PHARMACOTHERAPEUTICS

- Pharmacotherapeutics word derived from the two words, Pharmakon means 'the drugs' and therapeutic means 'diagnosis or treatment of any disease. It is the branch of the pharmacology which deals with the drug absorption, drug distribution, drug elimination and their action/effects.
- In the other word, we can say that it is provided the information regarding the drugs and their action for preventing/eliminating the disease. On the basis of receptor capability and bioavailability drugs are act on the body and cure the disease.
- **Pharmacotherapy** is included as the drug therapy to treating the disease after the surgical, radiation, or physical therapy.
- In the modern day, different types of chemical constituted medicine or available which show the effect (may be positive or negative), to correct these problem (according to the demand or patient need) pharmacotherapeutics play a major role in the Indian system of medicine.

Branches of the Pharmacotherapeutics—

1. Pharmacokinetics—
    a. Drug absorption— Initially, when we consume the medicine then it is dissolve or break down into the smaller or absorbable particle absorbed by the different-different route according to their solubility and protein/receptor binding capacity.

➢ In our G.I.T absorption are starting from the upper parts(mouth) to middle part(stomach, intestine) and finally lower part(rectal and anal) of G.I.T.

    b. Drug distribution— After the completion of absorption, drugs are distributed to effective area through the blood or other connective tissue and finally bind to the specific receptors and shows their action. Distribution is also based on the solubility and protein/receptor binding capacity.

c. Drug elimination— on the basis of bioavailability of any drug, drug is excreted/eliminated from the body. When the bioavailability is more than drugs are binds to the receptors and show effects and waste/metabolism products are eliminate but when bioavailability are less then more amount of drug are eliminated as such through urine or fecal matter.

2. Pharmacodynamics—
   a. Systemic effects- In this branch we are study about the drug action on body organs and their responses. Different chemical show the different mechanism of action on the different body parts.
   b. Cellular effects- when the drugs are absorbed then it reach to the cell and binds to the specific cell receptors and carry on the metabolism.

Scope and objective of Pharmacotherapeutics

- We gain the correct knowledge regarding the **drug chemical reactions** in the body
- We decide the **correct drugs** categories for treating the specific disease.
- We decide the **correct dose and formulation** for treating the specific disease.
- We decide the **right patient** for the particular drug.
- We decide the **suitable/effective route of administration**
- We decide the **correct time of administration** for particular drug (many gastric related drugs take by empty stomach and many of drugs take after taking the meals)
- Decide the **drug tolerance and resistance** capacity.
- We decide the **drug incompatibility or adverse effect** (aspirin is not taken in dengue condition).
- we decide about the **drug food interactions** (calcium rich food and antibiotics should not take together).
- We decide the **natural/environmental condition** for taking the particular drug.

Rational uses of medicines (RUM)

Introduction-RMU is an important, vital and caretaker aspects/subjects include in the pharmacodynamics because of- Body mechanism of persons varies individually (obese, thin, male female, age of person etc.) due to involvement of RUM we decide that- **correct medicine for correct individual at correct time for correct diseases.**

Day to day, human activities are increases like tree cutting, pollution, more use pesticides, natural conditions are changes leads to born new physiological condition of the body so, it is an also responsible for the RUM.

According to WHO- The rational use of drugs requires the patient receive medication appropriate to their clinical needs, in doses the meet their own individual requirements for an adequate period of time and at lowest cost to them and their community.

Factors responsible for the RUM—

A. **Arises during manufacturing procedures—** It is the initial step/factor responsible for the RUM because, during the manufacturing drug quantity and measure is the important aspects regarding the drug formulation, it is avoided by the involvement of-
   1. Correct platform.
   2. Correct equipment.
   3. Correct procedure.

B. **Arise due to individual—** Body physiology of individual varies person to person so chemical requirement also varies person to person for treating any disease. It depends on the different factors-
   1. Body structure.
   2. Sex of person.
   3. Age of person.
   4. Prior disease history.
   5. Drug contraindication and allergy.
   6. Drug tolerance or resistance.

C. **Environmental effects—** Environmental condition also varies places to places so, it also act as the factors
   1. Seasonal variation.
   2. Any pandemic.
   3. Any mutations.

How to improve the RUM—

1. **Educational promotion—**Pharmacy field is the most essential field regarding for the health, because in this, we will study about the human physiology and chemical physiology (action) both and compare. So after gaining the knowledge we are designed the different-different formulation with proper quantity and quality regarding the patient requirement. So we can conclude that, if pharmacy field develop more and more with proper education purpose then many of pharmacists grown up and participate in the health promotion. Education promote by-
   1. Providing skill educational behaviors.
   2. Providing laboratory and practical knowledge.
   3. Providing the library.
   4. Providing the health and yoga camp etc.
2. **Regulatory and management promotion—** Management is also an important factor for the regulation and promoting for the RUM.
   By proper inspection and supervision we are decide the correct requirements (skill person, raw material, perfect platform, perfect equipment etc.) for the manufacturing. If manufacturing are correct and appropriate manner then we finally achieve your goals (correct dose in correct formulation).
   QA (quality assurance), QC (quality control) and GMP (good manufacturing practice) are supervision department, which inspect the manufacturing and insure that it is proper or not.
3. **By proper compounding and dispensing—** Packaging and compounding is the final or attentive stage because, if any mistake are arises during the drugs compounding then may leads to positive or negative effect and sometime cause the serious problem.

Dispensing is also including to promoting factor because of, many drugs are required special conditions during the dispensing.

4. **Establishment** of a multidisciplinary national body to co-ordinate policies on medicine uses, development and use of national essential medicine lists, sufficient government expenditure to ensure availability of medicines and staff.

Irrational use of the drugs

Irrational use of drugs means, use of medicines in improper manner and improper formulation it involves as-

- By selection of wrong medicine.
- By selection of wrong doses.
- By selection of wrong person.
- By selection of wrong time.

It is cause due to-

1. Improper diagnosis

- Improper examination of patients
- Communication problem between RMP and patient
- Improper document history.

2. Prescription problems.

- Incorrect prescriptions.
- Multiple prescriptions.
- Prescription through multimedia (By calling)

3. Dispensing and compounding

- Due to unsuitable platform/place
- Counting and compounding.
- Unsanitary procedure.

4. Common factors

- Lack of knowledge.
- OTC medication.
- Financial and unbelieves

CHAPTER – 2
Cardiovascular system

Introduction— cardiovascular system includes the hearts and the blood vessels. Heart is the vital organ in the body, which is pump the blood and blood are flow through the vessels in whole body. Any obstruction or manifestation occurs in the heart or blood vessels then it leads to many of problems. Disease affecting the heart may be structural and functional.

Anything that damages the heart, makes it less efficient, reduces its ability to fill and pump, or decrease the heart supply of oxygen will disrupt the coordinated relationship between the heat, kidney, and blood vessels and will harm not only the heart but the rest of the body as well.

Clinical consideration—

- **Hypertension.**
- **Angina pectoris.**
- **Myocardial infraction.**
- **Hyperlipidemia.**
- **Congestive heart failure**.

Heart disease may occurs due to	
Alcohol use, cocaine use, smoke	Anabolic steroid use
Atherosclerosis	Auto immune conditions
Bacterial/viral infection	Congenital abnormalities
Injury or trauma conditions	Diabetes
Thyroid dysfunction	Anabolic steroidal use
Toxin like mercury& chemotherapy drugs like HIV/AIDS drugs	
Unbalanced diet, high in fat and cholesterol (major cause)	

Hypertension.

Definition—Hypertension is defined as the high blood pressure than the normal blood pressure.

Normal value— Systolic pressure— 110 to 140 mmHg
Diastolic pressure— 60 to 80 mmHg.

When the systolic and diastolic pressure remains elevated above 150 mmHg and 90 mmHg respectively then considered as the hypertension. Commonly there is increase only in systolic pressure, it is called as systolic hypertension.

Types of hypertension—

1. Primary/Essential hypertension.
2. Secondary/systemic hypertension.

Etiopathogenesis—Primary hypertension seen as the common types of the hypertension arises due to the increased peripheral resistance or external factors in the absence of any underlying diseases.

After the long term (without any precaution) it leads to develop the vascular damage, small blood vessels damage, cerebral hemorrhage, retinal hemorrhage and renal failure.

It varies— 100 mmHg to 250 mmHg. It can control but cannot cure.

Secondary hypertension arises due to some underlying disorders. It may be occurs during the body organs are not work proper or imbalance manner due to any internal effects or any infections. It is cured by treating the disease which responsible for hypertension.

Ex-

- Cardiovascular hypertension- it arises due to the any arterial blockage.

- Renal hypertension- it arises due to obstruction of renal artery or improper glomerular filtration (glomerulonephritis).
- Endocrine hypertension- it is arises due to the hyper activity of the endocrine glands.

Clinical manifestations—

- Renal failure.
- Myocardial infraction.
- Arrhythmia.
- Cerebrovascular accident (strokes).
- Retinal hemorrhage.
- Left ventricular failure.
- Dyspnea (shortness of breath).
- Epistaxis (nose bleeds).

Pharmacological managements—

1. **Calcium channel blockers—** which block the calcium channels in myocardium and thereby, reduce the contractility of myocardium. Ex- phenylalkylamine, benzothiazepine, dihydropyridines.
2. **Vasodilators—** reduce the blood pressure by the vasodilation. Ex- sodium nitroprusside, hydralazine, minoxidil, fenoldopam, diazoxide.
3. **Diuretics—** diuretics cause diuresis and reduce the ECF volume and blood volume. Ex-
4. Angiotensin converting enzyme inhibitors **(ACE inhibitors)**— it reduce the blood pressure by blocking the formation of angiotensin.
5. **Angiotensin (AT1) receptor blocker—** Ex- losartan, telmisartan, melavinus, valsartan, eprosartan.
6. **Sympathetic inhibitors—**
    a) Alpha Beta adrenergic blockers— arotinolol, labetalol, carvedilol, bucindolol.

b) Alpha adrenergic blockers— Prazosin, doxazosin, naftopidil, phenoxybenzamine.
c) Beta adrenergic blockers— Atenolol, metoprolol, timolol, oxprenolol, nipradilol.
d) Central sympatholytics— methyldopa, reserpine, clonidine.

Non pharmacological management—

- Follow the proper routine of the regular activities (wake up, sleeps, natural urges).
- Follow regular exercise and workout (prevents the fat deposition and remove the excessive fats).
- Follow the yoga and meditation (which maintain the oxygen and carbon dioxide level).
- Make the proper diet chart after consulting the specialist and follow them. (Take- green vegetables, natural fruit juice, less fatty substance, and avoid the street food items).
- Avoid the polluted area and spend the time where fresh air blown.

Angina and Myocardial infarction.

Definition & Etiopathogenesis.

Angina pectoris— Any obstruction in the coronary artery of the heart due to deposition or blockage, leads to chest pain or any discomfort and ischemia in the heart muscles called as the angina pectoris.

The word **angina** means a type of chest caused by reduced blood flow to the heart. It is not a disease it is only a condition which occurs in the heart disease.

Medical conditions, such as Atherosclerosis, cause the walls of the blood vessels to become narrow, thereby decreasing the flow of blood. During resting, the narrowed arteries allow enough blood to reach the heart. However, the heart requires more blood than it receives during emotional stress or strenuous physical

activity. Such conditions require the heart to work harder, thereby causing angina pectoris.

It is mainly three types—

1. **Stable angina—** Deposition of the fatty material in the inner wall of the coronary artery (atherosclerosis).
2. **Unstable angina—** Any damage in the coronary arteries, causing blood clot and partial blockage (atherosclerosis with clot).
3. **Variant angina—** variation in the coronary artery diameter by any condition leads to variant angina also called as coronary spasm. Also known as Prinzmetal's variant angina or Angina inversa. It is usually rare and typically occurs in younger patients who have other pre-existing heart conditions.

Myocardial Infraction— It is also called as the **Heart Attack or myocardial necrosis**. It is the critical condition arises due to the myocardial tissue death due to lack of the blood supply. It is begins when any blockage/obstruction occurs in the arteries.

Stages of myocardial infections.

Types	Clinical consideration
Type-1 MI	Spontaneous MI due to rupture/damage of coronary artery.
Type-2 MI	Appear due to either increase oxygen demand or decreased blood flow.
Type-3 MI	Due to starting myocardial necrosis.
Type-4 MI	Due to thrombotic occlusion of a coronary stent.
Type-5 MI	Associated with cardiac surgery.

Clinical manifestations—

- Chest pain
- Dyspnea
- Fatigue
- Arrhythmia.
- Increased sweating
- Weakness
- Nausea
- Anxiety.
- Palpitation

Pharmacological managements—

a. **Beta blockers—** metoprolol, propranolol, atenolol, alprenolol.
b. **Potassium channel openers—** nicorandil
c. **Calcium channel blockers—**amlodipine, verapamil, mibefradil, bevantolol, diltiazem, nitrendipine.
d. Nitrates—
    - Long acting nitrates—isosorbide dinitrate, molsidomine.
    - Short acting nitrates— nitroglycerin, erythrityl tetranitrate.
e. **Other drugs—** oxyfedrin, ivabradine, trimetazidine, dipyridamole, acadesine.

Non pharmacological management—

- Follow the proper routine of the regular activities (wake up, sleeps, natural urges).
- Follow regular exercise and workout (prevents the fat deposition and remove the excessive fats).
- Follow the yoga and meditation (which maintain the oxygen and carbon dioxide level).
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- Avoid the polluted area and spend the time where fresh air blown.

Hyperlipidaemia.

Definition— Lipids is an important nutritional components required in the optimum measures for our diet. When the lipids or fats (such as cholesterol and triglycerides) level increase from the optimum level then it accumulates in the blood vessels and increase viscosity of blood and leads to many organs blockage (mainly hearts) like diseases.

Cholesterol is the organic molecule, a type of lipid which are the essential component for the cell. When their level are increase in the blood then it accumulate or passively flow in the blood vessel depends upon their density.

Etiopathogenesis—

1. **Primary/familial/hereditary hyperlipidemia—** It is genetically present in the child. Hereditary disorders in lipid metabolism include Tay-Sachs disease, Gaucher disease, metachromatic leucodystrophy, Fabry disease, Refsum disease etc. it further divides in many class.

Class	Increased lipoprotein
Type-I (Chylomicronemia)	Chylomicrons
Type-IIa (Hypercholesterolemia)	LDL
Type-IIb (Combined hyperlipidemia)	LDL & VLDL
Type-III (Dysbetalipoproteinemia)	LDL
Type-IV (hypertriglyceridemia)	VLDL
Type-V (mixed hyperlipidemia)	VLDL & chylomicrons

2. **Secondary/acquired hyperlipidemia—** it occurs after birth due to any abnormality or disease.

a. **Hypercholesterolemia—** hypothyroidism, nephrotic syndrome, and drugs use.
b. **Hypertriglyceridemia—** DM, alcohol, gout, chronic renal failure.

Common symptoms—

1. **Less production of bile juice—** lipids or fats are not easily digested by the gastric glands, so bile juice are one of the liver secretion which are responsible for absorption of the lipid. Bile juice breakdown the large lipid molecule into smaller particle.
2. **Liver dysfunction—** liver is responsible for the lipid metabolism and convert into the LDL and HDL. Any abnormality occur in the liver then it lead to hyperlipidemia.

