of EVB?
- As an outbreak of ebola progresses, bodily fluids from diarrhea, vomiting, and bleeding represent a hazard.
- Do not share needles or use without adequate sterilization procedures.

Isolate patients, and observe strict barrier nursing procedures with the use of a medical-rated disposable face mask, gloves, and goggles. Wear a gown at all times, strictly enforcedfor all medical personnel and visitors. The aim of techniques is to avoid any person's contact with blood or secretions of any live or dead patient.

Vaccines have successfully protected nonhuman primates; however, the six months needed to complete immunization made it impractical in an epidemic.

The natural maintenance hosts of ebolaviruses remain to be identified. This means that primary infection cannot necessarily be prevented in nature. The avoidance of EVD risk factors, such as contact with nonhuman primates or bats, is highly recommended. Unfortunately this may not be possible for inhabitants of tropical forests or people dependent on nonhuman primates as a food source.

Prevention During outbreaks

What is the prevention of EVD?

Since Ebola viruses do not spread via aerosol, the most straightforward prevention method during EVD outbreaks is to;

- Avoid direct (skin-to-skin) contact with patients, their excretions and body fluids.
- Patients should be isolated and medical staff be trained and apply strict barrier nursing techniques. They include wearing of disposable face mask, gloves, goggles, and a gown at all times.

    Traditional burial rituals, especially those requiring embalming of bodies, should be discouraged.

Disorders Of The White Blood Cells (Wbc)

Congratulations that you continue learning. You are now going to learn on disorders of WBC .What are some common disorders of the WBC?

- Leukemia
- Lymphoma
- Hodgkin's lymphoma

LEUKAEMIA

You will start learning leukaemia and then proceed to other conditions of the white blood cell. I am sure you have heard of people who have suffered from Leukemia or you have seen some patients with Leukaemia on the ward.

In text question

Define leukaemia in your own words.

DEFINITION

Now that you have attempted to define leukaemia, let's see how others have defined it.

Leukemia is a malignant disease of the blood forming organs *(Black and Hawks, 2009*).

Leukemia is the general term used to describe a group of malignant disorders affecting the blood and blood- forming tissues of the bone marrow, lymph system and spleen (*Lewis et al, 2004).* Very exciting I am sure you will continue to learn on the causes and predisposing factors

Causes

What are the causes of leukemia?

The cause of leukaemia is idiopathic (unknown).

However there are some predisposing factors. Let us discuss these in detail.

Predisposing Factors

What are the predisposing factors of leukemia?

Leukaemia, like other cancers, results from mutations in the DNA. Certain mutations can trigger leukaemia by activating oncogenes (tumor cells) or deactivating tumour suppressor genes, and thereby disrupting the regulation of cell death, differentiation or division. These mutations may occur spontaneously or as a result of exposure to the following:

1. Genetic factors
2. Exposure to ionizing radiation and chemicals
3. Congenital abnormalities (e.g Downs Syndrome)
4. The presence of primary immunodeficiency and infection with the human T-cell leukemia virus type 1(HTLV-1)
5. Infectious agent- viruses like retrovirus
6. Chemotherapy treatment e.g cytotoxic drugs (vincristine)
7. Myloproliferative disorders-fibrosis of the bone marrow.
8. Excessive use of drugs e.g. sulphonamide

Classification

Classification of leukemia can be done based on;

1. Duration and character of disease that is acute or chronic.

The terms acute and chronic refers to the cell maturity and nature of disease onset. Chronic leukemia involves more mature forms of WBC and the disease is more gradual.

2.

3. **The type of white blood cells involved, that is myeloid, (myeloblastic or granulocytic) and lymphoid (lymphocytic, lymphoblastic).**

There are four types of Leukaemias and these include:

- **Acute non lymphoid (myelogenous) leukaemia**
- **Acute lymphocytic leukaemia**

- **Chronic myelogenous leukaemia**
- **Chronic lymphocytic leukaemia**

Acute non lymphoid (myelogenous) leukaemia

Acute myelogenous leukemia is a disorder of the hematopoietic progenitors caused by acquired oncogenic mutations that hinder differentiation (separation), leading to the accumulation of immature, non functional myeloblasts in the marrow.

There is hyperplasia (increase in size) of the bone marrow and spleen. Acute myelogenous leukaemia represents only ¼ of all leukemias and accounts for 85% of the acute leukemias in adults.

Its onset is often abrupt and dramatic. A patient may have serious infections and abnormal bleeding from the onset of the disease.

The clinical manifestations are usually related to the replacement of normal hematopoietic cells in the bone marrow by leukaemic myeloblasts and to a lesser extent, to infiltration of other organs. This leads to pancytopaenia (reduction of all cellular components in the blood).

Acute lymphocytic leukaemia (ALL)

Acute lymphoblastic leukaemia comprises a group of clonal disorders of lymphopoietic stem cells characterized by the accumulation of lymphoblasts. This is the most common type of leukaemia in children and it accounts for about 15% of acute leukaemias in adults.

In ALL immature lymphocytes proliferate (multiply) in the bone marrow. Signs and symptoms may appear abruptly with bleeding, fever, may be insidious with progressive weakness, fatigue and bleeding tendencies. Fever is present in the majority of patients at the time of diagnosis.

Chronic myeloid (myelogenous) leukaemia (CML)

Chronic myelogenous leukaemia is also termed chronic granulocytic leukaemia (CGL). There is excessive development of mature neoplastic granulocytes in the bone marrow. The excess neoplastic granulocytes move into the peripheral blood in massive numbers and ultimately infiltrate (penetrate) the liver and spleen causing hepatomegaly and splenomegaly.

The disease is most common in young and middle aged adults and slightly more common in men that women, but rare in children.

The clinical course of CML proceeds in two distinct phases:

-
- - *insidious chronic phase*, characterized by anaemia and bleeding abnormalities
- - *acute phase* (blast crisis) in which myeloblasts, the most primitive granulocytic precursors, proliferate rapidly.

Clinical features includes mild to moderate anaemia and hyper metabolism due to increased cell turnover leading to:

- fatigue
- weakness
- weight loss
- anorexia.

The patient may also complain of pain in the left upper quadrant due to spleenic infarction. There may also be dragging sensation in the abdomen due to splenomegaly.

Chronic Lymphocytic leukaemia (CLL)

Chronic lymphocytic leukemia is characterized by the production and accumulation of functionally inactive but long- lived mature appearing lymphocytes. The type of lymphocytes involved is usually the B- cells. The lymphocytes infiltrate the bone marrow, spleen and the liver. Lymphnodes enlargement (lymphadenopathy) is present throughout the body and there is an increased incidence of infection.

B-cell CLL is considered to be identical to the mature B- cell small lymphocytic lymphoma, a type of non- Hodgkin's lymphoma. Pressure on the nerves from the enlarged lymph nodes causes pain and even paralysis. Mediastinal node enlargement leads to pulmonary symptoms. CLL is a slow growing leukaemia that is seen most often in men order than 50 years of age.

Pathophysiology

Blood cells grow from a single precursor cell, known as a hematopoietic stem cell. These stem cells divide to become progenitor blood cells, which undergo further specializations to become red blood cells, white blood cells, or platelets. This happens in the bone marrow, the spongy tissue in the centre of bones.

In normal bone marrow, efficient regulation ensures that cell proliferation and maturation are adequate to meet a person's needs.

Leukemia begins when a progenitor cell divides uncontrollably and overriding the body's normal restrictions on cell division. Although not a tumor (not a growth), leukaemic cells demonstrate the same neoplastic properties as solid cancers. Therefore the resulting pathologic condition and the clinical manifestation are caused by infiltration and replacement of any tissue of the body with non functional leukaemic cells.

In all types of leukaemia the proliferating cells depress the production of formed elements of the blood in the bone marrow by competing for and depriving the normal cells of the essential nutrients for metabolism.

The most frequent presenting signs and symptoms of leukaemia are a result of infiltration of the bone marrow. The three main consequences are; anaemia from decreased red blood cells, infection from neutropenia and bleeding from decreased platelet production. The invasion of the bone marrow with leukemia cells gradually causes a weakening of the bone and a tendency towards fractures.

As leukaemic cells invade the periosteum increasing pressure causes severe pain. The spleen, liver and lymph glands demonstrate marked infiltration, enlargement and eventually fibrosis (scar formation).

The next most important site of involvement is the central nervous system (CNS) which may cause increased intracranial pressure. Leukemic cells may also invade the testes, kidneys, prostate, ovaries, GI tract and lungs.

Clinical manifestation

Clinical manifestations of leukaemia are varied, essentially they relate to problems caused by bone marrow failure and the formation of leukemic infiltrates.

Bone marrow failure results from:

- **Bone marrow overcrowding**
- **Inadequate production of normal marrow elements**

The patient may present with:

- Anaemia, Hemorrhage (petechia) due to thrombocytopenia
- Pallor, fatigue due to anaemia
- Fever due to neutropenia
- Tendency to fracture
- Pain due to enlarged liver or spleen
- Severe headache due increased intracranial pressure as a result of meningeal infiltration and spread
- Vomiting, anorexia, irritability, lethargy, papiloedema due to increased intracranial pressure
- Bone pain due to infiltration of bones
- Meningeal irritation due to infiltration of the brain
- Solid masses resulting from collection of leukaemic cells
- Hepatomegaly, splenomegaly, lymphadenopathy

Diagnostic evaluation

Getting a correct diagnosis is very important so that treatment is instituted promptly. Some investigations that will be done are:

1. **Complete Medical history – Ask about the presenting signs and symptoms and any existing genetic conditions.**
2. **Physical examination – A thorough head to toe examination is done, look for enlarged lymph nodes, enlarged lymphoid tissues like the spleen, liver; petechia (tinny red spot on the skin) that are due to ecchymosis (bleeding from broken blood vessels into surrounding).**
3. **Bone marrow aspiration or biopsy is a key diagnostic tool for confirming the diagnosis and identifying the malignant cell type. Typical findings are the increase in the number of the marrow cell especially immature cells.**
4. **Lumbar puncture- to determine meningeal involvement**
5. **Computed Tomography (CT) scan shows the affected organs**
6. **Cerebral spinal fluid analysis detects abnormal WBC invasion of the central nervous system**
7. **Blood examination – shows anaemia and reveals immature forms of leucocytes**

Medical management

Aim – Restoration of normal bone marrow functioning

Treatment modalities

There are 3 modalities namely;

- **Surgery**
- **Radiotherapy**
- **Chemotherapy**

Surgery

Surgery is done to remove the bulk of the tumor. Bone marrow transplantation and stem cell transplantation are done to eliminate leukemic cell from the body. Surgery can also relieve pain and obstruction and alleviate pressure. Surgery is combined with other treatment modalities like radiation chemotherapy and immunotherapy

Radiotherapy

This is the treatment of cancer by the use of radiations which destroy the cancer cells. It may be used in combination with surgery or following surgery. There are two kinds of radiation used;

Particle radiation- this uses alpha and beta particles.

Beta particles are electrons with higher energy and can penetrate anything up to the density of wood.

Alpha particles are weak and have low energy.

Electromagnetic radiation-this uses Gamma rays and X- rays.

They are similar to light or radioactive rays, but have a much higher energy level. They have the ability to penetrate deeply into the body tissues hence they are widely used.

Chemotherapy

This is treatment of cancer by the use of chemical which refers to any form of drug therapy. There are different groups of drugs that are used in the treatment of leukaemia. Let us discuss some of them in detail.

***Alkylating agents*- These drugs are the most widely used in cancer therapy.**

They act by damaging the DNA molecule of the nucleus of the cell, thus interfering with cell replication. eg.chlorambucil, cyclophosphomide, ifosfamide and busulfan

***Antitumor antibiotics*- These are cell cycle non- specific.**

They interfere with DNA function as well as alteration of the cell membrane or inhibition of certain enzymes; Examples are Actinomycin, daunorubcin, doxorubicine.

A*n*timetabolites - These are combined irreversibly with vital cellular enzymes thus preventing normal cellular division. E.g. methotrixate, fludarabin and cystosine Arabinosine

***Vinca alkaloids*- They act by interfering with DNA replication by binding to the DNA. Example Vincristine (Oncovin) and Vinblastine.**

***Steroids* – Act by altering the DNA transcription process, e.g predinisolone**

Chemotherapy – chemotherapy is given to destroy the malignant cells of the bone marrow the treatment protocol of acute leukaemia may involve up to 3 phases; induction phase, consolidation phase and maintenance phase which is usually used in ALL.

Induction phase –the client receives an intensive course of therapy designed to induce complete remission.

Consolidation phase- modified courses of intensive chemotherapy are given to eradicate any remaining disease. Usually one or more chemotherapeutic agents are administered.

Maintenance phase- small doses of different combinations of chemotherapeutic agents are given every 3 to4 weeks.

This phase may continue for a year or longer and is structured to allow the client to live as normal life as possible this phase is used more commonly with ALL.

Treatment is dependent on the type of leukemia

Treatment of Acute lymphocytic leukemia

Treatment for ALL is a combination of vincristine, doxorubicin, daunorubicin and predinisolone. The patient also receives intrathecal methotrixate because there is a risk of meningeal infiltration.

Radiation therapy is also given if brain and testicular infiltration occur.

Treatment for acute myelogeous leukaemia

Treatment consists of Intravenous daunorubicine, and cytarabine. If these fail to induce remission, treatment involves some or all of the following; combination of cyclophosphamide, vincristine, predinisolone and methotrixate.

- Other treatments include antibiotics or antifungal to control infection.

- Transfusion of platelets to control bleeding

- Bone marrow and stem cell transplant is performed in some patients

Treatment of chronic myelogenous leukaemia

Treatment of CML is usually divided into four areas;

- **Bone marrow and Stem cell transplantation**
- **Leukapheresis (selective leukocyte removal)**
- **Interferon alfa therapy with or without chemotherapy**
- **Single agent chemotherapy hydroxyurea**

Treatment of Chronic Lymphocytic Leukemia

The goal of therapy in CLL is palliation or control of undesired manifestation.

Drugs given are Chlorambucil, cyclophosphamide and prednisolone may be given orally to reduce the manifestation of CLL.

Chemotherapy is generally given for two weeks of every month.

Radiation to the body, Lymphnodes or spleen may be performed as a palliative treatment to reduce complication such as hemolytic anaemia resulting from autoimmune disorders and hypogammaglobulinemia**Splenectomy may also be done.**

Complications

- Hemolytic anaemia
- Lowered immunity
- Metastasis
- Obstruction
- Depression

Nursing Management Of A Patient With Leukemia

Aims

- To reduce haemorrhage/prevent haemorrhage
- To reduce and prevent infection
- To relieve effects of anaemia
- To minimize effects of treatment such as alopecia, nausea and vomiting, anorexia, stomatitis and mouth ulcers, diarrhoea or constipation, renal damage, bone marrow suppression.

Observations

- Full blood count, haemoglobin estimation
- Check vital signs 4 hourly
- Observe patient for indications of infection, haemorrhage, disorientation and loss of coordination
- Infection is indicated by increased temperature, increased pulse and respirations, a cough or a skin lesion
- Haemorrhage may be detected by blood in the urine or stools, petechiae, ecchymoses, bleeding gums, hemetemesis, or rapid weak pulse and a fall in blood pressure
- Assess patient's emotional status daily and amount of support and attention needed in this area determined
- Fluid intake and output are recorded and the balance determined

Rest

Unless the patient is in remission, rest and the prevention of unnecessary expenditure of energy are important because of the increased metabolic rate associated with the rapid, excessive production and destruction of leukemic cells. The patient is also experiencing some degree of hypoxia because of anaemia. The acutely ill patient requires assistance in turning and moving.