Clinical manifestations—

Hyperlipidemia usually does not cause symptoms. Sometime normal symptoms seen

- Loss of appetite.
- Arrhythmia.
- Diarrhoea.
- Abdominal pain.
- Eye disorders.
- Vomiting

But very much high level of lipids or triglycerides can leads to.

- Heart attack.
- Stroke.
- Atherosclerosis.
- Xanthoma.
- Pancreatitis.

Pharmacological managements—

- (3-hydroxy-3-methyl glutaryl CoA)HMG-CoA reductase inhibitors— lovastatin, simvastatin,atorvastatin, rosuvastatin.

- Bile acid sequesteants— Cholestyramine, colestipol,
- Fibric acid derivatives— fenofibrate, benzafibrate, gemfibrozil.
- Nicotinic acid.

Non pharmacological management—

- Follow the proper routine of the regular activities (wake up, sleeps, natural urges).
- Follow regular exercise and workout (prevents the fat deposition and remove the excessive fats).
- Follow the yoga and meditation (which maintain the oxygen and carbon dioxide level).
- Make the proper diet chart after consulting the specialist and follow them. (Take- green vegetables, natural fruit juice, less fatty substance, and avoid the street food items).
- Avoid the polluted area and spend the time where fresh air blown.

Congestive heart failure.

Definition— Heart failure or congestive heart failure is an abnormal condition involving impaired cardiac pumping. In this condition heart is fail to pump the sufficient blood to our organs due to the less nutrients and oxygen supply to the myocardial destruction (less ability of cardiac muscle).

Types of heart failure—

- Left-sided heart failure— most common form of heart failure. Fluid may back up in your lungs, causing shortness of breath.

- Right-sided heart failure— often occurs with left sided heart failure. Failure may back up into your abdomen, legs and feet, causing swelling.
- Systolic heart failure—the left ventricle cannot contract vigorously indicating a pumping problem.
- Diastolic heart failure—the left ventricle cannot relax or fill fully, indicating a filling problem.

Congestive heart defects may be diagnosed before birth, right after birth, during childhood or not until adulthood. It is possible to have a defect and no symptoms at all.

Etiopathogenesis—

1. **Coronary artery disease (CAD) —** it is major cause for the heart disease. The usual causes are the build-up of plaque. This causes coronary arteries to narrow, limiting blood flow to the heart.
2. **Auto immune disease—** one of the cause of heart disease. It may be congenital or acquired.
3. **Heart valve disease—** valve is responsible for the proper flow of direction of blood common valve (tricuspid, bicuspid, pulmonary, aortic valves).when the coordination of these valve disturbed then it leads to disease.
4. **Alcohol or cocaine abuse—** Alcohol has a variety of short term and long term effects on heart. When you drink something, your blood pressure increase and your pulse rate quickens. Heavy drinking may have a long term negative impact on your cardiovascular system, including possible heart and blood vessel manage.
   Hypertension, an elevated heart rate, and a greater chance of irregular heartbeat are all side effects of drinking. Too much alcohol can lead to an accumulation of LDL or poor cholesterol.
5. **Pregnancy—** During the pregnancy anemic condition is normal so, deficiency of the iron/anemic condition blood cell production is obstructed and leads to many types of disease like heart disease.

6. **A common infection—** during any infection (bacterial infection or viral infection or any other) disturbance appears in our normal physiological activities and leads to the heart disease.
7. **Nutritional deficiency—** For proper growth and development, nutrition is required in proper ways (quantitatively and qualitatively both). When any deficiency occurs then it leads to the heart disease
8. **An endocrine/metabolic disorder—** due to manifestation in the metabolism and hormonal secretion also leads to heat disease.

Clinical manifestations—

- Tachycardia
- Oedema (swelling in ankles, legs and abdomen).
- Cachexia and muscle wasting.
- Crepitations or wheeze.
- Third heart sound
- Hepatomegaly.
- Pulses alterations
- In infant and children common symptoms occurs- cyanosis, poor weight gain, recurrent lung infections, inability to exercise, fast breathing and poor feeding.

Pharmacological managements—

1. **Calcium channel blockers—** which block the calcium channels in myocardium and thereby, reduce the contractility of myocardium. Ex- phenylalkylamine, benzothiazepine, dihydropyridines.
2. **Vasodilators—** reduce the blood pressure by the vasodilation. Ex- sodium nitroprusside, hydralazine, minoxidil, fenoldopam, diazoxide.
3. **Diuretics—** diuretics cause diuresis and reduce the ECF volume and blood volume. Ex-
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6. **Sympathetic inhibitors—**
    e) Alpha Beta adrenergic blockers— arotinolol, labetalol, carvedilol, bucindolol.
    f) Alpha adrenergic blockers— Prazosin, doxazosin, naftopidil, phenoxybenzamine.
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Chapter-2 | Pharmacotherapeutics | (b) Respiratory System

(b) Respiratory System

- Asthma
- COPD

Introduction—Exchange of gases during internal and external respiration is the major function of the respiration system.

Respiration system includes the vocal cords for producing sound, lungs for controlling body PH level and olfactory bulbs for smelling.

Respiration includes the ventilation of lungs for inward and outward movement of air alveolar air. Excretion of water vapour. Supplying air to the larynx for voice production.

Asthma

Definition— Asthma is a condition in which our airways become narrow, swell up and become more glandular (produce extra mucus). This can make breathing difficult and trigger coughing, a whistling sound (wheezing) when we breathe out and shortness of breath.

The word "asthma" originates from the Greek meaning short of breath, meaning that any patient with breathlessness was asthmatic.

Classification of Asthma—

- **Seasonal asthma—**Some of the people not tolerate even the minute changes in the season (cold, hot, rain). Due to seasonal variation allergic reaction also occurs, and leads to complicated if not treated during the starting conditions.
- **Allergic asthma—**Allergens (pollen, animal dander, dust etc.)
- **Occupational asthma—**In the chemical industries many chemicals cause the asthma.
- **Exercise-induced asthma—**Due the excessive exercise also leads the asthma.

- **Asthma-COPD overlap—** COPD (emphysema and Bronchitis) also responsible the hyper activity of the glandular activity of the mucosal cells and leads to the Asthma.

Etiopathogenesis

1. It is arising due to the hyper- responsiveness of immune system causing variable and reversible airflow obstruction. Many factors for the hyperactivity include-

- Allergens (pollen, animal dander, dust etc.)
- Upper respiratory tract infections (URTIs).
- Air pollution, cigarette smoke, other chemicals.
- Drugs (aspirin, NSAIDS, Beta-blockers).
- Food allergens, cold air and other etc.
- Genetic factor includes HLA gene mutations, defects in bronchial airway epithelium.

2. When these factors are interacted with the respiratory/bronchial mucosa then cause the hypersensitivity reactions and stimulate/triggers the helper T-cell (TH1, TH2).
3. Stimulation of B-cells to produce IgE, which binds to mast cell surface. Now, activated Helper-T cells and IgE sensitized mast cells now line the airways.
4. Now again exposure with factors, activated mast cells release histamines, leukotrienes, and other inflammatory mediators which leads to vascular permeability (Oedema of airway mucosa), Glandular cell hyperplasia (more mucus secretion), Bronchial smooth muscle contraction.
5. In other ways helper T cell, and activated mast cell also secrete the cytokines and stimulate the maturation of the granular WBCs (eosinophils, basophils, neutrophils). Finally, these cells are migrated into the other passage like airways (leads to Bronchial contraction), eyes (conjunctivitis), nose (rhinitis) etc.
6. Repeated procedure leads to the Asthma condition.

Clinical manifestations—

- Wheezing.
- Dyspnoea.
- Cough.
- Chest tightness/pain.
- Expiration may prolong.
- Thick, gelatinous sputum/mucus.

- Cardiac diseases (tachycardia).

Pharmacological managements— For management of COPD general bronchodilator and fixed combination drugs are used.

General bronchodilator

1. β-agonists

- Short acting β-agonists (SABA).
  Ex- Albuterol/salbutamol, fenoterol, terbutaline.
- Long acting β-agonists (LABA).
  Ex- salmeterol, formoterol.

General bronchodilator

2. Muscarinic antagonist (anticholinergic).

- Short acting (SAMA).
  Ex- Ipratropium.
- Long acting (LAMA)
  Ex- Acilidinium, tiotropium, glycopyrrolate bromide.

FIXED COMBINATION.

- Albuterol + Ipratropium.
- Fenoterol + Ipratrropium.
- Budesonide + Formoterol.
- Fluticasone + Salmeterol.

Non-pharmacological management—

- Avoid the allergen which is responsible for the allergic condition.
- Avoid the smoking, drinking, chewing and risks factors which is responsible for other disease manifestations.
- Regular uses of home remedies and natural products in the daily life.
- Follow/doing the regular pranayama, yoga, exercise etc. to increase the lung capacity or health.
- Sometime diets plan also required to manage the disease so, always follow the rules and regulation which are regulated by our government.

COPD

Definition—COPD (Chronic obstructive pulmonary disease) is a multifactorial entity with a wide range of clinical manifestations and leading cause of morbidity and mortality globally.

It is characterised by progressive, partially reversible airflow obstruction and lungs hyperinflation with significant extra pulmonary manifestations and comorbid conditions.

It is a group of progressive lungs disease. It is a preventable and treatable respiratory disorder largely caused by smoking, and long-term exposure to irritating gases and particulate matter. COPD often occurs in people exposed to fumes from burning fuel during cooking and heated in poorly ventilated homes.

Symptoms—Symptoms include breathing difficulty (Dyspnoea on exertion), cough, more mucus production (sputum), and wheezing.

Etiopathogenesis—

- It is mainly cause by the smoking or allergic substances. Due to long term exposure with contaminant. Respiratory receptors are modified into the secretory/glandular receptors some extent and release the large amount of mucus and obstruct the path of air.
- In the smoking, heat is entering into the respiratory path and leads to damaging of the immunological cells or receptors create the resistance or functional deformity so it also leads to the COPD conditions.
- Some harmful gaseous present in the smoking and industrial waste which are particulate, enters into the alveolar sac or alveoli and accumulate by forming the ligand compound with alveolar chemical and leads to decrease the surface area of the alveoli.
- In about 1% of people with COPD, the disease results from the genetic disorder that cause low level of protein called alpha-1-antitrypsin. It is made in the liver and secreted into the blood stream.
- Two main causes of COPD—

1. EMPHYSEMA—Emphysema destroys the air sacs in the lungs and responsible for the fibration, reduce elasticity and surface decreasement of the alveoli, so finally obstruct the gaseous exchange.

2. CHRONIC BRONCHITIS—It cause inflammation and narrowing of bronchial tube, which carry air towards lungs. It is characterized by daily cough and mucus production.

Clinical manifestations— COPD leads to many clinical manifestations.

- Heart disease- congestive heart disease, ischemic heart disease.
- Liver disease.
- Lung cancer.
- Respiratory infection (URTIs commonly include: Cough, Sore throat).
- Mental disorders.
- Pulmonary hypertension.
- Muscle fatigue etc.

Pharmacological managements— For management of COPD general bronchodilator and fixed combination drugs are used.

General bronchodilator

2. β-agonists

- Short acting β-agonists (SABA). Ex- Albuterol/salbutamol, fenoterol, terbutaline.
- Long acting β-agonists (LABA). Ex- salmeterol, formoterol.

General bronchodilator

2. Muscarinic antagonist (anticholinergic).

- Short acting (SAMA). Ex- Ipratropium.
- Long acting (LAMA) Ex- Acilidinium, tiotropium,

FIXED COMBINATION.

- Albuterol + Ipratropium.
- Fenoterol + Ipratrropium.
- Budesonide + Formoterol.
- Fluticasone + Salmeterol.

Non-pharmacological management—

- Cigarette smoking is the major causes of the COPD, so best management of COPD to avoid the smoking or to stop smoking now.
- Tobacco consumption also leads to this disease so prevent the tobacco chewing (for managing tobacco chewing, many type of the pharmaceutical chewing products are available so you can replace these with tobacco).
- Occupational exposure of chemical or particulate matter is another risk factor for the COPD, so you apply all the precautions during the working condition.
- COPD with pneumonia is also many time the cause of death so regular vaccination requires against Pneumococcal pneumonia.
- Try to avoid the industrial area and visit in good environmental conditions.
- Follow/doing the regular pranayama, yoga, exercise etc. to increase the lung capacity or health.
- Sometime diets plan also required to manage the disease so, always follow the rules and regulation which are regulated by our government.

Chapter-2 | Pharmacotherapeutics | (C) Endocrine System

(c) Endocrine System

- Diabetes
- Thyroid disorders- Hypo and Hyperthyroidism

Endocrine System.

Introduction- Endocrine system is defined as the complex glandular structure (cell modification or aggregation) which secrete the hormones and neurotransmitter in the body and regulate the body physiology and major participate in the defensive mechanism of the body.

Hormones are non-nutrient chemicals which act as intercellular messengers and are produced in trace amounts. Most hormones enter interstitial fluid and then the bloodstream. In endocrine system we have discussed about two types of glands.

1. **Exocrine glands—**Exocrine glands secrete their products (enzymes) into ducts, that carry the secretions into body cavities, into the lumen of an organ, or to the outer surface of the body. Exocrine glands include sudoriferous (sweat), sebaceous (oil), mucous, and digestive glands
2. **Endocrine gland—**Endocrine glands secrete their products (Hormone) into the interstitial fluid surrounding the secretory cells rather than into ducts. From the interstitial fluid, hormones diffuse into blood capillaries and blood carries them to target cells throughout the body. It is depending upon the heart for distribution of products.

Clinical consideration— when the hormonal secretion impaired due to any reason include external as well as internal reason leads to disease like-

- Diabetes mellitus.
- Hashimoto thyroiditis.
- Grave's disease
- Polycystic ovarian.
- Hypothyroidism.
- Hyperthyroidism

Diabetes.

Definition—Diabetes mellitus is the group of metabolic disorders sharing the common feature of hyperglycaemia. Hyperglycaemia in diabetes results from defects in insulin secretion, insulin action or both. The chronic hyperglycaemia and attendant metabolic dysregulation may be associated with secondary damage in multiple organ systems, especially the kidney, eyes, nerves, and blood vessels.

Diabetes is associated with endocrine pancreas.

Pancreas consists of clusters of cells which contains the four major type of cell types

1. **β cell— The β cells** produce insulin, which regulates glucose utilization in tissues and reduces blood glucose levels, as will be detailed in the discussion of diabetes
2. **α cell— α cells** secrete glucagon, which stimulates glycogenolysis in the liver and thus increases blood sugar.
3. **δ cell— δ cells** secrete somatostatin, which suppresses both insulin and glucagon release.
4. **PP (pancreatic polypeptide) cells— PP cells** secrete pancreatic polypeptide, which exerts several gastrointestinal effects, such as stimulation of secretion of gastric and intestinal enzymes and inhibition of intestinal motility.

The World Health Organization estimates that as many as 346 million people suffer from diabetes worldwide, with India and China being the largest contributors to the world's diabetic load.

- Blood glucose is normally maintained in a very narrow range of 70 to 120 mg/dL. According to the ADA and WHO, diagnostic criteria for diabetes include:
    1. **A fasting plasma glucose ≥ 126 mg/dL.**
    2. **A random plasma glucose ≥ 200 mg/dL.**
    3. **2-hour plasma glucose ≥ 200 mg/dL during an oral glucose tolerance test (OGTT) with a loading dose of 75 gm.**
    4. **A glycated haemoglobin (HbA1C) level ≥ 6.5%.**

Classification of diabetes mellitus— Although all forms of diabetes mellitus share hyperglycaemia as a common feature, the underlying abnormalities involved in the development of hyperglycaemia vary widely-

- **Type 1 diabetes** is an autoimmune disease characterized by pancreatic β cell destruction and an absolute deficiency of insulin. It accounts for approximately 5% to 10% of all cases.
- **Type 2 diabetes** is caused by a combination of peripheral resistance to insulin action and an inadequate secretory response by the pancreatic β cells ("relative insulin deficiency"). Approximately 90% to 95% of diabetic patients have type 2 diabetes.

Etiopathogenesis—

Type 1 diabetes mellitus— Also called 'Insulin dependent diabetes mellitus' (IDDM). It is an autoimmune disease in which islet destruction is caused primarily by immune effector cells reacting against endogenous β-cell antigens. type 1 diabetes require insulin for survival; without insulin they develop serious metabolic complications such as ketoacidosis and coma. It usually occurs in children.

Type 2 diabetes mellitus— Also called 'Insulin Independent diabetes mellitus' (IIDDM). Type 2 diabetes is a complex disease that involves an interplay of genetic and environmental factors and a proinflammatory state. Unlike type 1 diabetes, there is no evidence of an autoimmune basis. It usually occurs in middle age.

Clinical manifestations—

- Excessive hunger and thirst.
- Frequent urination.
- Progressively weight loss.
- Sudden vision changes.
- Tingling or numbness in the hands or feet.
- Very dry skin and sometime skin disorder also.
- Obstruction in blood vessel (blood supply hampered).
- Cardiac diseases also induced
- Slow healing wounds.

Pharmacological managements—

1. **Insulin.**
    - Long acting— Ex- Insulin degludec, Insulin glargine.
    - Intermediate acting— Ex- Insulin Zinc suspension.

- Short acting— Ex- Biphasic Insulin.
- Rapid acting— Ex- insulin aspart, Insulin lispro.
-

2. Oral drugs.

A. Enhance insulin secretion.

- Dipeptidyl peptidase-4 inhibitors.
  Ex- Alogliptin, Linagliptin, Saxagliptin, Sitagliptin, Teneligliptin, Vidagliptin.
- Glucagon like peptide-1 agonists.
  Ex-Albiglutide, Dulaglitide, Exenatide, Liraglutide, Lixisenatide, Semaglutide.
- K-ATP channel blockers.

B. Overcome Insulin Resistance.

- Biguanides- Ex- Buformin, Metformin, Phenformin.
- Dual peroxisome Proliferator-activator receptor agonists- Ex-

C. Miscellaneous drugs.

- Aldose reductase inhibitors- Ex- Epalrestat.
- Alpha Glucosidase Inhibitors- Ex- Acabose, Miglitol, Voglibose.
- Amylin Analogue- Ex- Pramlintide.
- Dopamine D_2 Agonist- Ex- Bromocriptine.

Non-pharmacological management—

- Increasingly sedentary life styles and poor eating habits have contributed to the simultaneous escalation of diabetes and obesity, which some have called the diabesity epidemic.
- Avoid the any type of injury.
- Make the diet plan because, in diabetes condition sugar level maintenance is the major task.
- More hunger and thrust is the common condition in the diabetes so, availability of things is very important.
- Exercise and yoga are regular require because it help in the metabolism (BMR) process of the body and maintain the glucose level.
- Try to avoid the stress and depression and visit those places where we feel happy and pleasant.
- We also take ayurvedic/natural remedies and avoid the allopathic medications.

Thyroid Disorders.

- The thyroid gland, usually located below and anterior to the larynx, consists of two bulky lateral lobes connected by a relatively thin isthmus.
- The thyroid is divided by thin fibrous septae into lobules composed of about 20 to 40 evenly dispersed follicles, lined by a cuboidal to low columnar epithelium.

A. Hypothyroidism.

Definition— Diseases of the thyroid include conditions associated with thyroid hormone deficiency is considered as hypothyroidism. It is a condition caused by a structural and functional derangement that interferes with the production of thyroid hormone. Hypothyroidism is a fairly common disorder in female than in male. Some times on the factors involving it is divided in two-

1. **Primary hypothyroidism—** it is arising due to intrinsic abnormality in the thyroid itself, or occurs as a result of pituitary and hypothalamic disease.
    - Primary hypothyroidism accounts for the vast majority of cases, and may be accompanied by an enlargement in the size of the thyroid gland (goitre). It can be congenital (due to iodine deficiency in the diet), autoimmune or iatrogenic.
2. **Secondary hypothyroidism—** It is arising from processes outside of the thyroid gland.

Etiopathogenesis—

- **Congenital hypothyroidism—** Congenital hypothyroidism is most often the result of endemic iodine deficiency in the diet. Other rare form of congenital include inborn errors of thyroid metabolism.
  In rare instances there may be complete absence of thyroid parenchyma or gland may be greatly reduced in size due to germline mutations in genes responsible for thyroid development.

- **Autoimmune hypothyroidism—** Autoimmune hypothyroidism is the most common cause of hypothyroidism in iodine-sufficient areas of the world. The vast majority of cases of autoimmune hypothyroidism are due to Hashimoto thyroiditis. Circulating autoantibodies, including anti microsomal, antithyroid peroxidase, and antithyroglobulin antibodies, are found in this disorder, and the thyroid is typically enlarged.
- **Iatrogenic hypothyroidism—** This can be caused by either surgical or radiation-induced abnormality. A large resection of the gland (total thyroidectomy) for the treatment of hyperthyroidism or a primary neoplasm can lead to hypothyroidism. Drug also decreased the thyroid secretion ex- methimazole, propylthiouracil etc.

Clinical manifestations—

Pharmacological managements— 1. Levothyroxine 2. Triiodothyronine.

Non-pharmacological management—

- Make the diet plan according to indication of physician, because iodine level maintenance in the major task during thyroid disorders.
- Avoid consumption of goitrogenic foods such as broccoli and cauliflower.
- Increase intake of yellow vegetables, egg, carrots.
- Coconut water and green tea helps to control the hypothyroidism and aids in weight loss.
- Avoid the allergic causing substances.
- Try to avoid the stress and depression and visit those places where we feel happy and pleasant.
- Follow the exercise and yoga regularly.

B. Hyperthyroidism.

Definition— Diseases of the thyroid include conditions associated with excessive release of thyroid hormones is called as hyperthyroidism. Some times on the factors involving it is divided in two-

1. **Primary hyperthyroidism—** It is arising from an intrinsic thyroid abnormality, such as Grave's disease an autoimmune disease.
2. **Secondary hyperthyroidism—**It is arising from processes outside of the thyroid, such as a TSH-secreting pituitary tumour.

Etiopathogenesis—Thyrotoxicosis is a hypermetabolic state caused by elevated circulating levels of free T3 and T4. Because it is caused most commonly by hyperfunction of the thyroid gland, it is often referred to as hyperthyroidism. Three most common cause for the hyperfunctioning for the gland include as-

- Diffuse hyperplasia of the thyroid associated with Graves' disease (approximately 85% of cases).
- Hyper functional multinodular goitre.
- Hyper functional thyroid adenoma

Hyperthyroidism also cause by the—

- Genetic defects in thyroid development.
- Thyroid hormone resistance syndrome.
- Congenital biosynthetic defect.
- Hashimoto thyroiditis.
- Iodine deficiency.
- Hypothalamic/pituitary failure (Rare).

Clinical manifestations—

- Tachycardia
- Tremors.
- Heat intolerance.
- Infertility.
- Polyphagia
- Palpitations

- Fatigue and muscle pain.
- Hair loss.
- Swelling at base of neck.

Pharmacological managements—

- Thioamides— Methimazole, Propylthiouracil.
- Iodide salts— Lugol's solution.
- Iodinated contrast media— Ipodate.
- Beta blocker— Propranolol, Esmolol.
- Anion inhibitor— Thiocyanate, perchlorate.

Non-pharmacological management—

- Make the diet plan according to indication of physician, because iodine level maintenance (low iodine) in the major task during thyroid disorders.
- Take diet rich in calcium, vitamin D, magnesium, selenium.
- Avoid intake of sugar, caffeine, alcohol etc. products.
- Avoid the allergic causing substances.
- Try to avoid the stress and depression and visit those places where we feel happy and pleasant.
- Follow the exercise and yoga regularly.

Chapter—2 (D) Pharmacotherapeutics Central Nervous System

(d) Central Nervous System

- Epilepsy
- Parkinson's disease
- Alzheimer's disease
- Stroke
- Migraine

Central Nervous System

Introduction—The principal functional unit of the central nervous system (CNS) is the neuron.

- Neuron is the structural and functional unit of the nervous system; it has the unique ability to receive and transmit information.
- Neurons of different types and in different locations have distinct properties, including functional roles, distribution of their connections, neurotransmitters used, metabolic requirements, and levels of electrical activity at a given moment.
- In addition to neurons the CNS contains other cells, such as astrocytes and oligodendrocytes, which make up the neuroglia. During any injury or abnormality these cells undergo a range of functional and morphological changes and leads to many of neurological disorders.

Clinical consideration—

- Epilepsy.
- Parkinson's disease.
- Alzheimer's disease.
- Stroke.
- Migraine.
- Encephalopathy
- Seizure.

- Meningoencephalitis.
- Cerebral palsy.

Epilepsy

Definition— Epilepsy is a neurological disorder in which brain activity becomes abnormal, causing seizures, sensations and sometimes loss of awareness.

- Epilepsy can cause by brain injury from stroke, trauma, a mass lesion, or infection. About two-thirds of new cases arise in children, and most of these cases are idiopathic or caused by trauma. In contrast, seizures or epilepsy with onset in adult life is more often due to underlying brain lesions or metabolic causes.

Classification of seizures— Seizure is classified on the basis of behavioural and electrophysiologic pattern of activity as-

1. **Partial (Focal seizures) —** It mainly appears in only one hemisphere. That means symptoms only happen in a specific part or on one side of your body. But focal seizures can sometimes spread and become generalized seizures.
    a. Simple partial seizures with motor, sensory, or autonomic symptoms.
    b. Complex partial seizures.
    c. Partial seizures with secondary generalization.
2. **Generalized seizures—** These are seizures that happen in both hemispheres of your brain side. These seizures tend to cause more severe effects and symptoms.
    a. Absence seizures.
    b. Tonic- clonic seizures.
    c. Other (Myoclonic, tonic, clonic, atonic).

Etiopathogenesis—

- Normal neuronal activity occurs in a non-synchronized manner, with groups of neurons inhibited and excited sequentially during the transfer of information between different brain areas.
- Seizures occur when neurons are activated synchronously. The kind of seizure depends on the location of the abnormal activity and the pattern of spread to different parts of the brain, experimentally, this is known as the paroxysmal depolarizing shift.

- Most inhibitory synapses use the neurotransmitter GABA.
- In secondary epilepsy, loss of inhibitory circuits and sprouting of fibres from excitatory neurons appear to be important for the generation of a seizure focus. In several of the idiopathic epilepsies, genetic studies have identified mutations in ion channels.

Clinical manifestations—

- Cardiac syncope (Arrhythmia).
- Non-Cardiac syncope (vasovagal).
- Breathing problems (dyspnoea).
- Sleep disorders (Narcolepsy).
- Movement disorders (Paroxysmal dyskinesia).
- Metabolic disorders (Hypoglycaemia).
- Migraine's (generally confusion migraine).
- Loss of bowel or bladder control.

Pharmacological managements—

Chemically drugs are classified as-

- Benzodiazepines— ex- clonazepam, lorazepam, diazepam.
- Barbiturates— ex- phenobarbital, desoxyphenobarbital.
- Deoxy barbiturates— ex- primidone.
- Hydantoin— ex-phenytoin, ethotoin.
- Aliphatic carboxylic Acid— ex- valproic acid, magnesium valproate.
- Oxazolidine derivatives— ex- trimethadione, paramethadione.
- Cyclic GABA Analogues— ex- gabapentin, pregabalin.
- Iminostilbene— carbamazepine, oxcarbazepine.
- Other drugs— ex- levetiracetam, parampanel, brivaracetam, lacosamide.

Non-pharmacological management—

- Avoid and discard the all activities which cause/induce the depression, stress, sleep disorders etc.
- Follow and change the diet plans according to own demand or prescription by any RMP.

- Some small extent caffeine, alcohol, nicotinic substance is managing the brain disorder but avoid the ingestion of excessive amounts of these.
- Practice the yoga, meditation, physical exercise regularly. Ventilation is one of the reasons which leads to brain disorders and cardiac disorders also.
- Practice of herbal/natural medicine other than allopathic.
- Do such all activities which makes you happy and cheerful.

Parkinson's Disease

Definition—

- Parkinson Disease (PD) is a neurodegenerative disease marked by a prominent hypokinetic movement disorder that is caused by loss of dopaminergic neurons from the substantia nigra.
- The clinical syndrome of parkinsonism combines diminished facial expression, stooped posture, slowing of voluntary movement, festinating gait (progressively shortened, accelerated steps), rigidity, and a "pill-rolling" tremor.

Etiopathogenesis—

- PD is associated with protein accumulation and aggregation, mitochondrial abnormalities, and neuronal loss in the substantia nigra and elsewhere in the brain.
- Parkinsonism may also result from repeated head trauma or may be a feature of several basal ganglia diseases, including Wilson disease, some cases of early-onset Huntington disease, multiple system atrophy (MSA), and progressive supranuclear palsy.
- Parkinsonism can also result from exposure to certain toxins such as manganese, carbon disulphide, 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), and carbon monoxide.

Clinical manifestations—

1. **Primary symptoms.**
    - Bradykinesia.
    - Parkinson gait
    - Rigidity
    - Tremors
    - Postural instability.
2. **Secondary symptoms.**
    - Confusion.
    - Memory loss.
    - Anxiety.
    - Depression
    - Difficulty swallowing.
    - Increased sweating.
    - Constipation.

Pharmacological managements—

1. **Drugs acting on cholinergic system—**
    - Anti-histaminic— ex-promethazine, orphenadrine.
    - Central cholinergic— ex- biperiden, trihexyphenidyl, procyclidine.
2. **Drugs acting on dopaminergic system—**
    - Glutamate (NMDA receptor) agonist— ex- Amantadine
    - Catechol-o-methyltransferase (COMT) inhibitors—ex-entacapone.
    - Peripheral decarboxylase inhibitors—ex-carbidopa, benserazide.
    - Dopaminergic agonists—ex- bromocriptine, ropinirole, piribedil.
    - MAO-B inhibitors—ex- selegiline, rasagiline, safinamide.
    - Dopamine precursor—ex- levodopa.
3. **Adenosine receptor antagonist—**ex- istradefylline.

Non-pharmacological management—

- Avoid and discard the all activities which cause/induce the depression, stress, sleep disorders etc.
- Follow and change the diet plans according to own demand or prescription by any RMP.

- Some small extent caffeine, alcohol, nicotinic substance is managing the brain disorder but avoid the ingestion of excessive amounts of these.
- Practice the yoga, meditation, physical exercise regularly. Ventilation is one of the reasons which leads to brain disorders and cardiac disorders also.
- Practice of herbal/natural medicine other than allopathic.
- Do such all activities which makes you happy and cheerful.