Blood transfusion

Transfusions of whole blood may be given if there has been bleeding. If there has been no marked loss of blood and no decrease in the circulatory volume, transfusions of packed red blood cells are used to relieve the anaemia and of platelets to control and prevent bleeding. The patient is observed closely for reactions during and immediately following the transfusions. A reaction may be manifested by a chill and fever, urticaria, severe headache, lumbar pain, dyspnea, oliguria, and dis colouration of the urine due to hemoglobin released by hemolysis. If a reaction occurs the transfusion is discontinued and the physician notified promptly.

Protection from infection

- Reverse isolation technique is used. The patient is cared for in a room with filtered air to reduce the possibility of airborne infection.
- No one with an infection should be allowed to visit or care for the patient
- Daily examination of the skin for possible lesions

- Regular and frequent recording of the oral or axillary temperature and prompt reporting of and elevation
- Avoidance, if possible, of parenteral administration of medications, but if it is necessary, special cleansing and protection of the skin before and after
- Avoidance of taking rectal temperature to prevent mucosal damage that provides an entry for organisms
- Antiseptic mouth care and gentle cleansing of the teeth with a soft brush to avoid mucosal damage and ensuing infection
- Daily bathing with a mild antiseptic soap to reduce the skin flora (the patient's own body flora of the skin, mouth, nose and intestinal tract is frequently the major source of infection)
- Keeping the nails short and clean
- The avoidance of constipation and the giving of an enema to prevent possible rectal mucosal trauma. The stool may be kept soft by fruit and plenty of fluids in the diet. If necessary a stool softener may be given
- If a local infected area develops, a culture is made immediately

Fluids

A minimum fluid intake of 2500 – 3000 mls/24hrs is encouraged to promote elimination of increased serum uric acid by the kidneys. This results from the rapid leukemic cell destruction and, unless diluted, may crystallize in renal tubules, blocking them and impairing kidney function. An adequate fluid intake is also important if the patient's temperature is elevated. Intravenous infusion is preferred to provide adequate hydration.

Nutrition

Nutrition may be a problem with leukemic patient; he has anorexia and his mouth may be very sore. A high-calorie, high vitamin diet is desirable; the overproduction of cells makes an excessive demand on body nutrients. The patient's preferences and what can be tolerated are determined from day to day. Bland, non-irritating concentrated foods and nutritious fluids are used whenever possible. Cold or iced preparations are usually acceptable and less irritating to the mouth. When there is somatitis, rinsing the mouth with an anesthetic mouth was just before each meal may reduce the discomfort associated with eating.

Oral hygiene

Frequent special mouth care is very important because of the patient's fever, susceptibility to infection, haemorrhage, stomatitis and ulceration that are frequent side effects of several antileukemic drugs. The mouth is rinsed every 2 hours with a mildly alkaline mouth wash. A very soft-bristled toothbrush is used to gently clean the teeth. If the mouth is very sore an irrigation setup (solution container and tube) may be necessary, with the patient in lateral position or with the head and shoulders elevated and forward to prevent aspiration. An emollient or oil may be applied to the lips to prevent cracking and adherence.

Skin care

Because of the fever, weakness, and susceptibility to infection, the patient's skin requires special attention. Frequent bathing is necessary to remove perspiration and organisms and provide comfort. A mild antiseptic soap may be used for its antibacterial effect. The patient's position is changed frequently to prevent pressure sores and other complications. The alternating air pressure mattress (ripple mattress) is very useful. Extreme gentleness is required when doing anything for the patient to avoid pressure that might precipitate bleeding into the tissues. Following an injection, pressure is applied to the site for several minutes to reduce the amount of bleeding and the formation of a haematoma.

Relief of pain

The patient may experience bone pain because of the marrow hyperplasia and pain in viscera that are enlarged and swollen by infiltration by leukemic cells. Severe headache accompanies infiltration of the brain. Local application of ice packs may be used over the painful areas and a cradle will protect the parts from the pressure and weight of bedding. The administration of an analgesic may be necessary to provide relief, particularly as the disease becomes progressively more severe.

Because of the patient's fever and lowered heat tolerance (due to increase metabolic rate) and shortness of breath (due to anaemia), a cooler well ventilated environment is more comfortable for him.

Psychological support

The diagnosis of acute leukemia is very difficult for the patient and family to accept. There may be situations in which there is still some question as to whether the patient should be told that he has leukemia. The nature of disease and treatment should be discussed frankly with the patient as well as the family. The nurse must know what information they have received from the physician. The therapeutic program requires cooperation and participation by both. They are advised that the blood disorder is serious but drugs are available that control the problem for most patients.

Information, Education and Communication during remission

1. The patient is encouraged to resume as normal a pattern of life as possible. The doctor will indicate if he may return to his former employment. The employer or teacher is advised of the individual's limitations e.g. continuity of therapy will mean frequent absence from work.
2. The family is advised to guard against over protecting the patient. Promoting his independence within the limits of his ability is in his best interests as well as those of all members of the family
3. Information is given to the family about the importance of doing all they can to prevent infection, since and infection that would be a minor incident to a normal person could be very serious for the leukemic person. Early signs and symptoms of respiratory and skin infections and of gastrointestinal and urinary disturbances are reviewed. Hygiene of the mouth, cleansing of the teeth with a soft brush, bathing and keeping the nails short and clean

to prevent infection is outlined. Avoidance of exposure to or contact with persons with infection is stressed. They are advised that if a member of the family develops a cold or infection, he should not come near the patient. His dishes should be disinfected and his clothing and linen kept separate when laundered.

4. Chemotherapeutic schedule is reviewed and given in writing. Symptoms of potential toxic effects of the prescribed drugs are cited and the patient advised what to do if these reactions occur. Emphasis is placed on the need for the patient to continue the antileukemic drugs even though he feels good between doses. It is stressed that he should not take any drugs except those prescribed by the doctor. Aspirin is not taken because of its potential to increase the bleeding tendency.
5. The importance of keeping clinic or medical review is explained. These are necessary for the patient to receive an intravenous drug that is part of the chemotherapy regime. Blood counts are done on these visits because a change in cell count is usually the first indication of the onset of a relapse. It is suggested that it is well to have a family member accompany him to the clinic; the relative learns what the patient experiences, and the doctor may want to discuss the patient's progress and activities with him.
6. A well balanced nutritious diet is discussed, as is the need for the inclusion of fruit and vegetables to prevent constipation and the need for a laxative. A daily fluid intake of 2500 to 3000mls is indicated to promote normal bowel and renal function
7. If the patient has experienced alopecia he will be faced with the surprise and comment of fellow workers or students. Suggestions are made that a hair piece or wig might be obtained or, if the patient is female, that a scarf or a hat may be won.
8. A referral may be made to a visiting nursing agency requesting home visits to provide guidance and assistance. A history of the patient's illness and the prescribed plan of therapy is given to the agency so that there can be continuity of care. Home visits also provide opportunities to assess the family's reactions to the patient at home and to see how they are handling the situation. The family is advised of the assistance available from a local cancer society.

LYMPHOMAS

What is lymphoma?

Introduction

You have heard of different cancers and lymphoma is one cancer you may have heard about. It is also common in Zambia. But let us discuss it in detail. Lymphomas differ from leukaemias in the degree of maturation of the affected cells and location of cancer cell production. They are cancer of committed lymphocytes rather than stem cell precursors as in leukaemias. Certain types of lymphomas may occur due to the spread of abnormal white blood cells in other parts of the body. Lymphomas are solid tumours rather than cellular suspension within the blood and bone marrow

Definition- They are neoplasms of the lymphoid tissue that is usually malignant but in rare cases may be benign.

Lymphomas can also be defined as a group of cancers in which cells of the lymphatic system become abnormal and start to grow uncontrollably

Classification

They are currently classified on the basis of histological appearance (Presence or absence of a specific cancer cell type called the “Reed-Sternberg Cell”)

They are basically classified into two:

- Hodgikin’s lymphoma (Reed-Sternberg Cell present)
- Non Hodgikin’s lymphoma (Reed-Sternberg Cell not seen)

HODGKIN LYMPHOMA

It is a malignant disorder characterized by painless, progressive enlargement of lymphoid tissue.

The presence of the reed-Sternberg cell is the pathologic hallmark of this disorder. But four histological subtypes of Hodgkin's disease have been recognized:

- Lymphocyte predominant
- Nodular sclerosis
- Mixed Cellularity
- Lymphocyte depression

Prognosis

In terms of prognosis the lymphocyte predominant and nodular sclerosis have the best prognosis and lymphocyte depletion the worst. Nodular sclerosis is the most common type accounting for about 40-70% of cases.

Ann Arbor Clinical Staging

- Stage 1: Involvement of single lymph node region for a single extra lymphatic organ or site

Stage Ii: Involvement of 2 or more regions on the same side of the diaphragm. Or localized involvement of the extra lymphatic organ or site and one of more lymph node regions on the same side of the diaphragm.

STAGE III: Involvement of lymph node regions on both sides of the diaphragm which may also be accompanied by involvement of the spleen or by localized involvement of extra lymphatic organ or site or both

STAGE IV: Diffuse or disseminated involvement of one or more extra lymphatic organs or tissue with or without associated lymph node involvement

NON HODGKIN'S LYMPHOMAS

The presence or absence of fever, night sweats, or un explained loss of 10% or more of body weight in 6 months preceding admission are denoted by the suffix letters B and A, respectively .Biopsy-documented involvement of stages IV sites also is denoted by letter suffixes: M, marrow, L, lung; H, liver; P, pleura; O, bone; D, skin and subcutaneous tissue.

Signs And Symptoms

Non-Hodgkin lymphoma may involve any lymphatic or extra lymphatic tissue. Consequently, any symptom may present.

- painless enlargement of one or more superficial lymph nodes.
- chest pain due to enlargement of the lymph node of the chest,
- cough, due to enlargement of the lymph node of the chest
- shortness of breath, or due to enlargement of the lymph node of the chest
- swelling of the face and arms due to enlargement of the lymph node of the chest
- Enlarged lymph nodes in the abdomen or pelvis or an enlarged spleen may cause discomfort, fullness, loss of appetite, back pain, or swelling of the legs.
- lymphoma of the stomach may lead to abdominal discomfort, nausea, vomiting, or internal bleeding;
- lymphoma of the brain (primary central nervous system lymphoma) may cause headaches, alteration in mental status, seizures, or focal weakness;
- Lymphoma of the skin may result in localized or extensive skin lesions.

Symptoms may occur due to involvement of less common sites, such as bone, testis, spinal cord, eye, or sinus. Patients may have symptoms of anemia (fatigue, shortness of breath).

About 20 percent of patients have "B" symptoms (fever, night sweats or weight loss).

How can we diagnose Lymphoma?

DIAGNOSIS

- Full blood count: it will show high WBC cont (lymphocyte), Low platelets count and low HB
- Bone marrow biopsy to r/o leukaemia
- Lymph node biopsy can help to comfirm the diagnosis
- LP may show CNS involvement
- Chest X ray to rule out pulmonary metastesis and infection
- History of cancer in the family

Treatment

Cytotoxic drugs such as:-

Vincristine 2mg IV od

Cyclophosphamide 800-1000mg/kg IV od

Steroids such as:-

Prednisolone – begin with high dose then reduce slowly.eg 60mg od then reduce to 15mg then, 10mg then, 5mg until you stop.

Other forms of treatment include:

Radiotherapy.

Palliative treatment.

NOTE: Palliative care should begin immediately the diagnosis is made and note in the terminal stages of the illness.

Antibiotics to prevent infection e.g. Penicillin.

Haemopoietics – to correct anaemia e.g. Folic acid 5-10mg od for 14/7.

Blood transfusion – when there is severe anaemia.

Nursing care

1.4.As for leukaemia
1.5.Heart Disorders

Cardiac diseases are classified into two categories. These are:

1. Congenital Heart Diseases
2. Acquired Heart diseases

Acquired heart diseases are further sub classified into:

- Inflammatory diseases which may result in structural changes within the heart
- Deficiency in blood supply to the myocardium
- Disturbances in conduction
- Decompensation, or heart failure

Let us begin by discussing inflammatory conditions of the heart.

Inflammatory diseases which may result in structural changes within the heart

Rheumatic Heart Disease

Definition of Rheumatic Heart Disease: RDH refers to acute or chronic damage to the heart that is related to one or more previous attacks of rheumatic fever.

Definition of Rheumatic Fever: Rheumatic fever is an autoimmune collagen disease of childhood characterized by lesions in the heart, blood vessels, joint and other connective tissues.

Aetiology

The disorder is a result of a hypersensitive reaction to group A beta haemolytic streptococcal infection of the upper respiratory system. The exact etiopathogenesis is not clear. The preceding streptococcal infection may not always show clinical signs.

Pathophysiology

The body forms antibodies against group A beta-haemolytic streptococci. Instead of the antibodies attacking the antigen, it attacks the collagen tissues i.e. joints and the heart valves, especially the mitral valves, and the muscle coat of small arteries.

Lesions are found in the wall of the left ventricle, the inter-ventricular septum, the mitral and aortic valves, left atrium and pericardium and in blood vessels of many organs.

The disease is a complication of group A streptococcal infection usually of the upper respiratory system. A period of 1 to 5 weeks may lapse between the infection and the onset of the rheumatic fever, during which time the patient may have recovered completely from the infection.

The inflammatory response, which may occur in the joints as well as in the heart, is thought to be due to a sensitivity of the affected individual to the antibodies that were formed in response to the invading bacteria. The antistreptococcal lysine titer is found to be high in these persons at the onset of rheumatic fever. This sensitivity is only present in certain individuals, since not all those with streptococcal infection develop rheumatic fever. The symptoms of the acute stage vary in intensity and may be so mild that they go unrecognized. In some, joint involvement and fever may be predominant with no evident symptoms referable to the heart, until cardiac tissue becomes involved and permanent damage occurs.

Rheumatic fever may cause acute myocarditis with subsequent scarred areas that reduce myocardial efficiency and impairment of the conduction system.

The valves are the most common area of the heart to be affected, and the mitral and aortic valves are the most susceptible. They frequently become scarred, distorted and functionally impaired. Both the valve ring at the opening and the valve cusps may fuse. These changes result in resistance to the forward movement of the blood, thus increasing the work of the heart chamber behind the obstruction. Damage of this type is referred to as a stenosis. Normally, mitral opening in an adult is large enough to admit three fingers; in severe stenosis it can become so restricted that only one finger may be introduced.

In some instances, the scarring of the valvular cusps produces a thickening and loss of tissue that prevents them from coming together to close off the opening completely. This incomplete closure allows regurgitation or backflow of blood through the valve, and is called valvular insufficiency. An added strain is placed on the heart chamber, behind the insufficiency. Many patients with rheumatic heart disease have combined stenosis and insufficiency in the affected valve. If the mitral valve is involved, the left atrium develops dilatation and hypertrophy to compensate for the resistance of stenosis and the backflow of insufficiency. In the case of aortic valvular damage, the left ventricle dilates and hypertrophies. Prolonged strain created by a damaged valve, increased demands on the already weakened heart, or further progress of the initial rheumatic disease process may result in decompensation or heart failure.

Predisposing Factors

1. Genetic predisposition: the disease tends to run in families
2. Age: commonest in 4 to 15 years age group; rare below 3 years
3. Socio-economic status: common among children of overcrowded families with poor dietary background – all of which enhance exposure to streptococcal infection
4. Climate: temperate, subtropical and tropical areas – all have it in abundance

5. Season: high incidence in winter months
6. Previous attack: an attack of upper respiratory infection carries 0.5 to 3% risk of rheumatic fever. Previous history of rheumatic fever carries 50% risk of recurrence.

Clinical Manifestations/Diagnostic Criteria (revised/modified Jones criteria)

To make a diagnosis of rheumatic fever 2 major and 2 minor criteria are need together with evidence of preceding streptococcal infection.

Major Manifestations Or Criteria

1. **Carditis**
   - Significant apical systolic murmurs, apical mid-diastolic murmur or basal diastolic murmur
   - Increasing cardiac enlargement
   - Pericarditis
   - CCF in absence of the other causes
2. **Polyarthritis:** flitting (migratory) accompanied by pain and limitation of active movements or by tenderness, redness or swelling of two or more joints. Arthralgia alone without objective evidence of joint involvement is not a major manifestation.
3. **Chorea:** involuntary movements of which are moderate or severe.
4. **Subcutaneous nodules:** short –like hard bodies, seen or felt over extensor surface of certain joints (particularly elbows, knees and wrists), in the occipital region, or over the spinous processes of the thoracic and lumbar vertebrae
5. **Erythema marginatum:** This is a recurrent, pink characteristic rash of rheumatic fever, in which the colour gradually fades away from its sharp scalloped edge. It is mainly found over the trunk, sometimes extremities, but never on the face. It is transient, is brought about by heat and migrates from place to place

Minor Manifestations

1. **Fever:** A significant rise in temperature. It occurs mainly at night
2. **Arthralgia:** joint pain. Arthralgia must not be used as a minor criterion when polyarthritis is used as a major criterion
3. **Previous history of rheumatic fever or presence of inactive rheumatic heart disease:** the existence of either of these may be used as a minor criterion to aid in deciding the rheumatic nature of the illness in question. For this use, previous history must be documented by the same objective criteria.
4. **Prolonged P-R interval in ECG:** it is considered a minor criterion and is not diagnostic of carditis. It cannot be used if carditis is already included as a major manifestation.
5. **Increased ESR, leukocytosis or presence of C-reactive proteins:** elevation in one or more of these nonspecific tests may be considered as a single minor criterion.

Supportive Evidence

Evidence of preceding beta-haemolytic streptococcal infection which must be documented:

- Streptococcal infection (sore throat, positive throat culture, scarlet fever) preceding onset of rheumatic fever by one week to one month
- An elevated or rising antistreptolysin-O (ASO) titer

Other Investigations

- WBC will be increased
- RBC parameter will show anaemia
- Chest x-ray may show enlarged heart
- Echocardiography will show damaged or thickened valves

Other Manifestations include:

- Weight loss
- Easy fatigability
- Tachycardia out of proportion to fever
- Malaise
- Sweating
- Pallor
- Epistaxis
- Erythema nodosum
- Pericardial pain
- Headache
- Vomiting
- Positive family history of rheumatic fever

Treatment of Acute Episode

1. Prophylactic penicillin is prescribed in acute episodes of rheumatic fever and for several years thereafter lifelong antibiotic prophylaxis may be necessary for persons with significant rheumatic heart disease e.g.

a. Benzathine Penicillin 600,000 U in children below 6 years; 1,200,000 U in older children, 2.4 U in adults, i.m. single dose or
b. Procain Penicillin 50,000 U/kg/day in one dose i.m. for 10 days.
c. In allergy to penicillin give Erthromycin 40mg/kg/day in two doses orally for 10 days

2. Aspirin 80-100mg/kg/day (600-900mg daily) in 2-3 doses orally for 1-2 weeks in mild to moderate carditis.

3. Prednisolone 2mg/kg/day (40-60mg orally daily) in one morning dose orally. Slow reduction when child is improving and ESR is normalized.

Nursing Management Of Patient With Rheumatic Fever/Heart Disease

1. Routine nursing care
2. Specific care include:
   - Bed rest with minimal activity according to level of cardiac activity
   - Support and protection for painful joints e.g. ripple mattress, bed cradle
   - Recording sleeping pulse as an indication of cardiac involvement (raised sleeping pulse signifies sinus tachycardia persisting during sleep)
   - Diet – maintenance of nutrition with emphasis on adequate intake of protein and vitamin C
   - Recognition of side-effects of drugs, e.g. vomiting, tinnitus
   - Educational programme according to the child's/adult's physical ability
   - Gradual increase in activity and mobility with careful observation of the pulse rate and rhythm
   - Long convalescence either at home or hospice
3. If patient develops congestive heart failure, manage as for heart failure

Congenital Heart Disease

Definition: these are abnormalities or malformations of the structure of the heart or the large proximal blood vessels that were present at birth due to various causes.

Aetiology

1. Maternal factors e.g.

a. Rubella during the 1st trimester of pregnancy
b. Tetratogenic viral infections e.g. herpes simplex

2. Drugs consumed during pregnancy e.g. thalidomide
3. Idiopathic hypercalcemia
4. Hereditary
5. Environmental factors e.g. high altitudes
6. Genetic factors e.g. gargoylism, marfan syndrome. All genetic disorders are known to be accompanied by congenital heart lesions
7. Chromosomal defects e.g. down syndrome, trisomy 13-15, Turner syndrome etc are usually accompanied by congenital heart disease.

Classification of CHD: there three (3) main classifications namely:

1. <u>Cyanotic group (Right to left shunt)</u>

a. Tetralogy of Fallot
    i. Dextroposition or 'overriding' of the aorta
    ii. Ventricular septal defect
    iii. Pulmonary artery stenosis
    iv. Hypertrophy of the right ventricle
b. Transposition of the great vessels
c. Tricuspid atresia
d. Ebstein's anomaly (deformity and displacement of the tricuspid valve into the right ventricle).

2. **Acyanotic group (left to right shunt)**
    a. Ventricular septal defect
    b. Atrial Septal Defect
    c. Patent ductus arteriosus
3. **Obstructive (resistance to blood flow)**
    a. Coarctation of aorta
    b. Valvular pulmonary stenosis
    c. Congenital mitral stenosis
    d. Congenital mitral incompetence
    e. Congenital aortic stenosis
    f. Dextrocardia
    g. Vascular rings

Pulmonary Heart Diseases

Pulmonary Heart Disease (Cor-Pulmonale)

Cor pulmonale is a latin word which stands for Cor- heart and Pulmonale – lung meaning pulmonary heart disease. This is enlargement of the right ventricle of the heart in response to increased resistance or high blood pressure in the lungs (pulmonary hypertension). Pulmonary hypertension is an increase in blood pressure in the pulmonary artery, pulmonary vein and capillaries together known as the lung vasculature.

Definitions

1. Cor pulmonale is a term given to a condition in which there is a change in structure and function of the right ventricle as a result of respiratory disorder.
2. Cor pulmonale is defined as right ventricular hypertrophy and dilatation secondary to pulmonary hypertension (Rubin and Farber, 1999).

Types of cor-pulmonale

Cor pulmonale can either be acute or chronic.

Acute cor pulmonale refers to the sudden occurrence of pulmonary hypertension, most commonly as a result of massive pulmonary embolization. This condition causes acute right sided heart failure and is a medical emergency. It usually results in dilatations because of increased right ventricular pressure.

Causes

- Parenchymal diseases of the lung such as pneumonia, chronic bronchitis or emphysema
- Congenital diseases
- Pulmonary Embolism

Signs and symptoms

- Unexplained dyspnea
- Pruritic chest pains
- There may be hemoptysis when there is infarction

The patient may present with three critical signs and symptoms and these are:

1. **Small/ medium pulmonary embolism**
   - Pruritic chest pains
   - Breathlessness
2. **Massive pulmonary embolism**
   - There is sudden collapse due to acute obstruction of the right outflow tract.
   - Severe central chest pain which may be due to cardiac ischemia.
   - The patient may have pallor
   - Sweating or syncope (fainting due to reduced blood flow to the brain)
3. **Multiple Recurrent Embolism**

   The patient will present with:
   - Breathlessness
   - Fainting episodes on exertion
   - Weakness

Management

The patient will be nursed in the emergency room with all resuscitative equipment such as oxygen and working suction machine, emergency tray with drugs like hydrocortisone, digoxin atropine etc.

Level of consciousness

Assess the level of consciousness by calling out his name, if no response pinch the earlobe.

a) **Airway**
   - Check for the patency of the airway, if there is any blockage or secretions or any tongue collapse.
   - Check for loose teeth or foreign objects to clear the air way
   - Tilt the head to the side And suction to clear the airway

b) **Breathing**
   - Observe the rate and the depth of the breathing or respiration to rule out any respiratory distress
   - Prop up the patient to promote lung expansion
   - Initiate gas exchange by administering oxygen

c) **Circulation**
   - Palpate pulse for quality and rate
   - Assess the colour, temperature and moist of the skin to monitor any deviation from normal
   - Observe the vital signs such as temperature blood pressure to rule out shock if low. And also pulse if rapid and thread.

Administer fluids as ordered

d) Drugs

Give drugs as per doctor's order to help resuscitate the patient and initiate cardiac output.

Take a quick history from the patient of the family member for the name, age address all this is for identity.

Chronic cor pulmonale is a common heart disease, accounting for 30% to 40% of all cases of heart failure. It is defined as right- heart hypertrophy or right ventricular dilatation secondary to disorders of the respiratory system. Right ventricular hypertrophy is the predominant change in chronic cor pulmonale. The hypertrophy is an adaptive response to long term increase in pressure.

Causes

- Chronic obstructive pulmonary diseases
- Chronic bronchitis and emphysema
- Cystic fibrosis
- Pneumoconiosis
- Tuberculosis

Pulmonary fibrosis

Pulmonary vascular disease

- Primary pulmonary hypertension
- Recurrent pulmonary emboli
- Peripheral pulmonary stenosis
- Intravenous drug abuse

Schistosomiasis

Chest wall abnormalities including thoracic deformities

Pathophysiology

Cor pulmonale may be caused by any pulmonary disease that interferes with ventilator mechanics or gas exchange. For instance, pulmonary hypertension increases the heart's work load. To compensate, the right ventricle hypertrophies to force blood through the lungs. However, the compensatory mechanism begins to fail and larger amounts of blood remain in the right ventricle at the end of diastole. This causes ventricular dilatation. In response to hypoxia, the bone marrow produces more red blood cells, resulting in polycythemia (abnormal increase in the number of red blood cells in the blood). Then, the blood's viscosity increases, further aggravating pulmonary hypertension, increasing the right ventricle's work load and causing heart failure.

Signs and symptoms

1. Patient is cyanosed as heart failure compounds already impaired pulmonary function
2. Chronic productive cough
3. Dyspnoea
4. Oedema
5. Wheezing respiration
6. Fatigue
7. Weakness
8. Ascites

Investigations

1. History and physical examination
2. CXR- will reveal large central pulmonary arteries and right ventricular enlargement.
3. ECG- right ventricular R-wave will show dilation and tricuspid regurgitation
4. Arterial blood gas analysis will reveal increased levels of carbon dioxide
5. Hematocrit
6. Serum hepatic enzyme
7. Serum bilirubin level
8. Pulmonary artery catheterization to measure pulmonary pressure

Treatment

- Treat the cause
- Bed rest
- O_2 therapy to correct the hypoxaemia
- Antibiotics to treat underlying respiratory causes
- Digitalis such as Digoxin may be given to strengthen the heart muscles
- Diuretics such as Lasix 40- 80mg for right ventricular failure which will provide relief on the strained heart

Hydralazine **Nursing Care**

- ***Environment*** **–** nurse the patient in the acute bay near the sister's office for ease observation. The room should have all resuscitative equipment at hand.
- ***Position*** **–** patient should be nursed semi fowler's position with pillows and backrest to allow for lung expansion. Patient should be changing positions frequently to prevent pressure sores. The air-ring should be used to prevent pressure sores. Elevate the legs of the patient to promote venous return thus relieving edema.
- ***Observations*** **–** observe the vital signs 4hoursly to identify any deviation from normal. Observe the oedema if subsiding or not. Observe for cyanosis on the nail bed to rule out hypoxia. Weigh the patient daily to monitor the oedema. Observe the urine output to ascertain the renal functioning. Observe the early signs of infection such as raised temperature and report to the doctor.
- ***Psychological care-***explain the condition to the patient to allay anxiety. Allow patient to verbalize the fears and concerns about the effect of activity restriction to gain cooperation. Explain all the procedures done on the patient.
- ***Nutrition*** **–** offer a low salt diet to reduce fluid accumulation. Give small frequent meals to avoid tiring the patient. Restrict fluids intake to prevent overload. Give diet contacting roughage to prevent constipation as it can strain the heart. Perform oral care to stimulate appetite. And serve food in an attractive manner.
- ***Elimination*** **–** provide bedside commode as this method puts less stress on the heart than a bed pan. Monitor intake and output and record on the fluid balance chart.
- **Rest-** Advise complete bed rest in acute phase to conserve energy therefore cluster the nursing care to provide periods of rest. Avoid strenuous activities to promote rest. Nurse patient in a quiet room to promote rest. Restrict visitors and minimize noise.
- **Medication –** give medications as ordered and observe the side effects.

IEC- warn the patient to avoid using non prescribed drugs such as sedatives that can depress the heart. Explain the importance of rest. Emphasize the importance of low salt intake.

Infective Endocarditis

Definition: This is a severe infection of the endocardia surface of the heart characterized by high fever, heart murmurs and sometimes embolism. The endocardium, the inner layer of the heart is continuous with the valves of the heart; therefore inflammation from infective endocarditis usually affects the cardiac valves.

Classification

1. Acute infective endocarditis
2. Sub-acute infective endocarditis

Causative organisms

- Streptococci especially viridans
- Staphylococcus especially aureus and enterococcus
- Gram negative bacteria such as Escherichia Coli, Klebsialla and pseudomonas
- Haemophilus
    - Actinobacilus
- Cardiobacterium

Predisposing Factors

- Cardiac conditions especially valvular heart disease
- Endoscopy in patients with valvular heart disease
- Post-surgical procedures such as dental surgery
- Intravenous drug abusers
- Artificial heart valves
- Aortic valve leaflet abnormalities
- Nosocomial bacteria

Pathophysiology

Infective endocarditis occurs when blood flow turbulence within the heart allows the causative organism to infect previously damaged valves or other endothelial surfaces. Fibrin and platelets cluster on valve tissue and engulf circulating bacteria or fungi. This produces vegetation which in turn may cover the valve surfaces causing destruction of valvular tissue and sometimes causing perforation of the valves. As infection subsides, scarring occurs and if a valve was involved, incompetence occurs. Vegetation growth on the heart valves, endocardial lining of the heart chamber or the endothelium of the blood vessel may embolize to the spleen, kidneys central nervous system and the lung. Embolism disrupts the organ function. Some signs and symptoms depend on the organ affected For example, if it is the lungs there will be severe dyspnoea, pleuritic pain, haemoptysis. If the emboli lodges or obstructs the artery of the leg, gangrene may develop to the part affected. For kidneys, failure occurs and in the brain there will be cerebral vascular accident (CVA) or stroke

Symptoms

- Fatigue
- Low grade fever
- Weakness
- Chills
- Night sweats
- anorexia
- Weight loss
- Muscle aches and pains
- Heart murmur
- Shortness of breath with activity
- Swelling of feet, legs, abdomen
- Blood in urine
- Excessive sweating
- Petechial lesions in the mucous membranes of the mouth, pharynx, or conjuctiva
- Red, painless skin spots on the palms and soles
- Pallor
- Nail abnormalities (splinter haemorrhages under the nails)
- joint pains
- Red, painful nodes (Osler's nodes) in the pads of the fingers and toes

Complications

- Congestive heart failure if treatment is delayed
- Embolism causing severe damage to organs like the brain, kidneys, lungs, or abdomen.
- Arrhythmias such as atrial fibrillation
- Glomerulonephritis
- Valvular incompetence due to colonies in situ preventing proper closure of the valves and leaking of blood around and between colonies
- Renal insufficiency
- Septicaemia
- Brain abscess

Management Of A Patient With Infective Endocarditis

Investigations and Diagnosis

1. Patient's recent history regarding recent dental urologic, surgical or gynaecologic including normal or abnormal obstetric delivery, heart disease diagnostic procedures, infection of skin and respiration or urinary system.