Alzheimer's disease

Definition—

- Alzheimer disease (AD) is the most common cause of dementia in older adults, with an increasing incidence as a function of age.
- The disease usually becomes clinically apparent as insidious impairment of higher cognitive functions.
- As the disease progresses, deficits in memory, visuospatial orientation, judgment, personality and language emerge.

Etiopathogenesis—

- The fundamental abnormality in AD is the accumulation of two proteins (Aβ and tau) in specific brain regions, likely as a result of excessive production and defective removal.
- The two pathologic Indication of AD, particularly evident in the end stages of the illness, are **plaques and tangles.**
- Plaques are deposits of aggregated Aβ peptides in the neuropil, while tangles are aggregates of the microtubule binding protein tau, which develop intracellularly and then persist extracellularly after neuronal death.
- Both plaques and tangles appear to contribute to the neural dysfunction, and the interplay between the processes that lead to the accumulation of these abnormal aggregates is a critically important aspect of AD pathogenesis.

Genetic Evidences— Multiple lines of genetic evidence point to the likely importance of altered Aβ metabolism.

- Mutations in the protein from which Aβ is derived (APP) cause familial AD, as does increased copy number of the APP gene.
- Furthermore, point mutations in proteins that are part of the protease complexes that generate Aβ from APP also give rise to AD.

Clinical manifestations—

- Deficits in memory.
- Lack of enthusiasm.
- Poor or decreased judgement.
- Difficulty in having elaborate thoughts.
- Disorientation in time and space.
- Drastic behaviour changes.
- Problems in speaking, reading and writing.

Pharmacological managements— drugs used as-

- Cholinesterase inhibitors— ex- donepezil, rivastigmine, galantamine.
- NMDA antagonist— ex- memantine

Non-pharmacological management—

- Avoid and discard the all activities which cause/induce the depression, stress, sleep disorders etc.
- Follow and change the diet plans according to own demand or prescription by any RMP.
- Some small extent caffeine, alcohol, nicotinic substance is managing the brain disorder but avoid the ingestion of excessive amounts of these.
- Practice the yoga, meditation, physical exercise regularly. Ventilation is one of the reasons which leads to brain disorders and cardiac disorders also.
- Practice of herbal/natural medicine other than allopathic.
- Do such all activities which makes you happy and cheerful.

Stroke

Definition— Stroke is a clinical syndrome characterized by the sudden onset of a focal neurologic deficit that persists for at least 24 hours and results from an abnormality of the cerebral circulation. The incidence of stroke increases with age and is higher in men than in women. Significant risk factors include hypertension, hypercholesterolemia, diabetes, smoking, heavy alcohol consumption, and oral contraceptive use.

Etiopathogenesis— The focal symptoms and signs that result from stroke correlate with the brain area supplied by the affected blood vessel. There are two major categories of stroke, based on pathogenesis: ischemic and haemorrhagic stroke.

1. **Ischemic stroke—** Ischemic strokes result from thrombotic or embolic occlusion of cerebral vessels. Neurologic deficits caused by the occlusion of large arteries result from focal ischemia to the area of brain supplied by the affected vessel and produce recognizable clinical syndromes.
    - Anterior cerebral symptoms— Sensory loss in movement (foot).
    - Middle cerebral symptoms— Aphasia and neglect (dominant and non-dominant) hemisphere.
    - Posterior cerebral symptoms— Sensory loss, ataxia, nerve palsy etc.

Ischemic strokes involving occlusion of small arteries occur at select locations, where perfusion depends on small vessels that are end arteries. Most result from a degenerative change in the vessel, described pathologically as lipohyalinosis, which is caused by chronic hypertension and predisposes to occlusion.

2. **Haemorrhagic stroke**—
    - Epidural and subdural hematomas typically occur as sequelae of head injury. Epidural hematomas arise from damage to an artery, typically the middle meningeal artery, which can be ruptured by a blow to the temporal bone. Blood dissects the dura from the skull and compresses the hemisphere lying below. Initial loss of consciousness from the injury results from concussion and may be transient.
    - Subarachnoid haemorrhage may occur from head trauma, extension of blood from another compartment into the subarachnoid space, or rupture of an arterial aneurysm. Cerebral

dysfunction occurs because of increased intracranial pressure and from poorly understood toxic effects of subarachnoid blood on brain tissue and cerebral vessels.

- Intraparenchymal haemorrhage may result from acute elevations in blood pressure or from a variety of disorders that weaken vessels. The resultant hematoma causes a focal neurologic deficit by compressing adjacent structures.

Clinical manifestations—

- Face dropping
- Sudden vision disturbance.
- Arm or limb weakness.
- Loss of coordination
- Nausea.
- Trouble speaking.
- Sudden confusion and sometime memory loss.
- Tremors also occurs.

Pharmacological managements—

Tissue plasminogen activator (tPA) is the only stroke drug that actually breaks up a blood clot.

- Non fibrin specific— ex- streptokinase, urokinase, Anistreplase.
- Fibrin specific— ex- Alteplase, reteplase, Tenecteplase.

Non-pharmacological management—

- Avoid and discard the all activities which cause/induce the depression, stress, sleep disorders etc.
- Follow and change the diet plans according to own demand or prescription by any RMP.
- Some small extent caffeine, alcohol, nicotinic substance is managing the brain disorder but avoid the ingestion of excessive amounts of these.

- Practice the yoga, meditation, physical exercise regularly. Ventilation is one of the reasons which leads to brain disorders and cardiac disorders also.
- Practice of herbal/natural medicine other than allopathic.
- Do such all activities which makes you happy and cheerful.

Migraine

Definition—

- A migraine is a headache that can cause severe throbbing pain or a pulsing sensation, usually on one side of the head and also involving altered regulation and control of afferents, with a particular focus on the cranium. It's often accompanied by nausea, vomiting, and extreme sensitivity to light and sound.
- Current views concerning migraine will be reviewed concluding the disorder is a disturbance in the brain of the subcortical aminergic sensory modulatory systems, in addition to other brainstem, hypothalamic and thalamic structures.

Etiopathogenesis—

- Pathophysiology of migraine should be based upon the anatomy and physiology of the pain-producing structures of the cranium integrated with knowledge of their central nervous system modulation.

It involved/understand by four phases.

1. **Prodromal symptoms**—It is initial symptoms about one or two days before a migraine including constipation, mood changes from depression to euphoria, food cravings, neck stiffness, increased thirst and urination or frequent yawning, difficulty in speaking, reading, and sleeping.
2. **Aura**— Migraine with aura (also called classic migraine) is a recurring headache that strikes after or at the same time as sensory disturbances called aura it is about 5-60 minutes. These disturbances can include flashes of light, blind spots, and other vision changes or tingling in your hand or face.

3. **Migraine attack/Headache**— It is about 4-72 hours. These disturbances can include burning, nausea, vomiting, giddiness, insomnia, anxiety, depressed mood, sensitivity to light, smell, sound.
4. **Postdrome**— It is about 24- 48 hours. These disturbances can include as inability to concentrate, fatigue, depressed mood, euphoric mood, lack of comprehension.

Clinical manifestations—

- Throbbing, drilling, pounding headache.
- Nausea, vomiting, giddiness.
- Sensitivity towards light and sound.
- Pain in only one hemisphere.
- Diarrhoea, constipation, stomach pain.
- Inability to focus on concentrate.

Pharmacological managements—

- Prophylactic— beta blocker, calcium channel blocker, ACE inhibitor etc.
- Abortive—for blocking the pain pathways many drugs are used Example- NSAIDS, ergotamine, sumatriptan and rizatriptan.

Non-pharmacological management—

- Avoid and discard the all activities which cause/induce the depression, stress, sleep disorders etc.
- Follow and change the diet plans according to own demand or prescription by any RMP.
- Some small extent caffeine, alcohol, nicotinic substance is managing the brain disorder but avoid the ingestion of excessive amounts of these.
- Practice the yoga, meditation, physical exercise regularly. Ventilation is one of the reasons which leads to brain disorders and cardiac disorders also.
- Practice of herbal/natural medicine other than allopathic.
- Do such all activities which makes you happy and cheerful.

Chapter—2 (E) Pharmacotherapeutics
Gastro intestinal system

Introduction— The gastrointestinal (GI) tract is a hollow tube extending from the oral cavity to the anus that consists of anatomically distinct segments, including the oesophagus, stomach, small intestine, colon, rectum, and anus. Each of these segments has unique, complementary, and highly integrated functions, which together serve to regulate the intake, processing, and absorption of ingested nutrients and the disposal of waste products.

Clinical consideration—

- Irritable bowel syndrome.
- Hypertrophic pyloric stenosis.
- Oesophageal achalasia.
- Gastro oesophageal reflux disease (GERD).
- Peptic ulcer/gastric ulcer/duodenal ulcer.
- Inflammatory bowel diseases (IBDs).
- Alcoholic liver disease etc.

Gastro oesophageal reflux disease (GERD)

Definition— GERD also known as acid reflux, is most common in individuals older than age 40 but also occurs in infants and children. Symptoms are often worse at night, when lying supine, or after consuming foods or drugs that diminish lower oesophageal sphincter tone, such as caffeinated beverages. The stratified squamous epithelium of the oesophagus is resistant to abrasion from foods but is sensitive to acid. Submucosal glands, which are most abundant in the proximal and distal oesophagus, contribute to mucosal protection by secreting mucin and bicarbonate. More importantly, the tone of the lower oesophageal sphincter prevents reflux of acidic gastric contents, which are under positive pressure and would otherwise enter the oesophagus. Reflux of gastric contents into the lower oesophagus is the most frequent cause of GERD (Gastro oesophageal reflux disease).

Etiopathogenesis—

- The most common cause of gastroesophageal reflux is transient lower oesophageal sphincter relaxation. This is thought to be mediated via vagal pathways, and can be triggered by gastric distention, by gas or food, mild pharyngeal stimulation that does not trigger swallowing, and stress.
- Gastroesophageal reflux can also occur due to forceful opening of a relatively hypotensive lower oesophageal sphincter by an abrupt increase in intraabdominal pressure, such as that due to coughing, straining, or bending.
- Other conditions that decrease lower oesophageal sphincter tone or increase abdominal pressure and contribute to GERD include alcohol and tobacco use, obesity, central nervous system depressants, pregnancy, hiatal hernia, delayed gastric emptying, and increased gastric volume.

Clinical manifestations—

- Burning sensation in the chest region (Heart burn).
- Regurgitation (Backflow of food or liquid).
- Dysphagia (Trouble swallowing).
- Sensation of a lumb in throat.
- Barrett oesophagus (Chronic recurrent reflux can also result in a change in the oesophageal epithelium from squamous to columnar histology).

Pharmacological managements— Omeprazole, Rabeprazole, Pantoprazole, esomeprazole, Lansoprazole, dexlansoprazole etc.

Non-pharmacological management—

- Maintain/regulate the food intake (time to time) and take food after proper digestion of the previous food.
- If any complication appears and consults with doctors and make the diet charts and fallow strictly.
- Water help the regulating the acid reflex and helps in digestion, so water consumption also maintain the GERD.
- Sleeping/sitting posture also important for maintaining the GERD.
- Follow regular yoga and exercise according to need because physically activity also helps in the digestive activity.
- Electrolytes are also very important in this condition.
- Avoid more species food and diet (because it cause acidity).

Peptic Ulcer Disease.

Definition— Peptic ulcer disease (PUD) refers to chronic mucosal ulceration affecting the duodenum or stomach. Nearly all peptic ulcers are associated with *Helicobacter pylori* infection, NSAIDs, or cigarette smoking. PUD results from imbalances between mucosal defence mechanisms and damaging factors that cause chronic gastritis. Thus, PUD generally develops on a background of chronic gastritis. Gastric peptic ulcers are predominantly located along the lesser curvature near the interface of the body and antrum. Peptic ulcers are solitary in more than 80% of patients.

Etiopathogenesis—

- Various causes of absolute or relative increased acid production or decreased mucosal defences predispose to acid-peptic disease.
- Bacterium *Helicobacter pylori* is the root cause of a number of forms of acid-peptic disease, including duodenal ulcer, gastric ulcer, and gastritis. It can cause acid-peptic disease by multiple mechanisms, including direct alteration of signal transduction in mucosal and immune cells, which in turn can increase acid secretion and diminish mucosal defences.
- PUD may also be caused by acid secreted by ectopic gastric mucosa within the duodenum or an ileal Meckel diverticulum. PUD may also occur in the oesophagus as a result of GERD or acid secretion by oesophageal ectopic gastric mucosa (an inlet patch).

Clinical manifestations—

- Epigastric burning or aching pain.
- Iron deficiency anaemia.
- Haemorrhage/ GI tract bleeding.
- Hematemesis (vomiting of blood).
- Melena (tarry stools from the effect of acid on blood).
- Perforation and infections.

Pharmacological managements—

- Prostaglandin analogues. Ex- misoprostol, rioprostil.
- H_2 receptor antagonists. Ex- ranitidine, famotidine, cimetidine.
- Proton pump inhibitors. Ex- Omeprazole, Rabeprazole, Pantoprazole.
- Anti- cholinergics. Ex- anistropine, hyoscyamine.

Non-pharmacological management—

- Maintain/regulate the food intake (time to time) and take food after proper digestion of the previous food.
- If any complication appears and consults with doctors and make the diet charts and fallow strictly.
- Change the life style and take the warm water.
- Follow regular yoga and exercise according to need because physically activity also helps in the digestive activity.
- Electrolytes are also very important in this condition.
- Avoid more species food and diet (because it cause acidity).
- Avoid smoking and alcohol consumption.

Alcoholic liver disease

Definition— Alcohol (ethanol) is the most useful chemical agent in the pharmaceutical industry for the preparation of the drugs, as well as it is also used as the solvents. Now a days alcohol consumption is common in our traditional culture but regular practice of alcohol leads to the addiction and cause the many types of disease (heart disease, liver disease, G.I.T infections, lungs disease etc).

Alcoholic liver disease is a chronic disorder that can give rise to steatosis, alcoholic hepatitis, progressive steatofibrosis and marked derangement of vascular perfusion leading eventually to cirrhosis. Consumption of 80 gm/day of alcohol is considered to be the threshold for the development of alcoholic liver disease.

Excessive alcohol (ethanol) consumption is the leading cause of liver disease it is accounts for 3.8% of deaths globally, making it the eighth highest risk factor for death.

Etiopathogenesis—Short-term ingestion of as much as 80 gm of alcohol over one to several days generally produces mild, reversible hepatic steatosis. Daily intake of 80 gm or more of ethanol generates significant risk for severe hepatic injury, and daily ingestion of 160 gm or more for 10 to 20 years is caused severe injury. There are three manifested stages discussed-

1. **Hepatocellular steatosis (fatty liver)—** It is the initial condition arises due to the due to moderate consumption of alcohol, in this condition lipids droplets getting accumulate in the hepatocytes cell and finally small droplets coalesce into large droplets.
   Macroscopically, the fatty liver in individuals with chronic alcoholism is a large (as heavy as 4 to 6 kg), soft organ that is yellow and greasy. **Fatty change is completely reversible if there is abstention from further intake of alcohol.**

2. **Alcoholic hepatitis**— it is characterized by-
   - Hepatocyte swelling and necrosis— The swelling results from the accumulation of fat and water, as well as proteins that are normally exported.
   - Mallory-Denk bodies—These are usually present as clumped, amorphous, eosinophilic material in swell hepatocytes. It is not specific feature of alcoholic liver disease, since they are also present in non-alcoholic fatty liver disease also.
   - Neutrophilic reaction— Neutrophils permeate the hepatic lobule and accumulate around degenerating hepatocytes, particularly those having Mallory-Denk bodies.
3. **Alcoholic steatofibrosis (cirrhosis)—**Alcoholic hepatitis is often accompanied by prominent activation of sinusoidal stellate cells and portal fibroblasts, giving rise to fibrosis. Fibrosis begins with sclerosis of central veins.