2. Repeated blood culture and sensitivity
3. ESR will be raised
4. Full blood count may show low grade, microcytic anaemia
5. WBC count with differential rheuma old factor
6. Urinalysis will reveal blood in urine
7. Chest x-ray will reveal cardiomegally
8. CT scan of the chest
9. Echocardiography will reveal vegetative growths on the heart valves
10. ECG

Therapy: the main aim of therapy is to eliminate all microorganisms from the vegetative growth and prevent complications.

Therapy include:

1. Appropriate antibiotic
2. Antipyretics
3. Rest
4. Repetitions of blood cultures and sensitivity tests
5. Surgical valvular repair or replacement

Hospitalization may be required initially to administer intravenous antibiotics. Long-term, high dose antibiotic treatment is required to eradicate the bacteria from the vegetations on the valves. Treatment is usually administered for 4-6 weeks, depending on the organism. The chosen antibiotic must be specific for the organism causing the condition. This is determined by the blood culture and the sensitivities tests.

If heart failure develops as a result of damaged heart valves, surgery to replace the affected heart valve may be needed.

Prophylaxis

Before any invasive procedure is done on/to a patient who is predisposed to infective endocarditis, the following prophylaxis of infective endocarditis should be given:

1. For dental, oral, respiratory tract or oesophageal procedure, if streptococcus is a pathogen, the antibiotic of choice will be:
   - Amoxicillin 2g 1hour before procedure, alternative if unable to take oral or if allergic to penicillin.
   - Ampicillin IV
   - Clindamycin, oral or IV
   - First-generation Cephalospeins – Cefazin. IV azithromycin or Clatithromycin
2. For genito urinary and non esophageal GI procedures, if pathogens is faecalis, antibiotic choice will be parenteral Ampicillin or Amoxycillin, IV/IM Gentamycin, IV Nafacillin, IV Vancomycin (for staphylococcus)

Advise to the Patient and Relatives

- Teach the patient to avoid excessive fatigue and to stop activity immediately if chest pain, dyspnoea, light headedness, or faintness occurs.
- To avoid persons with infections
- To avoid overcrowding and overcrowded areas
- Patient should inform all primary care providers including physicians, and dentists about history of infective endocarditis, so that appropriate antibiotic therapy can be administered prior to invasive procedure.
- Patient should brush with soft-bristled tooth brush and floss regularly to protect the gums and or event cavities. Good dental hygiene is important in decreasing the risk of recurrent infective endocarditis.
- Patient should seek help when he experiences symptoms like weight loss without change in diet, blood in urine, chest pain, weakness, numbness or weakness of muscles and fever.

Pericarditis

The pericardium is the fibroserous sac that envelops supports and protects the heart.

Definition: Pericarditis is the condition caused by inflammation of the pericardial sac (pericardium) which may occur on an acute or chronic basis (*Lewis etal, 2004*).

Pericarditis can be divided into acute and chronic.

Acute pericarditis

Acute pericarditis is an inflammation of the pericardium that begins suddenly and is often painful. The inflammation causes fluid and blood products such as fibrin, red blood cells and white blood cells to pour into the pericardial space. It is more common than the chronic pericarditis. It can occur as a complication of infections, immunologic conditions even as a result of myorcadial infarction. It is characterized by:

- Fever
- Chest pain which typically extends to the left shoulder and sometimes down the left arm. The pain is worsened by lying down coughing or even deep breathing.

Chronic pericarditis

Also called constrictive pericarditis is inflammation that results in fluid accumulation or thickening of the pericardium and that begins gradually and is long- lasting. It is characterized by dense fibrous pericardial thickening. The fibrous tissue tends to contract over years, compressing the heart and making it smaller. Compression increases the pressure in the veins that return blood to the heart because higher pressure is needed to fill the heart. Fluid backs up and then leaks out and accumulates under the skin, in the abdomen, and sometimes in spaces around the lungs. It is associated with cancer, tuberculosis or low thyroid function.

Symptoms include:

- Dyspnea
- Coughing,
- Fatigue
- There is no pain

Causes

Idiopathic

Infections

- *Bacterial*- Pneumococci, staphylococci, streptococci and neisseria gonorrhoeae, septicemia from gram negative organisms.
- *Viral*- coxsackievirus, mumps, Epstein- barr virus, hepatitis B varicella zoster virus
- *Fungal* –Histoplasma, candida species

Non infectious

- Acute myorcadial infarction
- Neoplasms –lung cancer, breast cancer, leukaemia, lymphoma
- Cardiac injury- thoracic surgery and cardiac diagnostic procedure
- Dissecting aortic aneurysm
- Myxoedema

Hypersensitivity or autoimmune

- Rhematic fever
- Drug reaction

- Rheumatologic diseases; rheumatoid arthritis
- Post Myocardio- infarction

Diagnosis

1. History and physical examination
2. Culture of pericardial fluid will isolate the causative organism.
3. FBC will show normal or elevated white blood cell count and raised ESR
1. ECG shows characteristic changes in acute pericarditis
2. CXR will reveal pericardial effusion
3. Echo Cardiography diagnoses pericardial effusion

Treatment

- Viral or idiopathic treat with non-steroidal anti- inflammatory drugs.
- Severe cases may require antibiotics, corticosteroids for inflammation.
- Aspirin, Indomethacin for pain

Complications

1. Pericardial effusion

Cardiac tamponade (an emergency in which fluid accumulates in the pericardium affecting the heart ventricles from filling properly due to raised fluid pressure)

Myocarditis

Definition: This is a focal or diffuse inflammation of the myocardium which may be due to infection or toxins.

Causes

- Possible causes include :
- Viruses such as coxsackievirus, influenza, rubella, poliomyelitis and HIV
- Bacteria-diphtheria, tuberculosis, typhoid fever, staphylococcal, pneumococcal etc.
- Fungi,

- Parasites- toxoplasmosis, trypanosomiasis
- Hypersensitivity immune reaction such as acute rheumatic fever
- Radiation therapy especially large doses to the chest during the treatment of lung or breast cancer.
- Pharmacological and chemical factors carbon chronic alcoholism

Pathophysiology

Damage to the myorcadium occurs when an infectious organism triggers an autoimmune, cellular or humoral reaction. Non infectious causes lead to toxin inflammation. In either case, the resulting inflammation may lead to hypertrophy, fibrosis and inflammatory changes of the myorcadium and conduction system. The heart muscle weakens, and contractility is reduced. The heart muscle becomes flabby compromising the heart's pumping ability.

Signs and Symptoms

- It's difficult to diagnose clinically unless the cardiovascular function is impaired then it presents with fever, fatigue, malaise, myalgias, dyspnoea, pharyngitis and palpitations and nausea and vomiting. Chest pain, rapid or abnormal heart beat, shortness of breath

Investigation

1. Endomyocardial biopsy confirms a myorcarditis diagnosis
2. FBC reveal raised WBC count and ESR
3. Throat and stool cultures will isolate the causative organism

Treatment

Myocarditis may be mild, moderate or severe. Recovery from a mild episode happens without complications.

- Treat the underlying cause
- Bed rest, to reduce the work load of the heart.
- Diuretics to decrease fluid retention
- Digitalis to increase myocardial contractility
- Sedation
- Administer O_2 therapy when necessary.

Nursing Care

- ***Rest***: Activity restriction to minimize myocardial consumption of the heart
- ***Observation***: Observe for signs of digitalis toxicity which are anorexia, nausea, vomiting and blurred vision. Observe for signs of left ventricular failure which are dyspnoea, hypotension and tachycardia.
- ***Diet*****:** Restrict salt intake
- ***Psychological care*****:** Allow the patient to express his concerns about the effects of activity restrictions on his responsibility and routine. Reassure the patient that the restrictions are temporary. Explain the disease process and every procedure done on the patient to gain cooperation. Also provide diversional therapies that are undemanding.
- ***Elimination:*** Provide a bedside commode as this method puts less stress on the heart than a bed pan.

Complications

- Left ventricular failure.
- Chronic myocarditis

Congestive Cardiac Failure

INTRODUCTION

The heart is said to be failing when the cardiac output is unable to maintain the circulation of sufficient blood to meet the needs of the body.

In mild cases, cardiac output is adequate at rest and becomes inadequate only when increased cardiac output is needed such as during strenuous exercises. Heart failure may affect either side of the heart. However, when one side of the heart begins to fail it leads to increased strain on and eventually failure of the other side. Left ventricular failure is more common than right because of the greater workload of the left ventricle.

DEFINITION

Heart Failure is a clinical syndrome that results from structural or functional cardiac disorders in which the heart is unable to pump enough blood to meet the metabolic needs of the body at rest and during exercises (Ignatavicius & Workman, 2006)

Incidence

Prevalence of heart failure is higher in patients with history of rheumatic fever, hypertension and coronary heart disease. It is also prevalent in those leading a sedentary life style (have reduced physical activity and a high serum cholesterol).

Aetiology/Predisposing Factors

- Valvular heart disorders like infective endocarditis and syphilitic heart disease (stenosis & regurgitation).
- Long-standing systemic hypertension that is of long standing
- Hypervolemia e. g IVF overload
- Congenital heart diseases e. g ventricular septal defects
- Anemia
- Thyroid disorders e. g Thyrotoxicosis
- Pregnancy
- Myocardial Infarction
- Myocarditis
- Heredity plays a predisposing role - LDL.
- Ventricular aneurysms
- Constrictive pericarditis
- Chronic Obstructive Pulmonary Diseases (COPD) such as asthma, TB, Bronchitis
- Physical or emotional stress (strenous exrecises, excitement, fear) increase sympathetic nervous stimulation leading to increased HR, contractility & raised BP

Pathophysiology

The healthy heart can meet the demands of life by increasing output in response to stress, up to 5 times the resting level. However, the failing heart has limited ability to respond to body's needs for increased output in situations of stress. The heart in failure has 3 main compensatory mechanisms:

- 1. Ventricular dilation
- 2. Ventricular hypertrophy
- 3. Increased sympathetic nervous system stimulation

Ventricular dilation

This refers to the lengthening of the muscle fibres which increases the volume of the heart chambers. However, if muscle fibres are stretched beyond a certain point, they become ineffective and eventually fail.

Secondly, a dilated heart requires more oxygen and because the coronary blood flow can not supply more oxygen, hypoxia results, further leading to reduced cardiac muscle contractility

Ventricular hypertrophy:

This is the increase in the diameter of muscle fibres in an effort to increase contractile power.However, ventricular hypertrophy requires a corresponding increase in oxygen supply. Lack of increase in oxygen supply results in hypoxia and reduced contractile power

Increased sympathetic nervous system stimulat This leads to venous and arteriolar constriction resulting in increased peripheral resistance. Increased peripheral resistance leads to reduced blood supply to the kidneys.

Decreased blood supply to the renal activates the renin-angiotensin mechanism

Angiotensin – angiotensin mechanism leads to salt and water retention resulting in increased fluid volume and puts further strain on the already failing heart.

Clinical Features

The right and left sides of the heart are separate. Therefore, it is possible for one to fail, independently of the other. However since the ventricles are anatomically related, circulatory abnormalities from one affected side will eventually affect the other.

Left Sided Failure (Forward Failure)

Congestion occurs mainly in the lungs from backing up of blood in the left side of the heart and pulmonary veins.

- Shortness of breath: dysponea occurs on exertion at first as the failure progresses, patient becomes breathless even at rest,
- Orthopnoea
- Paroxysmal Nocturnal Dyspnoea
- Cough – may be dry, unproductive, often occurs at night due pulmonary congestion.
- Easy Fatigability from low cardiac output, - low O_2 to tissues and from dyspnoea
- Insomnia
- Tachycardia
- Restlessness

–

Right Sided Failure (Backward Failure)

- Signs and symptoms of elevated pressures and congestion in the venous system:
  - Pitting oedema after retention of excessive fluid.
  - Liver congestion – may produce upper abdominal pain
  - Distended neck veins
  - Ascites due to abnormal fluid accumulation in the peritoneum cavity
  - Pericardial effusion
  - Anorexia and nausea from hepatic and visceral engorgement

Management

Medical Management

Investigations

- History taking from the clients
    - Physical examination (Clinical manifestation)
    - Chest X- ray (frequently used)
    - Arterial blood gases levels
    - Electro cardiogram

Treatment

Treatment of CCF focuses on treating the symptoms and preventing the progression of the disease. If there is reversible cause of the heart failure such as (infection, alcohol ingestion, anaemia thyrotoxcosis or hypertension treat the cause reversible cause treatments include exercise eating healthy foods, reduction in salty food and many more.

Non Pharmacological Measures

Patients with CCF are educated on various measures which are non- pharmacological to improve symptoms and prognosis. These include:

- Moderate physical activities or bed rest when symptoms are severe.
- Weight reduction through physical activity and dietary modification.
- Sodium restriction (recommended 60-100 mmol total daily intakes.
- Fluid restriction – Generally water intake should be restricted to 1:5 liters or less per day.

Pharmacological Management

Drugs used include:

- Diuretics
    - Vaso dilators
    - Beta blockers
    - Diuretics

loop diuretics e.g. furosemide mostly used for moderate CCF

Thiazide diuretics e.g. hydrochlorothiazide

potassium sparing diuretics e.g. spinorolactone amilonde used to as first line to correct hypocalcaemia

Vaso dilators

Hydralazine in combination with isosorbide dinitratae

Surgery

Cardiac re synchronization therapy (CRT) which involves pacing both the left and right ventricles correctly through implantation of a biventricular pace makes of surgical modering of the heart.

Use of left ventricular assisted devices (LVADs which are battery operated mechanical pump type devices that are surgically implanted on the upper part of the abdomen. They take blood from the left ventricle and pump it through the aorta. These are commonly used for patients awaiting for heart transplants.

Heart transplantation.

Valvular repair

Complications

- Respiratory failure
    - Renal failure
    - Co- pulmonale
    - Death
    - PROBLEM INDENTIFIED
    - Dyspnea
    - Anxiety
    - Generalized Oedema

- Anorexia
- Chest and abdominal pains
- Coughing
- Fatigue
- Risk of falling
- High risk for infection
- Risk for impaired skin integrity
- Activity intolerance

Figure

	PROBLEM	**NURSING DIAGNOSIS**	**OBJECTIVES**	**NURSING INTERVENTIONS**	**EXPECTED OUTCOME**
1	Dyspnea	Difficulties in breathing related to impaired gas exchange as evidenced by rapid breaths of 30 respirations per minutes	Client will experience a reduction in dyspnea 16 to 20 respirations per minutes	- I will elevate the patient's head to promote adequate lung expansion. - I will give oxygen by mask. -I will ensure a clear airway by position patients head to one side for if unconscious drainage of secretions. - I will monitor patients respiration rate depth and quality.	- dyspnoea relieved as evidence by stable breathing.

	PROBLEM	NURSING DIAGNOSIS	OBJECTIVES	NURSING INTERVENTIONS	EXPECTED OUTCOME
2	Anxiety	Anxiety related to the condition and lack of knowledge about the prognosis as evidenced by asking too many questions and restlessness	The patient will be relieved of anxiety within six hours	-I will explain the condition to the client and the prognosis. -I will explain to the client about treatment availability. I will provide a calm environment to promote rest by reducing noise levels or putting a client in a corner or side ward away from traffic - I will explain all the procedures and equipment being used on him. - I will institute measures to relieve dyspnea	Patient relieved of anxiety evidenced by resting and sleeping
3	Oedema	fluid volume excess related to decreased cardiac output as evidenced by oedema and weight gain	Client will demonstrate adequate fluid balance manifested by reduction in oedeme.	- I will be checking clients body weight daily to asses what? - The reduction in fluid volume. - I will restrict salt intake diet to prevent increased sodium retention of 60-100 mmol / day. - I will restrict fluid intake to prevent further increase in fluid volume. - I will give prescribed divertics e.g. lasix to promote elimination of excess fluids	Patient has normal fluid volume evidenced by a reduction in oedema andweight.

	PROBLEM	NURSING DIAGNOSIS	OBJECTIVES	NURSING INTERVENTIONS	EXPECTED OUTCOME
4	Risk for impaired skin integrity	Risk of impaired such integrity related to decreased tissue perfusion,inactivity and oedema.	- Client will maintain normal skin integrity.	- I will help the client to change position frequently to reduce pressure on prominent areas for a long time. - I will assist client in bathing a lubricating the skin. -I will provide protective devices such as an airing or pillow. - I will asses the integrity of the skin for signs of pressure sore formation. - I will allow / help patient to move in and out of bed and provide a chair for sitting out of bed.	-Client has intact skin
5	Risk for Altered Nutrition	Risk for Altered nutrition less than body requirements related to anorexia secondary to portal congestion and abdominal fullness	Client will maintain good nutritional status	- I will give frequent meals according to patient's preference. - I will assist or encourage my client to perform mouth washes to promote appetite. - I will assist the client in feeding since he is weak. - I will remove things in the environment which will disturb the clients appetite example bed pans. - I give my client drugs for appetite example muilt-vitamin as prescribed.	Client's nutritional status maintained evidenced by healthy skin. Client able to eat with no problems.

Cardiac diseases due To deficiency In The blood supply to the myocardium

There are three (3) diseases due to deficiency in the blood supply to the myocardium. These are:

1. Coronary Artery (Heart) Disease
2. Myocardial Infarction

CORONARY ARTERY (HEART) DISEASE

Definition: this is an ischaemic heart disease that results from narrowing or obstruction in the coronary arteries which subsequently reduces the blood supply to the myocardium.

Pathology

The reduced blood supply to the myocardium in most instances is due to degenerative changes in the arteries that produce a narrowing of the lumen of the vessels. Fatty substances, which include cholesterol, are deposited within the intima of the arteries to cause atherosclerosis. Theses fatty plaques interfere with the nutrition of the cells in the intima, leading to necrosis, scarring and calcification, which leave the surface rough and the lumen reduced. These roughened constricted areas allow less blood through and predispose to thrombus formation and occlusion of the vessel.

While the blood supply through the artery is being reduced, a collateral circulation develops in an effort to increase the supply to the myocardium, but this supplementary circulation is rarely sufficient to provide enough oxygen to the heart muscle during strenuous physical exertion.

Incidence

The incidence increases with age and shows a strong familial tendency. It is less common in women of childbearing age than men of the same age but after this period gradually becomes as frequent in women. The incidence is higher among populations whose diet regularly contains high amounts of calories, total fats, saturated fats, cholesterol and refined sugars.

Aetiology

The major cause of coronary heart disease is atherosclerosis

Risk factors

1. Age and Sex: coronary heart disease is seen frequently in elderly persons. Incidence tend to be high in men than in women, however, after menopause, it tends to increase in women as well
2. Hereditary: the exact mechanism is not known but genetic predisposition has been implicated.
3. Diabetes: the incidence is high in diabetics. This may be due to elevated levels of circulating insulin which helps to form atheroma and damage arterial intima. Insulin also modifies lipid metabolisms.
4. Hypertension: hypertension affects the ability of blood vessels to constrict or dilate. Decreased elasticity of blood vessels, tearing effect on arteries, and increased resistance of ejection of ventricular volume may lead to coronary heart disease.

5. Smoking and tobacco use: nicotine found in tobacco has the following physiological effects and may cause coronary heart disease:
    - Decreased high density lipoproteins (HDL)
    - Displacement of oxygen from haemoglobin
    - Increased catecholamine in response to nicotine, increasing heart rate and blood pressure
    - Increased platelet adhesiveness
    - Accelerates atheroma formation
    - Coronary spasm
6. Sedentary lifestyle: this alters lipid metabolism and decrease in HDL. Physical inactivity may lead to CAD
7. Diet: dietary intake of more cholesterol and fat provides more substance for lesion formation. Hypercholesterdaemia, familial hyperlipidaemia, increased levels of low density lipoproteins, and increasing atherogenesis.
8. Obesity: obese persons are more prone to diabetes, hypertension, and hyperlipidaemias. These people often lead sedentary lifestyles.
9. Stress and behaviour pattern: catecholamine released during stress response increases platelet aggregation and may also precipitate vasospasm.
10. Excessive alcohol consumption

Clinical Manifestations

There are three major clinical manifestations in coronary artery disease. These are:

- Angina Pectoris
- Acute myocardial infarction
- Hidden cardiac death

MYOCARDIAL INFARCTION (IM)

Definition: this is an irreversible necrosis of part of the heart muscle, almost always due to coronary atherosclerosis.

A coronary artery becomes blocked and the myocardial area which it supplies suffers oxygen deficiency and necrosis. It occurs suddenly, and compensation through collateral channels is inadequate to maintain the myocardial cells. The resulting area of necrotic tissue is referred to as an infarction. The extent of the infarction varies from patches of 1 or 2 centimeters in diameter to widespread areas of necrosis. One or more layers may be involved. The area of infarction becomes soft and then eventually fills in with firm, fibrous scar tissue. Survival and the extent of subsequent restrictions depend upon the amount of myocardial damage and the area of the heart affected. Death may occur immediately or within a few hours. The remaining viable heart tissue must compensate for the loss of functional tissue. Occlusion may be preceded by some

manifestations of coronary insufficiency such as angina, or it may occur suddenly without any previous warning.

Figure FigureFigureTable 2: Similarities and Differences between Angina Pectoris and Myocardial Infarction

	ANGINA PECTORIS	MYOCARDIAL INFARCTION
Precipitating Factors	• Stress, either physiologic or psychologic • Digestion of heavy meal • Extreme temperatures • Sexual excitation • Cigarette smoking • Stimulants	• Exertion or rest • Physical or emotional stress • Often no precipitating factors are associated with the pain
Location	• Mid-anterior chest • Substernal • Abdominal with radiation to neck, back of arms, fingers • Diffuse, not easily located	• Mid-anterior chest • Substernal • Diffuse • Radiation to neck and jaw or down left arm or both arms to fingers
Description	• Deep sensation of tightness or squeezing feeling • Mild to moderate severity or pressure • Similar attack each time • Twinges or dullness in thoracic arc	• Severe pressure, squeezing or heaviness with a crushing oppressive quality • Report of such severe pain that the patient would rather die than experience pain again • Residual 'soreness' for several days following MI
Onset and duration	• Gradual or sudden onset • Usual duration of 15 minutes or less (usually less than 30 minutes) • Relief by nitroglycerine	• Sudden onset • Duration of 30 minutes to 2 hours • No relief from rest or nitroglycerine
Associated clinical manifestations	• Apprehension • Dyspnoea • Diaphoresis • Nausea • Desire to void • Belching	• Apprehension • Nausea and vomiting • Dyspnoea • Diaphoresis • Extreme fatigue • Dizziness or faintness (after abatement of pain)

Medical Management

1. Aims of management
2. To restore ability of the heart to maintain adequate circulation

Angina pectoris

- Start with 0.5 mg glyceryl trinitrate or spray 0.5mg/intra oral puff PRN up to every half hour

Then maximum doses of 'triple therapy' e.g.

- Beta blockers: atenolol 50-100mg/24hrs
- Calcium antagonists: nifedipine 10-20mg/8hrs orally, if using slow release form give 20mg/12hrs
- Isosorbide dinitrate 5-40mg/6hrs orally (slow release-20-60mg/24hrs) or adhensive nitrate skin patches 5-10mg/24 hrs, remove for 4-8hrs/24hrs or buccal absorption (e.g. 1mg, 2mg or 3mg)

The following measures should be taken during an attack:

- Patient to stop any activity engaged in, and rest
- Determine intensity of patient's pain; detected by non-verbal indicators like clutching, rubbing and stroking of the chest. The pain is not relieved by change of position or respiration.
- Administer oxygen
- Determine the vital signs
- Take a 12 lead ECG
- Prompt pain relief 1st with a nitrate followed by narcotic analgesic if needed
- Physical assessment of the chest
- Comfortable positioning of the patient.

Myocardial Infarction

Patient is nursed in the intensive care unit until condition stabilizes. Reassure patient and find time to talk to relatives.

- Unless contraindicated, administer 35% oxygen
- Carnulate for emergencies
- Diamorphin 5-10mg iv at 1mg/min (+ anti-emetic cyclizine 50mg iv) for anslgesic, anxiolytic, anti-arrhythmic, and venodialtor effects
- Glyceryl trinitrate 0.5mg for coronary artery vasodilatation
- If presentation is < 12hrs after onset of pain, give streptokinase 1.5 million units in 100ml of 0.9% saline iv over 1hr + aspirin 160mg/day orally for > 1 month

- Heparin 5000U/8hrs subcut, until mobilized, as prophylaxis against deep vein thrombosis
- If pain continues, give isosorbide dinitrate infusion 2-10mg/hr iv. If maximum tolerated dose is inadequate, give diamorphine 5-10mg iv PRN. If pain is still not controlled, refer for angiography with a view to urgent surgery.
- PRN prochlorperazine 12.5mg im up to 6 hourly; laxative
- Discontinue pre-infarction cardiac drugs
- Prohibit smoking
- Continuous ECG monitoring
- 24hr bed rest
- 4 hrly TPR, BP
- Daily 12 lead ECG, CXR, cardiac enzymes, and Urea and Electrolyte for 2-3 days
- Examine heart, lungs and legs for complications at least daily

Nursing Management Of Patient With Acute Myocardial Infarction

Patient is kept at rest in a position that is most comfortable and facilitates breathing – usually semi recumbent position. Tight clothing (collar, belts, etc) is loosened and the patient covered sufficiently to prevent chilling. Food and fluids are withheld.

The patient transported to hospital and admitted in ICU for about 3 to 5 days. When condition stabilizes, he is transferred to the general ward.

Assessment: constant observations of the patient are necessary in the acute stage. The nurse must be alert to significant changes and must make decisions about the need for prompt reporting, the seeking of assistance and the use of emergency measures.

TPR - Check patient's pulse, blood pressure and respirations every half to 1 hrly for the 1st 4 hours or until patient's condition is stable, then 2 to 4 hours during the acute stage and more frequently if there are great variations in the readings. When the vital sign observations become stable, the frequency of observations is changed to 4 times a day. Any changes in the rate or rhythm of the pulse are reported. Take note of the pulse in response to activity.

Blood pressure is also checked after administration of drugs such as morphine or meperidine (Demerol), and if the patient is experiencing signs or symptoms such as pain, faintness or cyanosis. Blood pressure is checked when the patient is lying down and after assuming the upright position to detect orthostatic hypotension. Either a fall in blood pressure or continuous hypertension is reported immediately.

Dyspnoea may be due to pulmonary congestion or pain. It may also occur as activity is increased. Note whether shortness of breath is present at all times or if intermittent, how it is precipitated and what measures relieve it. During convalescent stage, respirations are observed 4 times daily and in relationship to various activities.

Hourly ECG monitoring is important. Vital signs can be monitored using cardiac monitor, so patient should not be woken up when sleeping.

Blood for enzyme levels estimation; electrolyte and urea levels should be taken for 3 consecutive days. Test urine for sugar.

Skin – the skin may be cool, moist and a grayish colour in response to the decreased cardiac output. The colour, temperature and moistness of the skin are continually observed and any changes noted.

Temperature – myocardial necrosis causes an elevation of body temperature ranging from 37.7ºC to 39ºC. The temperature usually rises within 24 to 48 hours and returns to normal by the sixth or seventh day. If the temperature persists longer, it may be due to complications. The patient's temperature is taken 4 times daily until it has been normal for several days. It is then taken once or twice daily.

Nausea and vomiting – this may be experienced at the time of the attack, lasting from several hours to 2 or 3 days. Sometimes, nausea and vomiting occur in response to the opiates being administered. The nurse notes the presence of nausea and vomiting and any possible precipitating factors.

Weakness and Tiredness – weakness may persist for weeks after the attack. Observe the extent to which the patient tolerates any activity (such as walking). Note how much patient sleeps during the day and how quickly he fall asleep following activities.

Psychological reactions – patient may show various psychological responses such as anxiety (manifested by tenseness, restlessness, short attention span, inability to concentrate, crying, constant talking and verbalization of feeling of anxiety, or darting eye movements); denial, anger and/or depression. These signs should be taken note of.

Relief of pain: analgesics such as morphine sulfate or meperidine (Demerol) is usually given to relieve the severe pain associated with myocardial infarction. The effect of the drug on respiratory centre should be monitored.

Relief of hypoxia and Dyspnoea: oxygen is usually administered by mask or nasal catheter in order to increase arterial oxygen tension, which may help to relieve the myocardial pain caused by hypoxia and prevent extension of the infarction. Mechanical respiratory assistance may be needed in cases of severe insufficiency. Take note of the effectiveness of oxygen therapy indicated by reduced pulse rate, less dyspnea, improvement in colour and less restlessness.

Also tell the patient to avoid exertion by showing patient how to move easily in bed, and by assuming a low semi-Fowler's position. This allows for greater expansion of the lungs and reduces the venous return to the heart, thus reducing the stress on it.

Relief of Nausea and Vomiting: withhold oral intake for a period of time and resume gradually in small amounts. The mouth should be rinsed after each vomit and basin emptied promptly. Soiled bedding and clothing should be changed and the room ventilated. Ensure rest, quiet and minimum disturbance. Change of position should be done slowly.

Relief of anxiety: ensure mental rest by relief of physical symptoms. Give brief and precise explanation of all equipment, routines, tests and procedures. Ensure prompt attention to patient's needs and monitor patient constantly. Nurse should be calm, quiet and competent without apparent concern and hurry. Give honest and supportive answers to patient's questions. A close family member may be left by the bed side.