Clinical manifestations—

- Abdominal swelling.
- Jaundice
- Haematological disorders
- Indigestion and constipation.
- Fainting and mental disturbance.
- Renal disorders.

Pharmacological managements— Metadoxine, deoxycholic acid, ursodiol, l-arginine, obeticholic acid.

Non-pharmacological management—

- Maintain/regulate the food intake (time to time) and take food after proper digestion of the previous food.

- If any complication appears and consults with doctors and make the diet charts and fallow strictly.
- Change the life style and take the warm water.
- Follow regular yoga and exercise according to need because physically activity also helps in the digestive activity.
- Electrolytes are also very important in this condition.
- Avoid more species food and diet (because it cause acidity).
- Avoid smoking and alcohol consumption.

Inflammatory Bowel Diseases (Crohn's Disease and Ulcerative Colitis).

Definition—Inflammatory bowel disease (IBD) is distinguished from infectious entities by exclusion and by chronicity due to the inappropriate mucosal immune activation. Patients often experience recurrent episodes of mucopurulent (containing mucus and white cells), bloody diarrhoea due to pathogens and failure to respond to antibiotics alone. The two disorders that comprise IBD are Crohn's disease and ulcerative colitis. The distinction between ulcerative colitis and Crohn disease is based, in large part, on the distribution of affected sites and the morphologic expression of disease at those sites.

a. **Crohn's disease—Crohn disease**, which has also been referred to as regional enteritis (because of frequent ileal involvement) may involve any area of the GI tract (including the oral cavity, oesophagus, stomach, and proximal small intestine). The combination of deep mucosal ulceration and submucosal thickening gives the involved mucosa a characteristic "cobblestone" appearance.
    - In this condition intestinal lesions are appeared in skip manner like (patches of lesion) and transmural inflammation, ulcerations and fissures are present.
b. **Ulcerative colitis—Ulcerative colitis** is limited to the colon and rectum and extends only into the mucosa and submucosa. It typically begins at the anorectal junction and extends proximally
    - In this condition intestinal lesions are appeared in continuous manner (mainly in colonic and rectum) and pseudo polyp mucosal ulcer are present.

Etiopathogenesis—

- The two disorders that comprise IBD are ulcerative colitis and Crohn's disease.

- Most investigators believe that IBD results from the combined effects of alterations in host interactions with intestinal microbiota, intestinal epithelial dysfunction, aberrant mucosal immune responses, and altered composition of the gut microbiome. Most of the condition mucosal immune activation and defective immunoregulation contribute to the development of ulcerative colitis and Crohn disease.

Clinical manifestations—

a. **Crohn's disease**
   - Arthritis (inflammatory disorders of the joints).
   - Erythema nodosum (skin disorder).
   - In eye (uveitis, iritis).
   - Aphthous ulcers of the buccal mucosa.
   - In bile ducts (sclerosing cholangitis).
   - In liver (autoimmune chronic active hepatitis)
   - Renal disorders, especially nephrolithiasis.
   - Amyloidosis is a serious complication of Crohn disease

b. **Ulcerative colitis.**
   - Obstructions
   - Perforations.
   - Fistula formation.
   - Infections.

Pharmacological managements— alosetron, lubiprostone, rifaximin, loperamide, linaclitide, tenapanor, eluxadoline, Azathioprine,mercaptopurine.

Non-pharmacological management—

- Maintain/regulate the food intake (time to time) and take food after proper digestion of the previous food.
- If any complication appears and consults with doctors and make the diet charts and fallow strictly.
- Change the life style and take the warm water.
- Follow regular yoga and exercise according to need because physically activity also helps in the digestive activity.
- Electrolytes are also very important in this condition.
- Avoid more species food and diet (because it cause acidity).
- Avoid smoking and alcohol consumption.

Chapter-2 (f)
Haematological disorders.

Haematological disorders

- Iron deficiency anaemia
- Megaloblastic anaemia

Haematological disorders

Introduction

Blood is an extremely complex fluid, composed of both formed elements (red cells, white cells, platelets) and plasma. RBCs (erythrocytes) are the most common formed elements, carrying Oxygen and haemoglobin.

- White blood cells are function as mediators of immune responses to infection or other stimuli of inflammation.
- Platelets are the formed elements that participate in coagulation. Plasma is largely water, electrolytes, and plasma proteins. The plasma proteins most important in blood clotting are the coagulation factors.
- A group of haematological disorders characterized by the any disturbance in the physiological and morphological changes in the blood cell is called haematological disorder.

Clinical consideration— Most common haematological disorder is Anaemia.

Classification of Anaemia.

1. **Morphological classification**.
    a. Normocytic normochromic anaemia.
    b. Macrocytic normochromic anaemia
    c. Macrocytic hypochromic anaemia.
    d. Microcytic hypochromic anaemia.
2. **Etiological classification.**
    a. Haemorrhagic anaemia.
    b. Haemolytic anaemia.
    c. Aplastic anaemia.
    d. Anaemia due to other disease.

e. Nutritional deficiency anaemia.
   - Iron deficiency anaemia.
   - Protein deficiency anaemia.
   - Vitamin B_{12} anaemia.
   - Folic acid anaemia.

Iron deficiency anaemia.

Definition.

- Iron deficiency anaemia is most common nutritional disorder in the world. It develops due to inadequate availability of iron for haemoglobin synthesis. RBCs are microcytic and hypochromic. Dietary deficiency of iron is most common cause in the developing country.
- Blood loss in this case may result from relatively benign disorders, such as peptic ulcer, arteriovenous malformations, or angiodysplasia (small vascular abnormalities along the intestinal walls). More serious causes are inflammatory bowel disease and malignancy.
- Daily requirement of iron is about 10 to 20 mg. It varies person to person. About 80% of the functional iron is found in haemoglobin, myoglobin and iron-containing enzymes.

Etiopathogenesis.

- Dietary deficiency of iron is most common cause in the developing country. Menstrual blood loss, premenopausal is also major cause in the female.
- Helicobacter pylori infection, partial gastrectomy, bleeding disorders and haemoptysis are also the cause of iron deficiency anaemia.
- **Hepcidin (25-amino acid peptide) produced by liver,** is also cause for the anaemia because it exports the iron to plasma after recycling of senescent erythrocyte via macrophages phagocytosis and lysis.
- Hepcidin binds to ferroportin, a transmembrane protein, inducing its internalization and lysosomal degradation. When iron stores are low, hepcidin production is reduced and ferroportin molecules are expressed on the basolateral membrane of enterocytes, where they transfer iron from the cytoplasm of enterocytes to plasma transferrin. Conversely, when iron stores are adequate or elevated, hepcidin production is increased, resulting in the internalization of ferroportin and reduced export of iron into plasma.

Clinical manifestations.

- Increase in the BMR (basal metabolic rate).
- Heart disease occurs (Tachycardia, angina, heart attack).
- Spleen enlargement, liver enlargement.
- Hyperventilation and dyspnoea.
- Menorrhagia, oligomenorrhea/amenorrhea.
- Dysphagia, anorexia, nausea vomiting.
- Headache and dizziness.
- Fatigue and weakness.
- Skin and mucosa turn yellow in colour.
- Brittle hair and nails, atrophy of papilla in tongue.

Pharmacological managements—

- Oral iron— ferrous sulphate, ferrous aminoate, ferrous gluconate, ferrous succinate, carbonyl iron, iron calcium complex.
- Parenteral iron— Iron sucrose, iron dextran, iron isomaltoside, ferric carboxy maltose, ferric pyrophosphate citrate.

Non-pharmacological management—

- Daily intake of iron richest sources are green vegetables such as lettuce, spinach, asparagus, and broccoli. Certain fruits (e.g., lemons, bananas, melons) and animal sources (e.g., liver, meat especially dark meat).
- Digestive activity is very important factor for the absorption of iron, so follow all the regular regimen including yoga, exercise, balance diet etc.
- During the pregnancy/menstrual flow iron deficiency is common, so more iron rich supplement and more take care needed.

Megaloblastic anaemia.

Definition.

- **Megaloblastic anaemia** is developing due to the deficiency of maturation factor called **folic acid**. Ion this condition, RBCs are not matured and DNA synthesis is also defective/impairment leads to ineffective haematopoiesis and distinctive morphologic changes, including abnormally large erythroid precursors and red cells. The RBCs are megaloblastic and hypochromic.
- Decreased intake can result from either a nutritionally inadequate diet or impairment of intestinal absorption. Megaloblastic anaemia that results from a deficiency of folic acid is identical to that encountered in vitamin B12 deficiency. Thus, the diagnosis of folate deficiency can be made only by demonstration of decreased folate levels in the serum or red cells.

Etiopathogenesis— The three major causes of folic acid deficiency are Decreased intake of iron, Increased requirements of iron, Impaired utilization of iron.

- Humans are entirely dependent on dietary sources for their folic acid requirement, which is 50 to 200 µg daily. The richest sources are green vegetables such as lettuce, spinach, asparagus, and broccoli. Certain fruits (e.g., lemons, bananas, melons) and animal sources (e.g., liver). The folic acid in these foods is largely in the form of folylpolyglutamates. Intestinal conjugases split the polyglutamates into monoglutamates that are readily absorbed in the proximal jejunum. During intestinal absorption they are modified to 5-methyltetrahydrofolate, the normal transport form of folate. The body's reserves of folate are relatively modest, and a deficiency can arise within weeks to months if intake is inadequate.
- The megaloblastic anaemia is often accompanied by general malnutrition and manifestations of other avitaminoses, including cheilosis, glossitis, and dermatitis. Malabsorption syndromes, such as sprue, can lead to inadequate absorption of this nutrient, as can diffuse infiltrative diseases of the small intestine (e.g., lymphoma).

Clinical manifestations.

- Increase in the BMR (Basal Metabolic Rate).
- Heart disease occurs (Tachycardia, angina, heart attack).

- Spleen enlargement, liver enlargement.
- Hyperventilation and dyspnoea.
- Menorrhagia, oligomenorrhea/amenorrhea.
- Dysphagia, anorexia, nausea vomiting.
- Headache and dizziness.
- Fatigue and weakness.
- Skin and mucosa turn yellow in colour.
- Brittle hair and nails, atrophy of papilla in tongue.

Pharmacological managements—Iron containing preparation is given as-

- Oral iron— ferrous sulphate, ferrous aminoate, ferrous gluconate, ferrous succinate, carbonyl iron, iron calcium complex.
- Parenteral iron— Iron sucrose, iron dextran, iron isomaltoside, ferric carboxy maltose, ferric pyrophosphate citrate.
- Maturation factors— Hydroxocobalamin, methyl cobalamin, cyanocobalamin, folinic acid/leucovorin.

Non-pharmacological management.

- Daily intake of iron richest sources are green vegetables such as lettuce, spinach, asparagus, and broccoli. Certain fruits (e.g., lemons, bananas, melons) and animal sources (e.g., liver).
- Digestive activity is very important factor for the absorption of iron, so follow all the regular regimen including yoga, exercise, balance diet etc.
- During the pregnancy/menstrual flow iron deficiency is common, so more iron rich supplement and more take care needed.

Chapter-2 (g)

Infectious diseases

Infectious diseases

- Tuberculosis
- Pneumonia
- Urinary tract infections
- Hepatitis
- Gonorrhoea and Syphilis
- Malaria
- HIV and Opportunistic infections
- Viral Infections (SARS, CoV2)

Tuberculosis (TB).

Introduction.

- Tuberculosis is a serious chronic pulmonary and systemic disease caused most often by M. tuberculosis. The source of transmission is humans with active tuberculosis who release mycobacteria present in sputum. According to the World Health Organization (WHO), tuberculosis is estimated to affect more than a billion individuals worldwide, with 8.7 million new cases and 1.4 million deaths each year.
- Tuberculosis flourishes wherever there is poverty, crowding, and chronic debilitating illness. In the United States, tuberculosis is mainly a disease of older adults, immigrants from high-burden countries, racial and ethnic minorities, and people with AIDS. Certain disease states also increase the risk: diabetes mellitus, Hodgkin lymphoma, chronic lung disease (particularly silicosis), chronic renal failure, malnutrition, alcoholism, and immunosuppression.

Etiopathogenesis.

Pathogenesis of tuberculosis is complex and involves several stages.

- Transmission—TB is primarily transmitted through inhalation of respiratory droplets from an infected individual. The bacteria can survive in aerosols for several hours and can be spread in crowded and poorly ventilated areas.
- Infection—When TB bacteria are inhaled, they can penetrate the alveolar spaces of the lungs and are taken up by alveolar macrophages, which are the first line of defence against infection. The bacteria then replicate inside the macrophages, leading to the formation of granulomas, which are collections of immune cells and bacteria.
- Latent tuberculosis infection—In some cases, the immune response is able to contain the infection, and the bacteria remain dormant within the granulomas. This is known as latent tuberculosis infection (LTBI), which is asymptomatic and non-transmissible.
- Reactivation tuberculosis disease—In some individuals, the bacteria can reactivate and cause disease, which is characterized by symptoms such as cough, fever, night sweats, and weight loss. This can occur when the immune system is compromised, such as in HIV infection, or when the individual is exposed to risk factors such as smoking or malnutrition.

Clinical manifestations.

- Chronic cough.
- Sputum production.
- Appetite loss.
- Weight loss.
- Fever.
- Night sweats.

Pharmacological managements

First line of treatment— isoniazid, rifampin, ethambutol, pyrazinamide, streptomycin.

Second line of treatment— ofloxacin, amikacin, moxifloxacin, ethionamide.

Third line of treatment— Linezolid, amoxicillin, azithromycin etc.

Non-pharmacological managements.

- Follow the all precaution (wearing the mask, sterilize the hand etc.) prior to any activities.
- Take the nutritious diet and make diet chart as per the instruction by the physician.
- Regular check-up and advice of physician is most important factor for the treatment of tuberculosis.
- Follow the daily routine(sleep and awake) and try to practice regular yoga and pranayama.

Pneumonia

Introduction.

- Bacteria like *Streptococcus pneumoniae* and *Haemophilus influenzae* are responsible for the disease pneumonia in humans which infects the alveoli (air filled sacs) of the lungs. As a result of the infection, the alveoli get filled with fluid leading to severe problems in respiration.
- Factors that can increase the risk of developing pneumonia include age, smoking, underlying lung disease, immunodeficiency, and exposure to environmental toxins.

Etiopathogenesis.

The pathogenesis of pneumonia depends on the specific pathogen involved, but in general, it involves the following steps.

- Entry of pathogen— The pathogen enters the body through inhalation, aspiration, or hematogenous spread from another site of infection.
- Adherence and colonization— The pathogen adheres to the surface of the respiratory epithelium and colonizes the lung tissue. This can occur through various mechanisms, such as binding to specific receptors on the host cells or using secreted enzymes to break down host tissue.
- Invasion and inflammation— The pathogen invades the host cells and triggers an inflammatory response, which leads to damage of the lung tissue and production of exudate. The exudate can fill the air sacs of the lungs, leading to difficulty breathing and decreased oxygen exchange.
- Clearance and resolution— The immune system responds to the infection and attempts to clear the pathogen. This can involve activation of phagocytic cells, such as macrophages and neutrophils, and production of antibodies to neutralize the pathogen. In most cases, the infection resolves

without complications, although in severe cases, complications such as sepsis, respiratory failure, and lung abscess can occur.