Rest and restriction of activity: patient is placed on complete bed rest until symptoms such as pain and shortness of breath have reduced. The nurse therefore, should provide all care for the patient. Everything should be placed within patient's reach. Activity is introduced gradually e.g. patient is allowed mild activity such as cleaning his teeth, washing his face and hands and feeding himself. Exercising of lower limbs to prevent venous stasis may be commenced on the second day. Ensure undisturbed periods of rest after activities such as bathing and eating.

By the third day, activity may be increased, e.g. the patient may be allowed to dangle his legs over the side of the bed for 5 to 10 minutes. He is advised to sit up gradually to prevent feelings of dizziness or faintness. If pain free by the 3rd day, allow patient to sit in a chair for brief periods (10 to 15 minutes) and increase duration each day.

Continuous cardiac monitoring is essential to immediately identify any arrhythmias accompanying changes in activity. Record blood pressure in the lying and sitting positions and the patient is observed closely for reactions such as weakness, fatigue, shortness of breath and chest pain. It is important to increase activity gradually and to alternate rest periods with activity. Activities involving pushing, pulling, lifting or straining should be avoided.

By the 4th to 5th day, patient may be ready to leave the intensive care unit and may be sitting in a chair three times a day for 30 to 45 minutes, using a bedside commode, feeding and bathing himself and taking short walks around his bed. Progression of activities continues after the patient is transferred from the intensive care unit and in weeks after he leaves the hospital. He will begin walking short distances on the ward, progressing in length to outside the ward by time of discharge. Activities that precipitate dyspnoea and pain should be limited.

Even after pain stops, restrictions on activities should continue, so provide diversions which the patient is interested in e.g. reading and listening to the radio and television programs but these should be selected to avoid overexcitement and fatigue. Visitors should be restricted at first but later they help relieve the patient's boredom. Visitor selection should be made by the family to avoid those who might excite or distress the patient. Visitors should be made aware of time limitations as it may be difficult for the patient to ask them to leave if he becomes tired.

Nutrition: Liquids are given in small amounts at first following a myocardial infarction. The diet is increased gradually to easily digested, non-gas forming as tolerated. Large meals are

avoided, if possible decrease caloric intake. Salt intake should be restricted. Fluids may be restricted to 1 litre – 1.5 litres per day if the patient is in heart failure. Intravenous tubing to be changed every 24 hrs. Fluid intake and output should be charted strictly to ensure patient remains in a slightly negative balance in order to reduce work load of the heart.

Elimination: constipation should be prevented and the patient cautioned against straining at stool because of the stress it places on the heart. A mild laxative or stool softener may be given if necessary.

Patient Teaching

In order to make the best possible adjustment to his illness and recovery, the patient must have some understanding of the illness and how he can assist in recovery. Providing this information helps give the patient some control over his situation. Teaching programs are adjusted to specific patient needs and capabilities, since learning capacity varies among individuals.

Health teaching for patients with coronary artery disease includes use and storage of nitroglycerin in case of angina and guidelines for sexual activity after myocardial infarction.

1. Use and storage of nitroglycerin
    - Sit or lie down at onset of angina/chest pain
    - Place tablet under the tongue and allow tablet to dissolve, don't chew.
    - If pain not relieved within 5 minutes, take a second tablet. A third tablet can be used after an additional 5 minutes if pain persists. Continuing pain after 3 tablets and 15 minutes indicate need to receive immediate medical attention.
    - Keep a record of number of anginal attacks experienced, the number of tablets needed to obtain pain relief and precipitating factors if known
    - Carry tablets for immediate use if necessary. Do not pack in luggage when traveling.
    - Protect tablet from exposure to light, moisture and store in cool dry place. Check expiry date on prescription. Tablet should be discarded after 6 months once the bottle has been opened.

    Plan for replacement of supply.
2. *Risk factor modification*
    - Provide specific instruction on smoking cessation, daily exercise and diet modification.
    - Encourage adherence to a diet low in calories saturated fats and cholesterol

Discuss the benefits of stress management techniques in decreasing negative effect on oxygen demand.

3. Resumption f activities
    - Provide specific instructions on activities that are permissible and those that should be avoided.
    - Discuss resumption of driving and return to work
    - Discuss guidelines for resuming sexual relations (2 weeks for low risk patients to 4 weeks for post myocardial infarction patients).

Hypertension

What is hypertension? I am sure you have heard a lot of people saying they have high blood pressure. Take a minute and think about what it means to have high blood pressure.

Hypertension is defined as a systolic blood pressure of 140 mm Hg or more, or a diastolic blood pressure of 90 mm Hg or more (Roger et al., 2012).

The diagnosis of hypertension requires that elevated readings be present on at least three occasions during several weeks.

In hypertension, the heart works harder than usual and puts the heart and blood vessels under strain. This may lead to myocardial infarction, stroke, renal failure and atherosclerosis.

Classification

Hypertension is classified into two; Primary and Secondary.

Primary Hypertension

Primary (also known as essential) hypertension is elevated BP without an identified cause. Majority (about 95%) of hypertensive patients falls into this classification (Lewis et al., 2003).

Contributing factors to primary hypertension include:

- Increased sympathetic nervours system activity
- Overproduction of sodium retaining hormones and vasoconstrictors
- Increased sodium intake
- Greater than ideal body weight
- Diabetes mellitus
- Excessive alcohol intake

Secondary Hypertension

This is elevated blood pressure with a specific cause that can be identified and corrected.

Causes of secondary hypertension includes:

- Coarctation or congenital narrowing of the aorta
- Renal disease such as renal artery stenosis and parenchymal disease
- Endocrine disorders such as pheochromocytoma, Cushing's syndrome
- Neurologic disorders such as brain tumours, head injury
- Sleep apnoea
- Medications such as NSAIDS, oestrogen containing pills
- Pregnancy induced

Pathophysiology of Hypertension

Increased blood volume, cardiac rate and stroke volume or arteriolar vasoconstriction that increase peripheral resistance causes blood pressure to rise. Hypertension may also result from the breakdown or inappropriate response of the Rennin- angiotensin system. The renal hypo perfusion causes the releases of rennin.

Angiotensinogen, a liver enzyme, converts the rennin to angiotensin I which increases preload and after load.

Angiotensin I then convert to angiotensin II in the lungs. A powerful vasoconstrictor, angiotensin II also helps increase preload and after load by stimulating the adrenal cortex to secrete aldosterone. This serves to increase sodium reabsorption and in turn increases water reabsorption, plasma volume, cardiac output and blood pressure.

Clinical features

- Occipital headache due to raised blood pressure and increased peripheral vascular resistance
- Visual disturbance due to reduced blood supply to the retina
- Heart palpitations due to increased cardiac output
- Dizziness due to reduced blood supply to the brain
- Fatigue due to reduced tissue perfusion
- Flushing due to increased peripheral resistance

- Epistaxis as a result of rupture of small blood vessels in the nose due to raised blood pressure.
- Mild oedema due to renal insufficiency leading to water retention
- Shortness of breath due to increased oxygen demand

Management of Hypertension

Investigations

- History
- Blood pressure monitoring for 3 days to confirm the diagnosis
- Electrocardiogram (ECG) shows the electro activity of the heart reveals. Aids in the diagnosis of heart disease
- Echocardiography reveals the action of the heart
- Urinalysis to rule out diabetes mellitus
- Fasting blood sugar to rule out diabetes mellitus
- Chest x-ray to rule out cardiomegally
- Serum potassium and sodium levels to rule out renal and liver function

Drug therapy

- **Vasodilators**
- Hydralazine

Action – a direct acting vaso dilator, its effect relates to arterial smooth muscles

Dose- 10mg and maximum of 200mg

Route – per oral, IV, IM

Side effects – peripheral neuritis, headache, dizziness, palpitation

Nursing implications – monitor blood pressure, pulse and body weight, if IV give slowly with normal saline or ringer lactate

- **Calcium channel blockers**
- Nifedipine

Action – They interfere with inward displacement of calcium ions through the slow channels of active cell membrane. Prevents calcium ions from entering vascular smooth muscle cells thereby causing dilation of arterioles which in turn decrease peripheral vascular resistance.

Dose – 10- 20mg once daily

Route per oral, sublingual

Side effects - Headache, dizziness, hypotension, chest pains and heart palpitations.

Nursing intervention- Monitor blood pressure regularly especially during initial administration. Advise patient to take the drug with low fat meal or with grape fruit products

- **Adrenergic blockers**
- Atenolol

Action - Blocks beta adrenergic receptors, reduces cardiac output, peripheral resistance.

Dose- 50mg as a single dose

Route – per oral

Side effects- drowsiness, hypotension, lethargy, fatigue and bradycardia

Nursing intervention- check pulse before administering the drug, withhold if less than 60 beats/ minute. Monitor blood pressure

- **Angiotensin converting enzyme inhibitor**
- Captopril

Action –It inhibits ACE, preventing invention of angiotensin I to II a potent vaso constrictor, reduced formation of angiotensin II reduces peripheral arterial resistance thus reducing aldosterone secretion. Thus reducing aldosterone secretion

Dose -6.25 – 12.5mg 8hourly

Route per oral

Side effects- dizziness, fainting, headache, malaise, fatigue abdominal pains and dry mouth.

Nursing implication- use cautiously in patients with impaired with renal function and the elderly

- **Diuretics**
- Frusemide

Action – Loop diuretics inhibit reabsorption from the ascending limb of the loop of henle in the renal tubule a potent loop diuretic.

Dose - 20 – 80 per oral, IV, IM

Side effects –Volume depletion and dehydration, hypokalaemia, hypotension, polyuria, deafness

Nursing intervention- check blood pressure, give Slow - K 600mg once daily to replace the lost potassium. Advise the patient to beating foods rich in potassium such as bananas.

Nursing care

Environment

The patient must be nursed in a well ventilated and quiet environment to promote rest.

Nurse the patient near the sister's office for ease observation

Ensure all necessary equipment such as oxygen cylinder are at hand

Position – Semi fowlers position to aid breathing**.**

Psychological care

Explain the disease process to raise the patient's level of knowledge. It is also important that you explain all procedures done on the patient to allay anxiety. If the patient becomes very anxious, the blood pressure may rise further.

Allow the patient to express their worries and concerns pertaining to the condition to allay anxiety. Also explain the importance of life style modification such as sodium restriction as it helps in reducing complications.

Observations

As a nurse it is important that you monitor the blood pressure every 30 minutes and put the blood pressure profile and change depending on the patient's improvement. Observe pulse to monitor the cardiac functioning, respirations to assess the extent of dyspnoea and intervene appropriately, temperature to rule out other infections.

Monitor and record the intake and output to rule out kidney failure

Diet

Restrict sodium intake as it increases blood pressure by increasing fluid retention. Reduce foods containing fat and cholesterol, this increases blood pressure by reducing the lumen as a result of fat accumulation.

Restrict caffeine and caffeine containing products, they constrict blood vessels thereby increasing the blood pressure. Give enough roughage and adequate fluids to prevent constipation which increases the cardiac workload.

Elimination

Monitor the output of the patient to rule out any abnormalities in the renal and gastrointestinal tract functioning. Monitor the patient for constipation as this will cause him to strain when defecating hence increasing cardiac workload thus increasing blood pressure. Record the urine output in the intake and output chart

Exercises

The patient requires passive exercises in the acute phase. As the condition improves, encourage the patient to be doing regular and moderate exercises to facilitate cardiac functioning, as this help reduce weight in obese patients thereby reducing high blood pressure.

Information Education and Ccommunication

1. Diet – Explain the dietary changes and the importance of such changes for example low calorie intake to reduce the cardiac work load and reduced sodium intake to prevent fluid retention.
2. Life style modification – Encourage the patient to adjust their life style for instance, they should stop smoking if they do as smoking worsens the condition. There is need for mild to moderate exercises to strengthen the heart muscles and burn the fat. And also to reduce alcohol intake.

3. Medication – Emphasize the importance of drug compliance to prevent resistance and to keep record of drugs used in the past such as the ones that have been of help. Advise the patient to avoid buying drugs over the counter as some drugs may cause vasoconstriction.
4. Stress management – Advise the patient to avoid extreme emotional/ stress for example excessive anger, excitement, these factors may raise blood pressure.
5. Review dates- Patient should observe review dates
6. Teach patient signs and symptoms of raised blood pressure and to report to the hospital as uncontrolled hypertension may cause stroke and heart attacks.

Complications of hypertension

- Congestive cardiac failure
- Cerebral vascular accident
- Renal failure
- Retinopathy

Peripheral Vascular Disorders

Introduction

Peripheral vascular disease (PVD), commonly referred to as peripheral arterial disease (PAD) or peripheral artery occlusive disease (PAOD), refers to the obstruction of large arteries not within the coronary, aortic arch vasculature, or brain. PVD can result from several factors such as atherosclerosis, inflammatory processes leading to stenosis, an embolism, or thrombus formation. It causes either acute or chronic ischaemia (lack of blood supply). It is important to note that PVD is a term used to describe atherosclerotic blockages found in the lower extremities and also includes micro vascular diseases resulting from episodic narrowing of the arteries (Raynaud's phenomenon).

Definitions

Peripheral vascular diseases are conditions of the blood vessels that leads to narrowing and hardening of the arteries that supply the legs and feet (Lewis, 2007) or

Any disease or disorder of the circulatory system outside of the brain and heart which may be arterial (occlusive or functional), venous, or lymphatic.

Atherosclerosis is the deposition greasy (lipids) called plaque on the inside of arteries and partially/completely restrict the flow of blood.

Arteriosclerosis is the thickening and hardening of arterial wall.

Thrombosis this is the formation or presence of a thrombus; clotting within a blood vessel which may cause infarction of tissues supplied by the vessel.

Deep vein thrombosis is the blockage of blood flow by a clot which may be partial or complete.

It is often the result of long periods of immobility.

Arteriosclerosis

It is the thickening, hardening and loss of elasticity of the walls of arteries. This results in altered function of tissues and organ.

Cause

The cause is idiopathic. However, there are predisposing factors and these include:

- Degenerative changes/ serenity
- Altered lipid metabolism leading to a condition called atherosclerosis (deposition of fat in the blood vessels. Atherosclerosis is a type of arteriosclerosis.
- Heredity
- Hypertension
- Obesity
- Cigarette smoking

Pathology

Arteriosclerosis will lead to narrowing of arterial vessels. This narrowing causes malnutrition of tissue cells which in turn causes ischemia and then necrosis. Healing by fibrosis causes further hardening. Sclerosis causes degeneration of major organs due to lack of blood supply. Major organs affected include; brain, kidney, heart, liver.

Clinical features

- Pain in the affected part
- Absence of pulse in the area distal to the occlusion.
- Cyanosis of the limb distal to the occlusion, coldness of part
- Tissue atrophy
- Necrosis or gangrene finally develops

Management

Aim:

Increase blood flow to the affected part, exercises are very paramount.

Diagnosis

- History
- Clinical features
- Investigations ie
- Venography (phlebography) there is visualisation of the vascular tree after injection of contrast media.
- Doppler's U/S detects blood flowin the veins and arteries.
- Angiography: a radio-opaque die is introduced in the arteries to visualise the flow of blood in the arteries and X-ray films can also be taken. The site of occlusion can be located.