Clinical manifestations.

- Shortness of breath.
- Chill and sweating
- Fever.
- Productive cough.
- Pleuritic chest pain.
- Hypoxemia.
- Fatigue.
- Tachypnoea.

Pharmacological managements.

- Antibiotics- Azithromycin, moxifloxacin, ceftriaxone, cefepime, tazobactam, vancomycin, metronidazole, trimethoprim.

Non-pharmacological managements.

- Follow the all precaution (wearing the mask, sterilize the hand etc.) prior to any activities.
- Take the nutritious diet and make diet chart as per the instruction by the physician.
- Follow the daily routine (sleep and awake) and try to practice regular yoga and pranayama.

Urinary tract infections

Introduction.

- UTIs stands for Urinary Tract Infections, which is an infection that affects any part of the urinary system, including the bladder, ureters, kidneys, and urethra. UTIs are most commonly caused by bacteria, such as *Escherichia coli* (E. coli), which is commonly found in the gastrointestinal tract.

Etiopathogenesis— It depends on several factors, including host factors, bacterial virulence factors, and anatomical and physiological factors.

Host factors— Factors that impair the immune response, such as immunosuppressive medications, HIV infection, or diabetes, can increase the risk of UTI. In addition, female anatomy (shorter urethra and closer proximity of urethral opening to the anus) makes women more prone to UTIs.

Bacterial virulence factors—Bacteria can produce virulence factors that help them colonize and infect the urinary tract. Examples of virulence factors include adhesins, which allow bacteria to adhere to host cells, and toxins, which damage host cells and promote bacterial survival.

Anatomical and physiological factors: The urinary tract is normally sterile, but certain anatomical and physiological factors can increase the risk of infection. These include obstructions to urine flow, such as kidney stones or an enlarged prostate, which can prevent complete emptying of the bladder, and urinary catheterization, which can introduce bacteria into the urinary tract.

Clinical manifestations.

- A strong, persistent urge to urinate.
- A burning sensation when urinating.
- Passing frequent, small amounts of urine.
- Cloudy or strong-smelling urine.
- Pain or discomfort in the lower abdomen or back.
- Fatigue or feverishness.

Pharmacological managements.

- Trimethoprim and sulfamethoxazole, Fosfomycin, nitrofurantoin, Cephalexin, Ceftriaxone, ofloxacin, ciprofloxacin, norfloxacin, amoxycillin etc.

Non-pharmacological managements.

- Regular hygiene and cleaning are the most important measure to prevent the UTIs.
- Do the sexual activity by using of the proper protections.
- During the menstruation use the sanitizing sanitary pad.

Hepatitis

Introduction.

Hepatitis is an inflammation of the liver that can be caused by a variety of factors, including viral infections, alcohol consumption, and autoimmune disorders.it is classified as—

Hepatitis A—It is typically transmitted through contaminated food or water, and symptoms include fatigue, nausea, vomiting, and jaundice. Most people recover within a few weeks without specific treatment.

Hepatitis B and C— These transmitted through blood and bodily fluids, and can lead to chronic infection, cirrhosis, and liver cancer. These types of hepatitis can be asymptomatic for years, and people may not realize they have the infection until liver damage has already occurred.

Hepatitis D— It is a rare form of hepatitis that only occurs in people who are already infected with hepatitis B. It can lead to severe liver damage and cirrhosis.

Etiopathogenesis.

The etiopathogenesis (causes and mechanisms) of hepatitis varies depending on the specific type of hepatitis. Common causes are-

1. Hepatitis A— Hepatitis A virus is a non-enveloped RNA virus that is primarily transmitted through the faecal-oral route, usually by ingestion of contaminated food or water. Once inside the body, HAV replicates in the liver and causes inflammation, which can lead to liver damage.
2. Hepatitis B— Hepatitis B virus is a partially double-stranded DNA virus that is transmitted through blood and bodily fluids. HBV enters liver cells and uses its own reverse transcriptase enzyme to create a DNA copy of its genome. This DNA can then integrate into the host cell's DNA, leading to chronic infection.
3. Hepatitis C: Hepatitis C virus is a single-stranded RNA virus that is also transmitted through blood and bodily fluids. HCV enters liver cells and replicates using host cell machinery. Like HBV, HCV can lead to chronic infection and liver damage.
4. Hepatitis D: Hepatitis D virus is a small, enveloped RNA virus that can only replicate in the presence of HBV.

Clinical manifestations.

- Jaundice.
- Weight loss.
- Lethargy.
- Hyporexia.
- Abdominal distension.
- Emesis/diarrhoea.
- Hepatoencephalopathy.
- Renal insufficiency haematuria.
- Ascites.

Pharmacological managements.

For hepatitis B- Interferon alpha, lamivudine, telbivudine, adenofovir, tenofovir, emtricitabine.

For hepatitis C- Ribavirin, boceprevir, imeprevir, sofosbuvir.

Non-pharmacological managements.

- Prevention of hepatitis involves good hygiene practices, such as hand-washing and safe food preparation, vaccination (for hepatitis A and B), and avoiding high-risk behaviours such as unprotected sex and sharing needles.
- Early diagnosis and treatment of hepatitis is important to prevent long-term liver damage and complications.

Gonorrhoea.

Introduction.

Gonorrhoea, also known as "the clap," is a sexually transmitted infection caused by the bacterium *Neisseria gonorrhoeae*. It can affect both men and women, and is spread through unprotected vaginal, anal, or oral sex with an infected person.

Etiopathogenesis.

- Gonorrhoea is primarily transmitted through sexual contact, including vaginal, anal, and oral sex with an infected person. The bacterium can enter the body through the mucous membranes of the genitals, rectum, or throat.

- When bacteria enter inside then uses pili and outer membrane proteins to adhere to and invade the epithelial cells lining the mucous membranes. This process involves binding to specific host cell receptors and activating bacterial enzymes that allow the bacterium to penetrate the host cell.
- The invasion of host cells bacteria triggers an inflammatory response, which can cause the symptoms of gonorrhoea.

Clinical manifestations.

- Painful or burning sensation during urination
- Increased vaginal or penile discharge
- Painful bowel movements or rectal itching
- Sore throat or swollen lymph nodes in the neck
- Painful or swollen testicles in men

Pharmacological managements.

- Penicillin G, benzathine P, doxycycline, cefixime, ceftriaxone, azithromycin.

Non-pharmacological managements.

- Prevention of gonorrhoea involves practicing safe sex, including using condoms correctly and consistently, limiting the number of sexual partners, and getting regular STI testing.
- Early diagnosis and treatment of gonorrhoea are important for preventing complications and reducing the risk of transmission.

Syphilis

Introduction.

Syphilis is a sexually transmitted infection caused by the bacterium *Treponema pallidum*. It can affect both men and women and is spread through vaginal, anal, or oral sex with an infected person. On the appearance of symptoms, it is categorized as-

1. Primary syphilis— Appearance of a painless sore, known as a chancre, at the site of infection. The sore may be on the genitals, anus, or mouth and typically lasts 3-6 weeks before disappearing.

2. Secondary syphilis— This characterized by a widespread rash that can appear all over the body, including the palms of the hands and soles of the feet. Other symptoms may include fever, swollen lymph nodes, sore throat, and fatigue.
3. Latent syphilis: This stage has no visible symptoms, but the infection persists and can be detected through blood tests.
4. Tertiary syphilis: This stage can occur years after the initial infection and can cause serious complications such as damage to the brain, nerves, eyes, heart, blood vessels, liver, and bones.

Etiopathogenesis.

- Syphilis is primarily transmitted through sexual contact, including vaginal, anal, and oral sex with an infected person. The bacterium can also be transmitted from a mother to her baby during pregnancy or childbirth.
- *Treponema pallidum* can enter the bloodstream and invade various tissues and organs. The bacterium uses its outer membrane proteins to adhere to and penetrate host cells, where it can evade the immune system and replicate.
- The invasion of host cells triggers the inflammatory response, which can cause the symptoms of syphilis such as the appearance of a chancre or rash.
- In some cases, untreated syphilis can progress to the tertiary stage, which can cause serious complications such as damage to the brain, nerves, eyes, heart, blood vessels, liver, and bones.

Clinical manifestations.

- Painful or burning sensation during urination
- Increased vaginal or penile discharge
- Painful bowel movements or rectal itching
- Sore throat or swollen lymph nodes in the neck
- Painful or swollen testicles in men

Pharmacological managements.

- Penicillin G, benzathine P, doxycycline, cefixime, ceftriaxone, azithromycin.

Non-pharmacological managements.

- Prevention of syphilis involves practicing safe sex, including using condoms correctly and consistently, limiting the number of sexual partners, and getting regular STI testing.
- Early diagnosis and treatment of syphilis are important for preventing complications and reducing the risk of transmission.

Malaria

Introduction.

- Plasmodium, a tiny protozoan is responsible for this disease. Different species of Plasmodium (P. vivax, P. malaria and P. falciparum) are responsible for different types of malaria. Of these, malignant malaria caused by Plasmodium falciparum is the most serious one and can even be fatal.
- It is transmitted in humans through the bites of infected female Anopheles mosquitoes (vector/transmitting agent). It is interesting to note that the malarial parasite requires two hosts – human and mosquitoes.

Etiopathogenesis.

- Plasmodium enters the human body as sporozoites (infectious form) through the bite of infected female Anopheles mosquito.
- The parasites initially multiply within the liver cells and then attack the red blood cells (RBCs) resulting in their rupture.
- The rupture of RBCs is associated with release of a toxic substance, hemozoin, which is responsible for the chill and high fever recurring every three to four days.
- When a female Anopheles mosquito bites an infected person, these parasites enter the mosquito's body and undergo further development. The parasites multiply within them to form sporozoites that are stored in their salivary glands.
- When these mosquitoes bite a human, the sporozoites are introduced into his/ her body, thereby initiating the events.

Clinical manifestations.

- Fever and headache.
- Fatigue and pain.
- Chill and sweating.
- Nausea and vomiting.
- Spleen enlargement.
- Kidney disfunction.

Pharmacological managements.

- Atovaquone-proguanil (Malarone)
- Quinine sulphate with doxycycline.
- Primaquine phosphate.

Non-pharmacological managements.

- Anopheles vectors are grown on the dirty place so, cleaning is very important.
- Follow the guidelines, release by the government.
- Take the balance diet and regular practice of yoga and exercise is very important.
- Use the mosquito net and mosquito repellent.

HIV

Introduction.

- HIV, or human immunodeficiency virus, is a virus that attacks the immune system and can lead to acquired immunodeficiency syndrome (AIDS). A widely used diagnostic test for AIDS is enzyme linked immuno-sorbent assay (ELISA).
- AIDS was first reported in 1981 and in the last twenty-five years or so, it has spread all over the world killing more than 25 million persons.
- AIDS is caused by the Human Immune deficiency Virus (HIV), a member of a group of viruses called retrovirus, which have an envelope enclosing the RNA genome. Transmission of HIV-infection generally occurs by

➢ Sexual contact with infected person.
➢ By transfusion of contaminated blood and blood products.
➢ By sharing infected needles as in the case of intravenous drug abusers.

- From infected mother to her child through placenta.

Etiopathogenesis— Infection spread in several stages.

- First pathogen enters in body through any route mention above then. The virus enters into macrophages where RNA genome of the virus replicates to form viral DNA with the help of the enzyme reverse transcriptase.
- This viral DNA gets incorporated into host cell's DNA and directs the infected cells to produce virus particles.
- The macrophages continue to produce virus and, in this way, acts like a HIV factory. Simultaneously, HIV enters into helper T-lymphocytes (TH), replicates and produce progeny viruses.
- The progeny viruses released in the blood attack other helper T-lymphocytes. This is repeated leading to a progressive decrease in the number of helper-T lymphocytes in the body of the infected person. Due to decrease in the number of helper T lymphocytes, the person starts suffering from lots of infections (mycobacterium, viruses, fungi and even parasites like Toxoplasma).

Clinical manifestations.

- Fever and headache.
- Chills.
- Muscles aches and pains.
- Joint pain and fatigue.
- Swollen, lymph nodes mainly on the neck.
- Mouth ulcers and sore throat.
- Night sweat.
- Others infection also appears like skin disease etc.

Pharmacological managements.

- Abacavir, Emtricitabine, Lamivudine, Stavudine, Tenofovir, Zidovudine

Non-pharmacological managements.

- AIDS has no cure; prevention is the best option.
- Follow the all precaution (wearing the mask, sterilize the hand etc.) prior to any activities.
- Take the nutritious diet and make diet chart as per the instruction by the physician.

- Regular check-up and advice of physician is most important factor for the treatment of HIV.
- Follow the daily routine (sleep and awake) and try to practice regular yoga and pranayama.

Opportunistic infections.

Introduction.

- Opportunistic infections are infections that occur in people with weakened immune systems, such as those with HIV/AIDS, cancer, or who are taking immunosuppressive medications.
- These infections are caused by organisms that are normally harmless or even beneficial to healthy individuals, but can cause disease in people with compromised immune systems. Some common examples are-
  - **Tuberculosis.**
  - **Candidiasis.**
  - **Pneumocystis pneumonia.**
  - **Cytomegalovirus (CMV) infection.**
  - **Toxoplasmosis.**
  - **Cryptococcal meningitis.**
  - **Herpes simplex virus (HSV) infection.**

Prevention and treatments.

- Preventing opportunistic infections involves managing the underlying condition that is weakening the immune system and taking steps to prevent exposure to the infectious organisms. This may include taking antifungal, antiviral, or antibacterial medications, practicing good hygiene, and avoiding contact with people who are sick.

Viral Infections (SARS-CoV)

Introduction.

SARS, or severe acute respiratory syndrome, is a viral respiratory illness caused by the SARS coronavirus (SARS-CoV). The SARS outbreak occurred in 2002-2003, mainly in Asia, but cases were also reported in other parts of the world.

Etiopathogenesis.

- The SARS coronavirus (SARS-CoV) is primarily transmitted through close person-to-person contact, such as respiratory droplets produced when an infected person coughs or sneezes. The virus can also be transmitted through contact with contaminated surfaces or objects.
- Then it binds to angiotensin-converting enzyme 2 (ACE2) receptors on the surface of cells in the respiratory tract. This allows the virus to enter and infect the cells.
- The virus replicates and produces more SARS-CoV particles. This process leads to damage and inflammation in the respiratory tract, which can result in pneumonia and other respiratory symptoms.

Clinical manifestations.

- Fever, weariness, headache, stroke.
- Dyspnoea, rhinorrhoea, anosmia, ageusia.
- Dry cough, sputum production, sore throats.
- Acute respiratory distress syndrome.
- Lymphopenia.
- Acute cardiac injury.
- Pneumonia.
- Poor appetite, diarrhoea.
- Multiple organ failure.

Pharmacological managements.

- Remdesivir, Favipiravir, Hydroxychloroquine, Azithromycin, Lopinavir/Ritonavir, Nafamostat mesylate.

Non-pharmacological managements.

- Public health measures such as vaccination, social distancing, and wearing masks, which remain the most effective means of preventing the spread of viral disease.
- Take the balance diet and regular practice of yoga and exercise is very important.

Viral Infections (SARS-CoV2)

Introduction.