Atherosclerosis

This problem occurs when fatty material (plaque) builds up on the walls of your arteries. This causes the arteries to become narrower. Other risk factors are;

- Smoking- promotes changes in blood vessel lining (endothelium) which is a precursor to atherosclerosis.
- Diabetes mellitus - causes between 2 and 4 times increased risk of PVD by causing endothelial and smooth muscle cell dysfunction in peripheral arteries (Mary and Donna, 2007).
-
- Hypertension- elevated blood pressure is correlated with an increase in the risk of developing PVD as it puts stress on the arterial wall.
- Risk of PVD also increases in individuals who are over the age of 50, males,
- Obese, or with a family history of vascular disease and diet high in lipids (Sellenberg, 2007).

Signs and Symptoms

- **Pain, weakness on walking, numbness, or cramping in muscles due to decreased blood flow hence there will be increased oxygen demand of the**

muscle during activity.

- **Weak or absence of pulse in the affected area because blood flow is restricted or blocked.**
- **Sores, wounds, or ulcers that heal slowly may develop.**
- **Noticeable change in color (blueness or paleness) unilateral dependent rubor or bilateral dependent rubor.**
- **Decreased temperature (coolness) when compared to the other limb distal to the blockage because of restricted blood flow.**
- **Diminished hair and nail growth on affected limb and digits.**
- **Colour change of the feet**

Diagnosis

- **Checking the strength of the pulse in the leg arteries.**
- **Listening for a whooshing sound in a leg artery or the abdomen using a stethoscope**
- **Ankle-brachial index—checking blood pressure at various points in the leg and comparing it to the normal arm blood pressure.**
- **Blood tests for blood lipids.**
- **Ultrasound and Doppler analysis of the arteries, especially the carotid arteries in the neck which supply the brain with blood.**
- **Electrocardiogram (ECG) a test that records the heart's activity by measuring electrical currents through the heart muscle.**
- **Angiography of the arteries in the legs—x-rays of blood vessels that have been injected with a dye show narrowing or thickening.**
- **Magnetic resonance imaging—a test that uses magnetic waves to make pictures of the blood vessels**

Management

Early treatment can slow or stop the advancement of the disease.

Lifestyle Changes

- **Smoking cessation**
- **Diabetes control**
- **Blood pressure control**

- **Increased physical activity for example walking program up to the limit of the patient probably 3-4 times a day**
- **Weight loss if overweight**
- **Low-saturated fat, low-cholesterol diet**
- **Attentive foot care very important for people with diabetes patient should use shoes that fit properly,**
- **Proper treatment of all foot injuries because healing is slowed when circulation is poor, and the risk of infection is higher.**

Medicine

- **Antiplatelet agents, such as aspirin 300 -600 tds and clopidogrel to thin blood**
- **Medicines to reduce leg pain for example pentoxifylline as it decreases blood viscosity and improve blood flow.**
- **Cholesterol-lowering agents like statins**

Surgery

- **To open up narrowed arteries is performed in severe cases.**
- **Atherectomy (removal of lipid deposits from the artery)**
- **Endarterectomy—the lining of the artery is removed**
- **Bypass surgery—a vein from another part of the body or a synthetic graft replaces the vessel.**
- **Balloon angioplasty - a balloon is inflated in the artery to stretch it**
- **Stent implant -a wire mesh tube is placed in the artery; the stent expands and stays in place, keeping the artery open**

Complications

- **Blood clots or emboli that block off small arteries**
- **Coronary artery disease –narrowing of coronary artery due to fat deposits leading to blockage.**
- **Impotence due to lack of blood supply to help erection.**
- **Open sores (ischemic ulcers) on the lower legs**
- **Tissue death (gangrene) due to lack of blood supply.**

Nursing management

Problems identified

- **Activity intolerance**
- **Pain**
- **Change of body image**
- **Anxiety**

Malaria

Introduction

Malaria is the most common life threatening tropical disease in the world's population causing approximately 500 million infections and more than one million deaths each year. It is one of the commonest diseases in tropical countries and is responsible for much ill health and many deaths. The word 'malaria' comes from the Italian and it means 'bad air' that is Mal=bad and aria= air. It is transmitted through the bite of an infected female mosquito belonging to the genus anopheles. In Zambia there are three species of anopheles that can transmit human malaria.

These are anopheles gambiae, anopheles arabiensis and anopheles funestus. They differ from many other mosquitoes by being late night feeders thus the rationale for sleeping under insecticide treated mosquito nets (ITNs).

Defnitions

1. Malaria is a protozoan infection of red blood cells transmitted by a bite of a blood-feeding female anopheles mosquito. (White, 2003).
2. Malaria is a protozoa infection of the genus plasmodium, transmitted to human being through the bite of an infected female anopheles mosquito characterized by paroxysms of chills, fever, sweating, splenomegaly and a chronic relapsing course." (Standard Treatment Guidelines, 2004)

Causes

There are four species of parasites that cause infection in human. Each species has a different biological pattern in which it affects man. The four species of parasites include:

- Plasmodium ovale
- Plasmodium vivax
- Plasmodium malariae
- Plasmodium falciparum.

The most common species that is clinically significant causing the most lethal of malaria is Plasmodium falciparum. It account for more than 95% of malaria cases. Plasmodium malariae accounts for 3% of malaria cases while plasmodium ovale 2%. Plasmodium vivax is very rare in Zambia.

Mode Of Transmission

- **Direct**

Malaria is transmitted through the bite of an infected female *anopheles mosquito*.

- **Indirect**

This is less frequently through blood transfusion with unscreened or improperly screened blood. It can also be transmitted through inoculation (needle prick).

- **Transplacental**

Malaria can be transmitted from the pregnant mother to the unborn baby. The unborn baby can get infected with malaria parasites from the infected mother through the placenta.

Risk Groups

- Children under 5 years
- HIV infected individuals
- Pregnant women (parasites concentrate in the placenta).

Incubation Period

The incubation period of malaria is from 10 to 14 days depending on the species causing the disease. (M oH Treatment Guideline, 2010).

Life Cycle Of Malaria Parasite

All species undergo a complex life cycle, with division and cyst formation. Part of this life cycle takes place in the blood cells and liver of a human being (asexual cycle) and part in the stomach and salivary glands of the anopheles female mosquito (sexual cycle).

A) in man

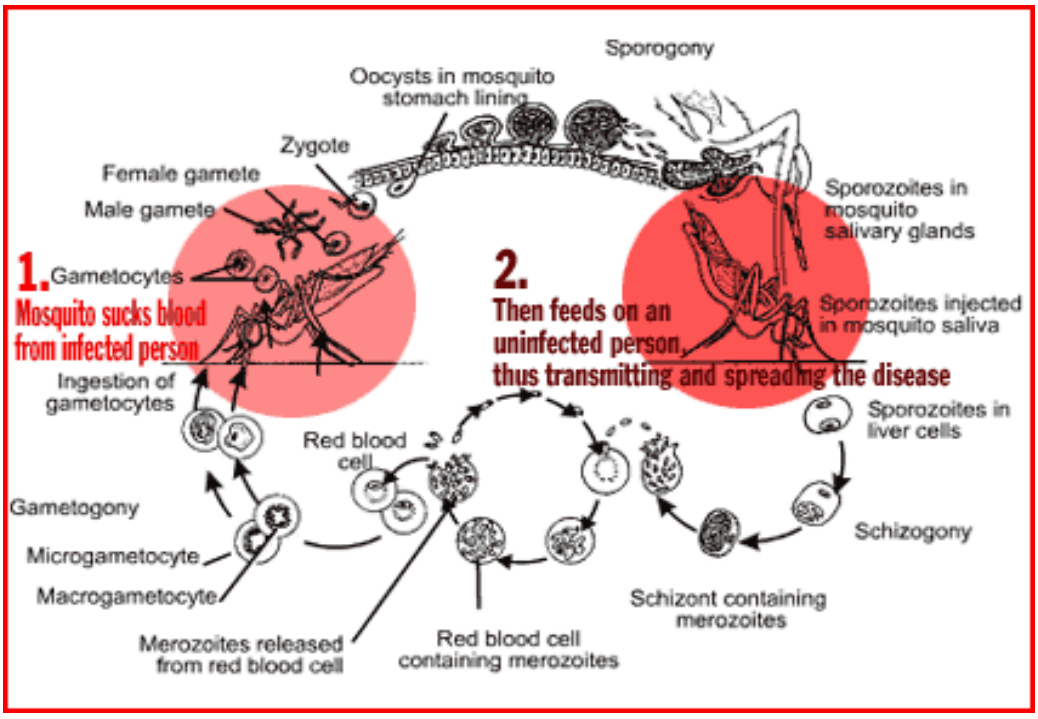
The definitive host for malaria parasites is female anopheles mosquito which acts as a transmission vector. Infection with human malaria begins when the feeding female anopheles mosquito inoculates sporozoites at the time of feeding. The sporozoites enter the blood stream and rapidly enter the liver (pre-erythrocytic or exo-erythrocytic phase in the liver) where they infiltrate the hepatic parenchymal cells. They then develop and multiply into hepatic schizonts that reproduce to generate large number of merozoites. Within 2 to 3 weeks of hepatic infection, the hepatic schizonts rupture releasing thousands of merozoites into the blood stream.

In the blood stream, they invade the red blood cells (erythrocytic stage) feed on haemoglobin, grow and multiply within the erythrocytes. Within 2 to 4 days, mature merozoites are produced and they rupture and are released in the blood stream invading the unaffected red cells, initiating another cycle of erythrocytic parasitism. This further increases the number of parasites in the human host. The release of merozoites produces the chills and fever in the patient. With Plasmodium falciparum, the parasites in the liver die after first release into blood, but with other species like plasmodium vivax and plasmodium ovale, intrahepatic parasites do not develop but remain dormant as (hypnozoites) and continue to live in the liver and cause relapses by invading

the blood stream weeks or months later. Some of the merozoites develop into gametocytes, thus the male and female sexual forms of the plasmodium ready to be picked by a mosquito.

In The Mosquito

A mosquito becomes infected when it takes a blood meal from an infected person. Once ingested the parasites gametocytes taken up in the blood will further differentiate into male and female gametes and then fuse in the mosquito's gut. This produces an ookinete that penetrates the gut lining and produces an oocyst (encyst) in the gut wall. When the oocyst ruptures it releases sporozoites that migrate through the mosquito's body to the salivary glands, where they are then ready to infect a new human host.



FigureTable**Pathophysiology**

The Pathophysiology of malaria results from red blood cell infection, destruction of red blood cells, release of parasites and erythrocyte material into the circulation and the host reaction to these events causing anaemia (Cook and Zumla, 2003)

P falciparum malaria- infected erythrocytes also sequester in the micro circulation of vital organs interfering with microcirculation flow and host tissue metabolism. In P.falciparum parasites display adhesive protein(histidine rich protein) on the surface of the infected blood cells causing the blood cells to stick to the capillary walls of the small blood vessels (*cytoadherence).*

Cytoadherance causes the cells to adhere to one another and to the walls of the capillaries in the brain, heart, spleen, intestines and lungs and this has two consequences.

Sequestration occurs, a process whereby erythrocytes containing mature forms of P.falciparum adhere to microvascular endothelium and thus disappearing from the circulation.

Parasitized erythrocytes attached to the endothelium do not circulate, so patients with severe p. falciparum have very few circulating parasites.

2) Capillaries of deep organs become obstructed, congested and eventually rupture resulting in ischemia and hypoxia of the brain, kidneys, lungs etc. There is also increased vascular permeability causing plasma and unparasitized cells to leak into the extra- vascular space causing oedema. This leads to complications in malaria such as cerebral malaria, black water fever, and severe anaemia hypoglycemia and complications in pregnancy.

Clinical Features

Disease manifestation is related to the rupture of the red blood cells, an occlusion of capillaries in major organs and organ function impairment.

- Fever which may be sudden with;

Cold stage

Patient feels intensely cold and shivers with teeth usually chattering. Temperature is rapidly elevated reaching its highest at 40 Degrees Celsius. Vomiting and headache frequently occur in this stage. It lasts 15 minutes to 1 hour.

Hot stage

The patient feels very hot and may be confused, delirious and may even be in coma. It occurs 30 minutes after the cold stage. Vomiting may continue. The patient may complain of heat in the body and simultaneously feeling cold on the outside. It lasts 2-6 hours.

Sweating stage

After about 1-6 hours, the patient sweats profusely, the temperature drops and the patient becomes relatively comfortable. The fever reoccurs after 48-72 hours depending on the periodicity characteristics of the infecting species.

Other Signs And Symptoms

- ❖ Malaise
- ❖ Drowsiness
- ❖ Abdominal pains
- ❖ Hepatosplenomegally
- ❖ Anorexia and nausea
- ❖ Fatigue
- ❖ Convulsions if the brain is involved
- ❖ Headache
- ❖ General body pains
- ❖ Arthralgia (joint pains)
- ❖ Dry cough

Investigations

- History, physical examination and clinical presentation
- A thick or thin blood film/ slide examination will demonstrate the presence of parasites in the peripheral blood.
- Rapid Diagnostic Test (RDT), this will detect the enzyme produced by the parasites.
- Blood for haemoglobin level to rule out complications like anaemia
- Blood sugar should be checked to rule out hypoglycemia
- Lumber puncture to rule out meningitis

Treatment

Antimalarials

- ➢ The first line treatment in Zambia is a combination therapy of Artemether-lumefantrine (Coartem) in uncomplicated malaria except where it is contra-indicated.
- ➢ Sulfadoxine-pyrimethamine (SP) (Fansidar) where coartem is contra-indicated and where the later is not available.
- ➢ Fansidar (SP) as first line in second and third trimester of pregnancy for Intermittent Presumptive Treatment (IPT).
- ➢ Quinine as first line in first trimester and all severe or complicated malaria.

Artemether lumefantrine

It contains Artemether 20mg + Lumefantrine 120mg.

Indications: Uncomplicated malaria

Coartem should not be given to children less than 5kg body weight.

How it is given: First dose given at 0 hours, then second dose after 8 hours then 12 hourly for two days. For example a person weighing above 35kgs would get 4 tablets stat, 4 after 8 hours then 4 twice daily for 2 two days.

- **Side effects**
- Sleep disorders, Headaches, Dizziness, Palpitations, Pruritis, rash, Abdominal pain, diarrhea, Anorexia, nausea and vomiting.

Quinine Oral—10 mg /kg body weight 8 hourly for 7 days

By I.V 20 mg/ kg body weight in 5% dextrose over 4 hours (over 8 hours in children) as loading dose, After 8 hours, 10 mg / kg body weight over 4 hours repeated 8 hourly until the patient can swallow, then oral to complete the 7days course of treatment.

- **Side effects**
- Tinnitus , Headache ,Hot and flushed skin, Rash Hypoglycaemia Confusion and Visual disturbance

IN IPT

First dose (3tablets) in second trimester or 16weeks (4months), then second and third doses, at least one month apart.3 doses are given in total.

Other treatment

- Antibiotics
- Analgesics
- Anti-anemic
- Blood transfusion in Hb of 4g/dl

Nursing Care

AIMS

- To treat malaria parasites
- To reduce temperature to normal
- To prevent complications

Care during rigors

Cold stage – Give warm fluids to stimulate internal temperature

- Close nearby windows
- Add more linen(do not over heat the patient)
- Use a hot water bottle between linen
- Monitor temperature, pulse, respiration $^{1}/_{4}$ hourly
- Observe the patient constantly

Hot stage- Open nearby windows

- Remove extra linen and clothing
- Give plenty of cold drinks
- Do tepid sponging
- Give prescribed anti-pyretic drugs orally
- Switch on the electric fan

Sweating stage- Make patient comfortable

- Keep wiping with clean cotton flannel to keep patient dry
- Change linen and continue giving cool drinks
- Switch of the fan
- Stop tepid sponging
- Prevent chilling to prevent a crisis

Environment –Nurse Patient in a dim lit room as there is a tendency of experiencing photophobia. Patient should be nursed in a quiet room as they are irritable when it involves the brain. The Room should be well ventilated to reduce temperature.

Psychological care -Create a therapeutic relationship with the patient to create confidence. Encourage the patient to express the feelings on the disease. Explain the disease process. Reassure the patient that the medical team is doing everything possible to help him.

Observation- Monitor vital signs, temperature, pulse, respiration and blood pressure at least 4hourly. Observe side effects of drugs. Observe the signs of complication like black water fever that is black urine. Also observe for any convulsions. Observe blood chemistry levels for hyponatremia and increased blood urea nitrogen creatinine and bilirubin levels. Observe signs of

hypoglycemia such as weakness and excessive sweating. Observe the level of level of consciousness.

Hygiene – Change linen frequently as the patient sweats a lot. Offer daily baths to promote comfort and oral care to prevent halitosis and promote appetite.

Exercises –Encourage coughing and deep breathing exercises especially if the patient is on bed rest to prevent pulmonary complication.

Nutrition- Encourage oral fluid intake as the patient is at risk of dehydration due to excessive sweating**.** Give balanced diet in small frequent meals. If vomiting is severe give antiemetics.

Elimination – Monitor urine output hourly and maintains it at 40 to 60 ml/hour and immediately report any decrease in output which can be suggestive of renal failure. Strict record intake and output because patients sweat a lot.

Drugs – Administer drugs as prescribed and observe the patient response to treatment as well as adverse side effects of drugs.

Prevention / Control

1) **Physical destruction of adult mosquitoes**
   - Achieved by use of long lasting insecticides sprays (DDT) to the walls of all houses at regular intervals.
   - Using short acting insecticides like target
2) **Getting rid of mosquito breeding sites near residential sites**
   - Ensure proper functioning of drains (no stagnant water).
   - Draining swamps, ponds and collection of stagnant water
   - Cutting tall grasses around the house holds
   - Covering water containers and tanks
   - Removing any container around the house that may hold water
   - Pouring oil on water surfaces to prevent larva from breathing
   - Use larvicide to kill larva
3) **Protection of the individual**
   - Wearing of long-sleeved shirts at night
   - Use of fansidar (IPT) as prophylaxis especially for pregnant women
   - Use of mosquito coils.
   - Use of mosquito repellants

- Closing of windows early in the evening (biting 22:00- 06:00)
- Making use of Insecticide Treated Nets(ITNs)
- Completing treatment

4) Health education to the public on

- Mode of transmission
- Signs and symptoms of malaria
- Seek treatment early
- Preventive measures

Complications

If untreated, the following complications may occur:

- Anaemia due to the destruction of the red blood cells containing parasites at merozoite stage and there is also accelerated rate of destruction of blood cells without parasites.
- Cerebral malaria- the brain is congested and has small hemorrhages and patches of necrosis due to blockage of the small blood vessels by parasites.
- Black water fever (haemoglobinuria) due to massive intravascular haemolysis and obstruction of the glomeruli leading to acute tubular necrosis and passage of "coca-cola' coloured urine.
- Jaundice due to failure of the liver to deal with large amounts of bilirubin presented to it by the destruction of many RBC's.
- Pulmonary oedema resulting from damaged alveolar capillaries
- Hepatosplenomegally due to invasion of the liver and spleen by parasites
- Acute Renal failure due to acute tubular necrosis resulting from reduced blood supply to the kidney as a result of renal microvascular obstruction and cellular injury following sequestration.
- Hypoglycemia- results from increased metabolic demand of febrile illnessdemands from the parasites which use glucose as their major fuel. Also impaired gluconeogenesis and limited glycogen stores.

Filariasis

Filariasis (Philariasis) is a parasitic and infectious tropical disease, that is caused by thread-like filarial nematode worms in the superfamily Filarioidea, also known as "filariaet". This infection with filarial worms results in blocking of the lymphatic causing swelling of the surrounding tissue.

Causes

There are 9 known filarial nematodes which use humans as the definitive host. These are divided into 3 groups according to the niche within the body that they occupy:

- Lymphatic Filariasis,
- Subcutaneous Filariasis,
- Serous Cavity Filariasis.

Lymphatic filariasis

Lymphatic Filariasis is caused by the worms *Wuchereria bancrofti*, *Brugia malayi*, and *Brugia timori*. These worms occupy the lymphatic system, including the lymph nodes, and in chronic cases these worms lead to the disease Elephantiasis.

Subcutaneous Filariasis

Subcutaneous Filariasis is caused by *Loa loa* (the African eye worm), *Mansonella streptocerca*, *Onchocerca volvulus*, and *Dracunculus medinensis* (the guinea worm). These worms occupy the subcutaneous layer of the skin, the fat layer.

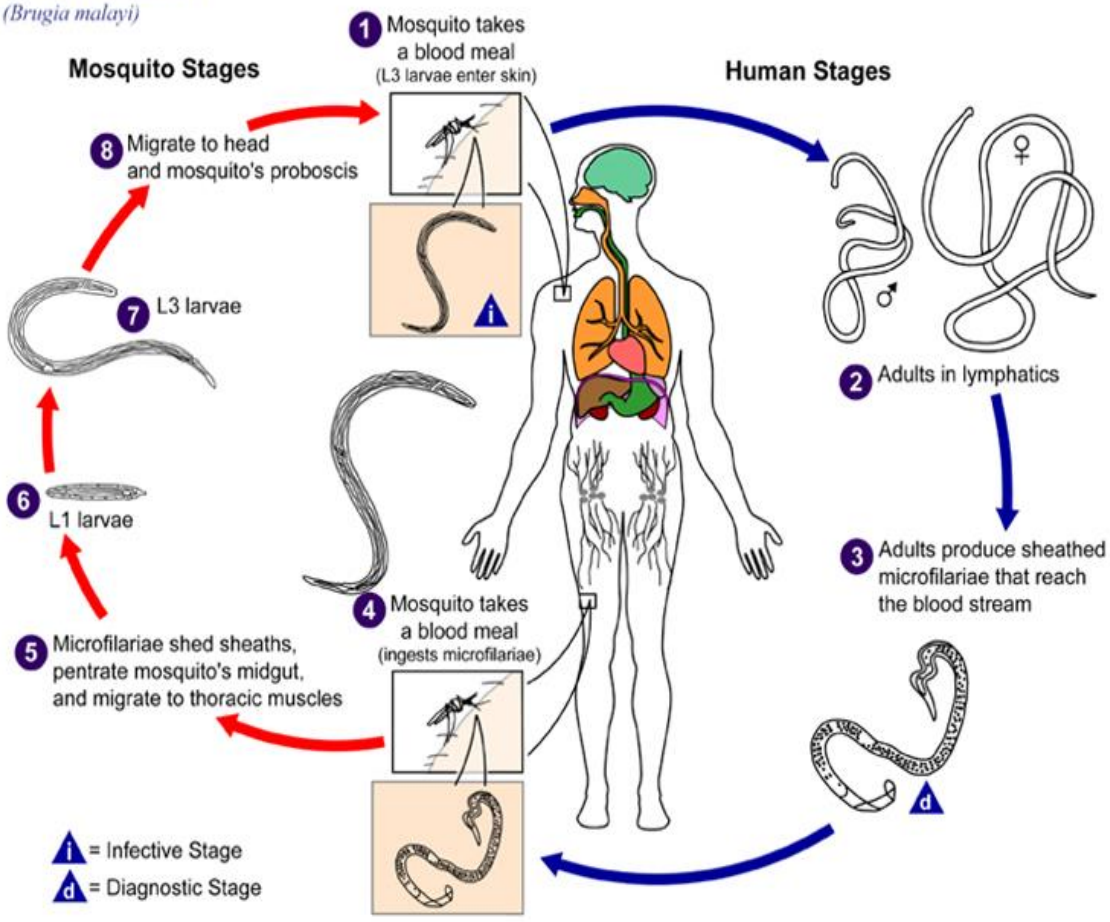
Serous Cavity

Serous Cavity Filariasis is caused by the worms *Mansonella perstans* and *Mansonella ozzardi*, which occupy the serous cavity of the abdomen. In all cases, the transmitting vectors are either blood sucking insects (fly or mosquito) or Copepod crustaceans in the case of *Dracunculus medinensis*.

Pathology

Filariasis

(Brugia malayi)



This illustration depicts the life cycle of Brugia malayi, one of the parasitic worms that cause the tropical disease lymphatic filariasis. Credit: CDC

back to Neglected Tropical Diseases

Human filarial nematode worms have a complicated life cycle, which primarily consists of five stages. After the male and female worm mate, the female gives birth to live microfilariae by the thousands. The microfilariae are taken up by the vector insect (intermediate host) during a blood meal. In the intermediate host, the microfilariae molt and develop into 3rd stage (infective) larvae. Upon taking another blood meal the vector insect injects the infectious larvae into the dermis layer of the skin. The larva enters circulation and reaches the lymphatic system. The lymphatic system of the groins is mainly affected.Fertilisation then occurs and the female worm passes large number of micro-filaria into the blood stream. After approximately one year the larvae molt through 2 more stages, maturing into to the adult worm.

Individuals infected by filarial worms may be described as either "microfilaraemic" or "amicrofilaraemic," depending on whether or not microfilaria are found in their peripheral blood. Filariasis is diagnosed in microfilaraemic cases primarily through direct observation of microfilaria in the peripheral blood. **Occult filariasis** is diagnosed in amicrofilaraemic cases based on clinical observations and, in some cases, by finding a circulating antigen in the blood.

FigureTable**Incidence**

Any age group can be affected but common in tropical and forest areas, North Africa, West and North Southern Africa. Filariasis is endemic in tropical regions of Asia, Africa, Central, South America and Pacific Island nations.

Incubation Period

3 months-3 years

Clinical Features

- The most spectacular symptom of lymphatic Filariasis is elephantiasis—edema with thickening of the skin and underlying tissues—which was the first disease discovered to

be transmitted by mosquito bites. Elephantiasis results when the parasites lodge in the lymphatic system.

- Lymphangitis of the affected vessels-pain, tenderness along the coarse of the lymphatic vessels
- Elephantiasis affects mainly the lower extremities, while the ears, mucus membranes, and amputation stumps are affected less frequently.
- *Wuchereria bancrofti* can affect the legs, arms, vulva, and breasts.
- Epidydimitis,orchitis and may develop a hydrocele
- Inflammation of ankles,leges,femoral regions
- *Brugia timori* rarely affects the genitals.
- Those who develop the chronic stages of elephantiasis are usually amicrofilaraemic, and often have adverse immunological reactions to the microfilaria as well as the adult worm.
- The subcutaneous worms present with skin rashes, urticarial papules, and arthritis, as well as hyper- and hypopigmentation macules.
- *Onchocerca volvulus* manifests itself in the eyes causing "river blindness" (onchocerciasis), the 2nd leading cause of blindness in the world.
- Serous cavity Filariasis presents with symptoms similar to subcutaneous Filariasis, in addition to abdominal pain because these worms are also deep tissue dwellers.

Investigations

- Presenting signs and symptoms
- Blood slide- Blood must be drawn at night, since the microfilaria circulate at night (nocturnal periodicity), when their mosquito vector is most likely to bite. Filariasis is usually diagnosed by identifying microfilariae on a Giemsa stained thick blood film.
- X-ray of the limbs affected may reveal dead worms
- Blood count- increased white blood cell count
- In loiasis-exam of the eye may reveal keratitis
- Lymph fluid for aspiration-. Lymph Node aspirrate, Chylus fluid may also yield Microfilriae
- Polymerase chain reaction (PCR) and antigenic assays are also available for making the diagnosis. The latter are particularly useful in amicrofilaraemic cases
- Imaging like CT, MRI may reveal "Filarial Dance Sign" in Chylus fluid. X-ray can show calcified adult worm in lymphatics.
- DEC provokation test is performed to obtain satisfying number of parasite in day-time samples.
- Circulating Filarial Antigen (CFA) may be detected by PCR.

Xenodiagnosis is now obsolete EOsinophilia is a non-specific primary sign.

Treatment

- Complete bed rest

- The recommended treatment for killing adult filarial worms in patients outside the United States is albendazole (a broad spectrum anthelmintic) combined with ivermectin. A combination of diethylcarbamazine (DEC) and albendazole is also effective.
- In 2003 the common antibiotic doxycycline was suggested for treating elephantiasis. Filarial parasites have symbiotic bacteria in the genus Wolbachia, which live inside the worm. When the symbiotic bacteria are killed by the antibiotic, the worms themselves also die. Clinical trials in June 2005 by the Liverpool School of Tropical Medicine reported that an 8 week course almost completely eliminated microfilaraemia.
- Diethylcarbamazine

    Dose-3-6mg/kg body weight daily for 21 days

    Side effects-fever, nausea, headache, urticaria, prostration

  - Inerimectin

    Dose-150mg in a single dose

  - Suramin-macrofilariendal but is liable to cause severe toxic effects and has no action on micofilariae

Complications

- Hydrocele-accumulation of fluids
- Irreversible elephantiasis
- Blindness on loiasis (when loaloa worm penetrate eye ball)
- Ascites caused by blockage of lymphatic vessels of the abdomen

4.7 Summary

In this unit you've learnt the applied anatomy and physiology of the cardiovascular system. You went on to discuss the role of the nurse in investigations and procedures of the cardiovascular system.

We also looked at the management the patient with disorders of the formed elements of the blood such as the red blood cells where we discussed anaemias, white blood cells where we looked at leukaemia and lymphomas as well haemorrhagic disorders where we covered conditions like thrombocytopenia, haemophilis Ebola haemorrhagic disease etc.

We further discussed diseases of the heart which included rheumatic heart disease, congested cardiac failure coronary artery disaes and hypertention.The unit further discussed the peripheral vascular disorder which included atherosclerosis and arteriosclerosis. The tropical disease under cardiovascular disease included malaria and filariasis.

I Hope you have enjoyed and learnt something from this lesson. Below is the acticity to test your understanding of this unit .

4.8 Self Test

Self test

1. The blood vessels which carry blood away from the heart is,
   a) Aorta
   b) Coronary artery
   c) Superior vena cava
   d) Pulmonary vein
2. In severe anaemic patients the nurse is expected to find
   a) Dyspnoea and tarchycardia
   b) Cyanosis and pulmonary oedema
   c) Cardiomegaly
   d) Wheezing
3. The type leaukamei that is common in child hood is
   a) Acute myelogenous leaukaemia
   b) Acute lymphocytic leukaemia
   c) Chronic lymphocytic leukaemia
   d) Chronic granucytic leukaemia
4. The examples of megaloblastic anaemias are
   a) Iron deficiency anaemia
   b) Aplastic anaemia
   c) Folic acid deficiency and pernicious anaemia
   d) Haemolytic anaemia

Indicate true or false to the statementsT/F

5. Factors influencing the blood pressure
    a) Peripheral resistence
    b) Age
    c) Race
    d) Cardiac output
    e) Religion
    f) Emotions

Answers Q1A, ,Q2 A,Q3B,Q4C,Q5 a T,b T,c F,d T, e F, f T.

4.9 References

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