- CoV2, or SARS-CoV-2, is the virus responsible for the COVID-19 pandemic. COVID-19 is a respiratory illness caused by the SARS-CoV-2 virus, and it was first identified in Wuhan, China in December 2019
- Overall, the pathogenesis of SARS-CoV-2 is similar to that of other respiratory viruses, but the severity of COVID-19 can be influenced by a variety of factors, including the individual's age, health status, and immune response.

Etiopathogenesis.

- SARS-CoV-2 is primarily transmitted through close person-to-person contact, such as respiratory droplets produced when an infected person coughs or sneezes. The virus can also be transmitted through contact with contaminated surfaces or objects.
- Then it binds to angiotensin-converting enzyme 2 (ACE2) receptors on the surface of cells in the respiratory tract. This allows the virus to enter and infect the cells.
- The virus replicates and produces more SARS-CoV-2 particles. This process leads to damage and inflammation in the respiratory tract, which can result in pneumonia and other respiratory symptoms.

Clinical manifestations.

- Fever, weariness, headache, stroke.
- Dyspnoea, rhinorrhoea, anosmia, ageusia.
- Dry cough, sputum production, sore throats.
- Acute respiratory distress syndrome.
- Lymphopenia.
- Acute cardiac injury.
- Pneumonia.
- Poor appetite, diarrhoea.
- Multiple organ failure.

Pharmacological managements.

- Remdesivir, Favipiravir, Hydroxychloroquine, Azithromycin, Lopinavir/Ritonavir, Nafamostat mesylate.

Non-pharmacological managements.

- Public health measures such as vaccination, social distancing, and wearing masks, which remain the most effective means of preventing the spread of viral disease.
- Take the balance diet and regular practice of yoga and exercise is very important.

Chapter-2 (h)

Musculoskeletal disorders

Musculoskeletal disorders

- Rheumatoid arthritis
- Osteoarthritis

MUSCULOSKELETAL DISORDERS.

Introduction— Musculoskeletal disorders comprise diverse conditions affecting bones, joints, muscles, and connective tissues. These disorders may result in pain and loss of function. It is happening due to the lack of knowledge and irregular/improper diet plan. Now a day, it is big challenges for the modern society and pharmaceutical science.

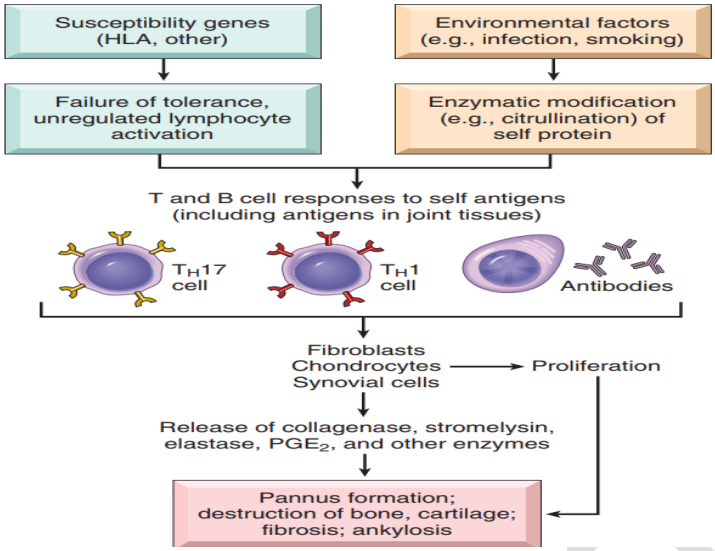
Rheumatoid arthritis

Definition.

Rheumatoid arthritis is a chronic inflammatory disorder of autoimmune origin that may affect many tissues and organs but principally attacks the joints, producing a nonsuppurative proliferative and inflammatory synovitis. Rheumatoid arthritis often progresses to destruction of the articular cartilage and ankylosis of the joints. Extraarticular lesions may involve skin, heart, blood vessels and lungs and, therefore, the clinical manifestations can resemble other systemic autoimmune disorders also.

Etiopathogenesis—

- Immunological factors—As in other autoimmune diseases, genetic predisposition and environmental factors contribute to the development, progression, and chronicity of the disease. The pathologic changes are mediated by antibodies against self-antigens and cytokine-mediated inflammation, predominantly secreted by CD4+ T-cells.



Major processes involved in the pathogenesis of rheumatoid arthritis

- Hormonal factors— Sex hormones may play a role in rheumatoid arthritis, as evidenced by the disproportionate number of females with this disease, its amelioration during pregnancy, its recurrence in the early postpartum period, and its reduced incidence in women using oral contraceptives. Hyperprolactinemia may be a risk factor for rheumatoid arthritis.
- Others factors— Smoking is the most significant non-genetic risk with rheumatoid arthritis being up to three times more common in smokers than non-smokers, particularly in men. Vitamin D deficiency is more common in patients with rheumatoid arthritis also.

Clinical manifestations—

- Systemic lupus erythematosus or scleroderma.
- Stiffness and pain in the joints.
- Swelling of joint.
- Redness and warmth of joints.
- Muscle destruction and fatigue.
- Loss of appetite, which can lead to weight loss.
- Fever and neurological disorders.

Pharmacological management—

- Adjuvant drugs— Diacerein, auranofin, hyaluronate sodium, gold sodium thiomalate, aurothioglucose, prednisolone.
- DMARDs (Disease-modifying antirheumatic drugs)—
  1. Biological agents—
     - TNF-α Inhibitor—Etanercept, infliximab, tasonermin, afelimomab, abatacept.
     - IL-1 antagonist— Tocilizumab, sarilumab.
  2. Non-biological agents— Chloroquine, hydroxychloroquine, methotrexate, azathioprine, cyclosporine, sulfasalazine.
- Other drugs— bucillamine, upadacitinib.

Non-Pharmacological management—

- Make the diet charts and follow accordingly and avoid the fattier and lipids contents in the diet.
- Change the lifestyle and apply the home remedies means replace the allopathic medicine with ayurvedic medicine (because of less side effects)
- Regular practice of yoga exercise and other physical exercise
- During more pain condition rest is required and follow the heat and cold for managing pain (Both heat and cold can relieve pain in joint. Heat also relieves stiffness, and cold can relieve muscle spasms and pain).
- Avoid the smoking and alcoholism.

Osteoarthritis

Definition.

Osteoarthritis, also called degenerative joint disease, is characterized by degeneration of articular (hyaline) cartilage that results in structural and functional failure of synovial joints. The term osteoarthritis implies an inflammatory disease, it is considered to be an intrinsic disease of cartilage in which chondrocytes respond to biochemical and mechanical stresses resulting in breakdown of the matrix. **The knees and hands are more commonly affected in women and the hips in men.**

It is understood by two stages-

- Primary osteoarthritis/idiopathic— In most instances osteoarthritis appears insidiously, without apparent initiating cause, as an aging phenomenon is called primary osteoarthritis. In these cases, the disease is usually oligoarticular (affects few joints) but may be generalized.
- Secondary osteoarthritis— osteoarthritis appears in younger individuals with some predisposing condition, such as joint deformity, a previous joint injury, or an underlying systemic disease such as diabetes, hemochromatosis, or marked obesity that places joints at risk. In these settings the disease is called secondary osteoarthritis.

Etiopathogenesis— The articular cartilage responsible for frictionless movement of the joint and also provide resistance against tension and compression, from type II collagen and proteoglycans, respectively, both synthesized by chondrocytes. Any deformity or pathological changes occurs in the chondrocyte and matrix leads to the osteoarthritis.it is understood by three stages.

1. Chondrocyte injury, related to genetic and biochemical factors.
2. Early osteoarthritis, in which chondrocytes proliferate and secrete inflammatory mediators, collagens, proteoglycans, and proteases, which act together to remodel the cartilaginous matrix and initiate secondary inflammatory changes in the synovium and subchondral bone.
3. Late osteoarthritis, in which repetitive injury and chronic inflammation lead to chondrocyte drop out, marked loss of cartilage, and extensive subchondral bone changes.

Environmental and genetic influences contribute to the pathogenesis of OA. The major environmental factors relate to aging and biomechanical stress.

Clinical manifestations—

- Radicular pain
- Muscle spasms, muscle atrophy/weakness.
- Joint tenderness and stiffness.
- Loss of flexibility and
- Neurologic deficits.

Pharmacological management—

- NSAIDs— Ibuprofen, naproxen, indomethacin, sulindac, fenoprofen, piroxicam, diclofenac, tramadol.
- Corticosteroids— Prednisone, prednisolone.

Non-Pharmacological management—

- Make the diet charts and follow accordingly and avoid the fattier and lipids contents in the diet.
- Change the lifestyle and apply the home remedies means replace the allopathic medicine with ayurvedic medicine (because of less side effects)
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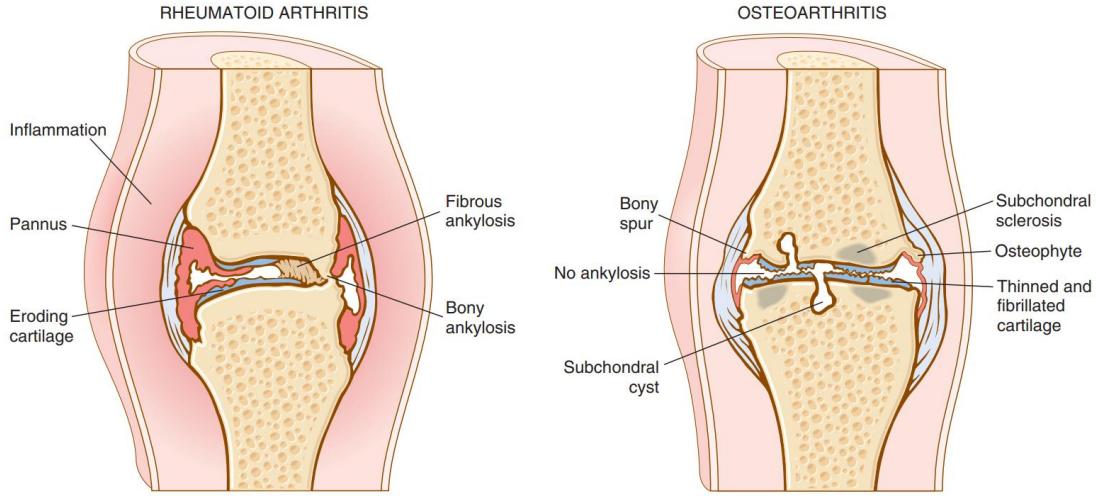

Morphological comparison.

Chapter-2 (i)
Dermatology

Dermatology

- Psoriasis
- Scabies
- Eczema

Dermatology

Dermatology is the branch of medical science in which we study about the skin abnormality appears due to any infections or allergic conditions. In skin disease we discuss many conditions like-

- Melanocyte nevus (pigmented nevus).
- Actinic keratosis.
- Ichthyosis.
- Urticaria.
- Acute eczematous dermatitis.
- Psoriasis.
- Scabies.
- Verrucae (warts).

Psoriasis.

Introduction.

Psoriasis is a common chronic inflammatory dermatosis, persistent or relapsing, scaling skin condition. Individual lesions are distinctive in their classic form sharply marginated and erythematous and surmounted by silvery scales. Most patients with psoriasis have a limited number of fixed plaques, but there is great variation in clinical presentation. Persons of all ages may develop the disease.

Several lines of evidence have established that genetic factors contribute to the development of psoriasis. Approximately 15% of the patients with psoriasis have associated arthritis. It can affect any joint in the body and may be

symmetrical or affect one side only. In addition, psoriasis may also be associated with myopathy, enteropathy, and AIDS.

Etiopathogenesis.

- Psoriasis results from interactions of genetic and environmental factors. As in the case of many autoimmune diseases it is linked to genes within the HLA locus. There is a strong association with HLA-C.
- Lymphocytes also produce growth factors for keratinocytes/epidermopoiesis (epidermal proliferation) that may contribute to epidermal thickening.
- In other predisposed individuals, a number of environmental factors, including infection, physical injury, stress, and drugs, can serve as triggers for the development of psoriasis.

Clinical manifestations.

- Psoriatic arthritis.
- Patches covered with silver white flakes.
- Raised and thick skin.
- Dry, swallow and inflamed patches.
- Pain, itching, and burning sensation.
- Red spots on the body.

Pharmacological managements.

- **Anti-allergic drugs—** Ex- Cetirizine, levocetirizine, fexofenadine, diphenhydramine, chlorphenamine, pheniramine.
- **Antifungal agents—** Terbinafine, flucytosine, ketoconazole, miconazole, luliconazole, clotrimazole, salicylic acid, tolnaftate, fluconazole.
- **Antibiotics—** ofloxacin, moxifloxacin, clarithromycin.
- **NSAIDs—** Ex- Naproxen, ibuprofen, piroxicam, diclofenac.
- **Immunosuppressants**— Ex- betamethasone, dexamethasone, deflazacort, cortisone, prednisolone.

Non-pharmacological managements.

- Diet is an important factor in the skin disorder because some food causes the skin allergy and leads to severe conditions.
- Self-awareness is very important because we need to know which substance cause allergy (allergens like dust, smoke, pollen, food etc).

- Regular hygienic activity is very important factors to overcome the skin disorder conditions. (Like regular bathing, wearing dry and clean clothes etc).
- Regular yoga and physical activity also overcome the disease by maintain the immune system in proper condition.

Scabies

Introduction.

Scabies is an infectious disease of the skin by the human itch mite. The microscopic scabies mite burrows into the upper layer of the skin where it lives and lays its eggs. The most common symptoms of scabies are intense itching and a pimple-like skin rash. The scabies mite usually is spread by direct, prolonged, skin-to-skin contact with a person who has scabies.

Scabies is one of the commonest dermatological conditions, accounting for a substantial proportion of skin disease in developing countries. Globally, it is estimated to affect more than 200 million people at any time, although further efforts are needed to assess this burden.

Etiopathogenesis.

Human scabies is a parasitic infestation caused by *Sarcoptes scabiei var hominis*. The microscopic mite burrows into the skin and lays eggs, eventually triggering a host immune response that leads to intense itching and rash. Scabies infestation may be complicated by bacterial infection, leading to the development of skin sores that, in turn, may lead to the development of more serious consequences such as septicaemia, heart disease and chronic kidney disease.

Clinical manifestations.

- Formation of plaques.
- Thickening of skin.
- Puffy and red eye lids.
- Hyperpigmentation of skin.
- Oozing of lesions.
- Formations of cracks.
- Itching, and burning sensation.

Pharmacological managements.

- Anti-allergic drugs— Ex- Cetirizine, levocetirizine, fexofenadine, diphenhydramine, chlorphenamine, pheniramine.
- Antifungal agents— Terbinafine, flucytosine, ketoconazole, miconazole, luliconazole, clotrimazole, salicylic acid, tolnaftate, fluconazole.

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Eczema

Introduction.

The Greek word eczema, meaning “to boil over,” vividly describes the appearance of acute eczematous dermatitis one of the most common skin disorders. Based on initiating factors, eczematous dermatitis can be subdivided into the following categories.

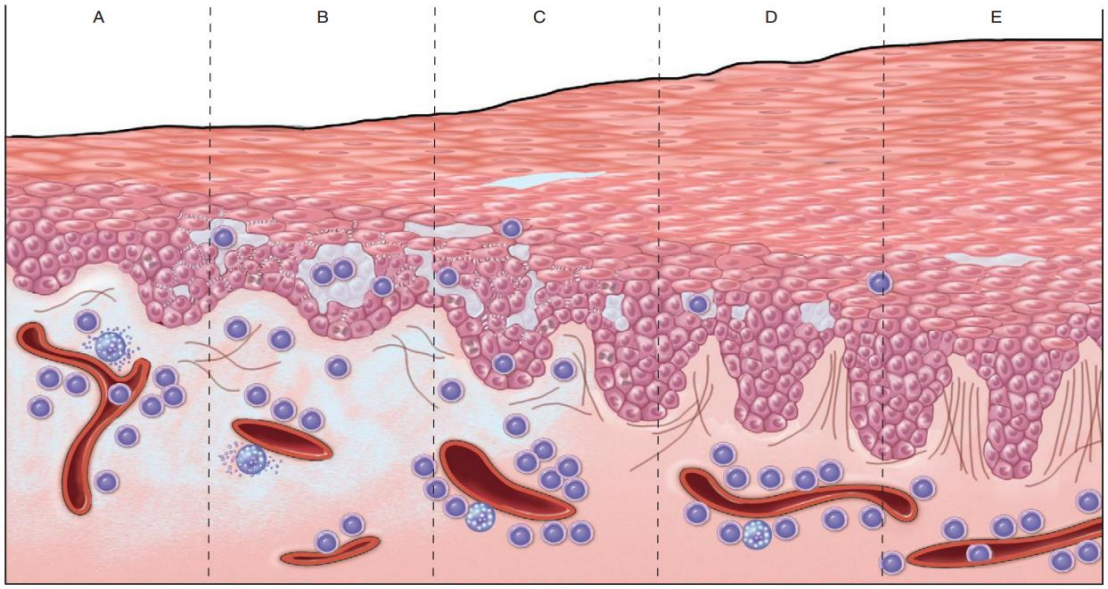
- Allergic contact dermatitis.
- Atopic dermatitis.
- Drug-related eczematous dermatitis.
- Photoeczematous dermatitis.
- Primary irritant dermatitis.

All types of eczematous dermatitis are characterized by red, papulovesicular, oozing, and crusted lesions that, if persistent, develop reactive acanthosis and hyperkeratosis that produce raised scaling plaques.

Etiopathogenesis.

Eczematous dermatitis typically results from T cell-mediated inflammatory reactions (type IV hypersensitivity). It is developed in 5 stages.

A. Initial dermal oedema and perivascular infiltration by inflammatory cells is followed within 24 to 48 hours.
B. Epidermal spongiosis and micro vesicle formation.
C. Abnormal scale, including parakeratosis, along with progressive acanthosis.
D. Hyperkeratosis.
E. Appear as the lesion becomes chronic.



Clinical manifestations.

- Itching, and burning sensation.
- Dryness of skin.
- Cutaneous reactivity.
- Chances of secondary or internal tissue infections.
- Eczematous areas with crusting.
- Macular erythema, papules.
- Lichenification and excoriation.

Pharmacological managements.

- Anti-allergic drugs— Ex- Cetirizine, levocetirizine, fexofenadine, diphenhydramine, chlorphenamine, pheniramine.
- Antifungal agents— Terbinafine, flucytosine, ketoconazole, miconazole, luliconazole, clotrimazole, salicylic acid, tolnaftate, fluconazole.

Non-pharmacological managements.

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Chapter-2 (j)

Psychiatric Disorders

(j) Psychiatric Disorders

- **Depression**
- **Anxiety**
- **Psychosis**

Psychiatric disorders.

Psychiatric disorder also called mental health disorders, refers to a wide range of mental health conditions — disorders that affect your mood, thinking and behaviour. It includes as-

- **Depression.**
- **Anxiety disorders.**
- **Schizophrenia.**
- **Eating disorders and**
- **Addictive behaviours**
- **Psychosis.**

Depression.

Introduction— It is not a disorder. It is just a imbalance condition of the brain activity of the individual leads to sadness or downswings in mood are normal reactions to life struggle, setbacks, and disappointments. Many people use the

word “depression” to explain of some kinds of feeling/emotion, but depression is much more than just sadness.

Some people describes the depression as “living in a black hole” or having a feeling of impending doom. However, some depressed people do not feel sad at all-they may feel angry, aggressive, and restless. Depression interferes our ability to work, study, eat, sleep, and have fun leads to helplessness, hopelessness, and worthlessness.

Etiopathogenesis.

Depression is varying on sex or age. It is describing as

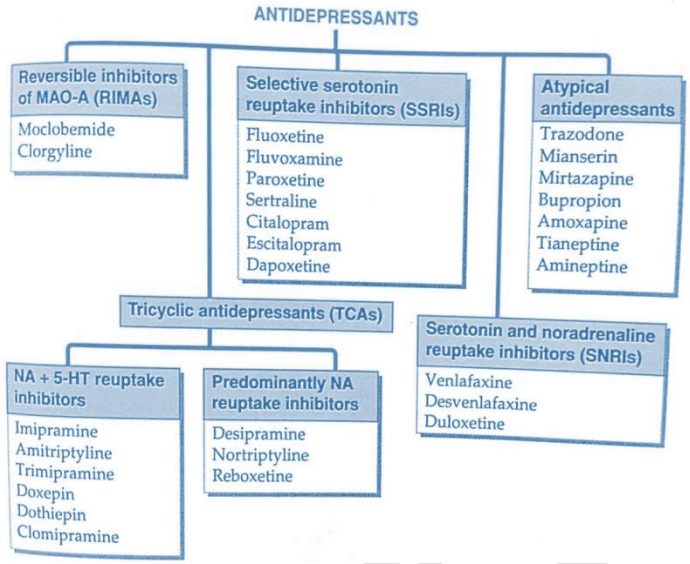
1. Depression in men— Depressed men are less likely than women to acknowledge feelings of self-loathing and hopelessness. Instead, they tend to complain about fatigue, irritability, sleep problems, and loss of interest in work and hobbies.
2. Depression women— Rate of depression in women are twice as high as they are in men. This is due in part to hormonal factors, particularly when it come to premenstrual syndrome (PMS), premenstrual dysphoric disorder (PMDD), postpartum depression, and perimenopausal depression.
3. Depression in teens— Major cause for depression in teens is sadness. A depressed teenager may be hostile, grumpy, or easily lose his or her temper.
4. Depression in older adults— older adults face bereavement, loss of independence, and health problem can lead to depression. It is associated with poor health, a high mortality rate, and an increased risk of suicide.
5. Postpartum depression— Postpartum depression, in contrast, is a longer lasting and more serious depression triggered, in part, by hormonal changes associated with having a baby. It develops soon after delivery, but any depression that occurs within six months of childbirth may be postpartum depression.

Clinical manifestations.

- Epilepsy (repeated, unpredictable seizures)
- Appetite or weight changes.
- Sleep changes (Insomnia or hypersomnia).
- Anger or irritability.
- Loss of strength, feeling fatigued, sluggish, and physically drained.
- Self-loathing.

- Reckless behaviour.
- Concentration problem.
- Unexplained aches and pains.

Pharmacological managements



Non-pharmacological managements.

- Follow the diet rules and pattern because any disturbance in the digestive activity leads to the mental manifestations.
- Visit the religious and graceful places and try to spending much time on that place.
- Multimedia, internet etc. are one among the cause of the psychiatric disorder, so try to use those such things with proper time and need.
- Avoid the overthinking and bad habits and try to always indulges with your own work, which makes you pleasant.
- Regular practice of yoga and meditation it is the most important factor for managing the psychiatric disorders.

Anxiety.

Introduction— The term anxiety has been used for decades to refer to thoughts and behaviours that were distressful in nature. But before they referred to these as “anxiety disorders”, they are called “neurosis” which means nervousness that was not based in fact. Anxiety can be described as a feeling of alarm or worry. It may be about something specific or it may be non-specific in nature. At certain level it improves our performance and allow people to avoid dangerous situations.

This normally lasts for a short period causing no impairment in social or occupational functioning. When this anxiety is prolonged and affects social or occupational functioning, it is abnormal and accounts for anxiety "disorder".

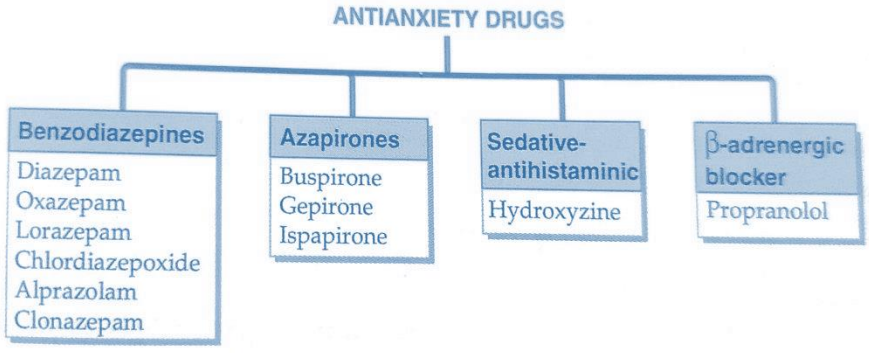
Etiopathogenesis.

- Anxiety is of a greater degree than just everyday worries and patients do mention that they are not able to control these worries. They are frequently accompanied with physical symptoms as well. These symptoms have to present for most days at least for several weeks at a time.
- Patients often experience a state of intense apprehension, uncertainty, and fear resulting from the anticipation of a threatening event or situation, often to a degree that normal physical and psychological functioning is disrupted. This may be precipitated due to several reasons which are generally easily identified by patient himself.

Clinical manifestations.

- Feelings that something undesirable.
- Dry mouth, swallowing difficulty, hoarseness.
- Rapid breathing and heartbeat, palpitations.
- Twitching or trembling.
- Muscle tension headaches backache.
- Dizziness or faintness.
- Difficulty in concentrating.
- Nausea, diarrhoea, weight loss.
- Memory problems and difficulty in concentrating.
- Sweating, fatigue, irritability.
- Sleeplessness and nightmare.

Pharmacological managements



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Psychosis.

Introduction— Psychosis and the specific diagnosis of schizophrenia represent a major psychiatric disorder (or cluster of disorders) in which a person's perceptions, thoughts, mood and behaviour are significantly altered.

Psychosis is a common and functionally disruptive symptom of many psychiatric, neurodevelopmental, neurologic, and medical conditions and an important target of evaluation and treatment in neurologic and psychiatric practice.

Etiopathogenesis.

A psychotic episode or disorder will result in the presence of one or more of the following five categories: delusions, hallucinations, disorganized thought, disorganized behaviour, negative symptoms. Some common causes similar to the anxiety but is also induced as-

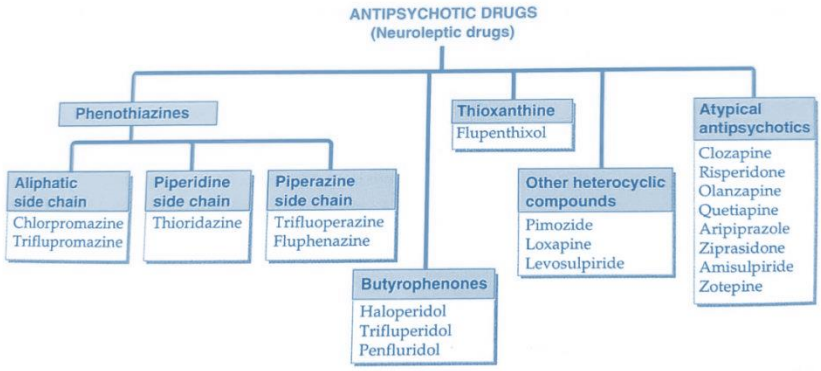
- Genetic/heredity.
- Chemical imbalances.

Clinical manifestations.

- Unusual and extremely slowed movements.
- Incoherent or disorganised speaking.
- Hallucinations, usually related to hearing voices or strange sounds.

- Delusions
- Isolating behaviour.
- Feeling suspicious paranoid or afraid.
- Not caring about their hygiene and appearance.
- Depression anxiety and suicidal thought.

Pharmacological managements



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Chapter-2 (k)| Ophthalmology

(k) Ophthalmology

- Conjunctivitis (bacterial and viral)
- Glaucoma

Ophthalmology

Conjunctivitis (bacterial and viral)

Introduction.

- The conjunctiva lining the interior of the eyelid, the palpebral conjunctiva, is tightly tethered to the tarsus and may respond to inflammation by being thrown into minute papillary folds as may occur in allergic conjunctivitis and bacterial conjunctivitis. The conjunctiva in the fornix is a pseudostratified columnar epithelium rich in goblet cells. The fornix also contains accessory lacrimal tissue, and the ductules of the main lacrimal gland pierce through the conjunctiva in the fornix superiorly and laterally.
- Many cases of bacterial or viral conjunctivitis cause redness and itching, but most heal without sequelae.

Etiopathogenesis.

- Conjunctivitis can be caused by a variety of factors including bacterial or viral infections, allergic reactions, and irritants such as smoke, dust, or chemicals.
- Bacterial conjunctivitis—It is a common eye infection that can occur in people of all ages, but it is most common in children. Bacterial conjunctivitis can be caused by various bacteria such as *Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae*, and others.
- Viral conjunctivitis—Viral conjunctivitis is typically caused by a group of viruses known as adenoviruses. Other viruses that can cause viral conjunctivitis include herpes *simplex virus, varicella-zoster virus, and picornaviruses*. The infection can spread through direct contact with contaminated surfaces, such as towels, doorknobs, or shared makeup.

Clinical manifestations.

- Redness of the eye.
- Itching or burning sensation.

- Excessive tearing.
- Discharge from the eye, which may be clear, yellow, or greenish.

Pharmacological managements.

- Antibiotics— Tobramycin, cotrimoxazole, sulfacetamide, bacitracin, erythromycin, ciprofloxacin, moxifloxacin, ofloxacin, gentamycin.
- Anti-allergic drugs— Chloropyramine, naphazoline, bepotastine.
- Antiviral— Ganciclor, Acyclovir.
- Analgesic—flurbiprofen, ketorolac, fluorometholone.
- Others— dexamethasone, prednisolone

Non-pharmacological managements.

- It is important to practice good hygiene, such as washing hands frequently, avoiding touching the eyes with hands, and avoiding sharing personal items such as towels or cosmetics.
- People who are infected with bacterial conjunctivitis should avoid touching their eyes and wash their hands frequently to prevent spreading the infection to others.
- Avoid the allergic causing place (dust or smoke).
- Clear airway of secretion and allows for allergen removal.

Glaucoma

Introduction.

The term glaucoma refers to a collection of diseases characterized by distinctive changes in the visual field and in the cup of the optic nerve. Most of the glaucoma are associated with elevated intraocular pressure, although some individuals with normal intraocular pressure may develop characteristic optic nerve and visual field changes (normal or low-tension glaucoma).

Etiopathogenesis—Pathophysiology of glaucoma is understood by the, formation and drainage of aqueous humor. It is two type-

1. Open-angle glaucoma— In open-angle glaucoma aqueous humor has complete physical access to the trabecular meshwork, and the elevation in intraocular pressure results from an increased resistance to aqueous outflow. It further classified as-
    - Primary open-angle glaucoma— Mutations in the myocilin (MYOC) gene have been associated with a subset of individuals with juvenile and adult primary open-angle glaucoma and mutations in optineurin

(OPTN) may also be responsible for a subset of adult patients with open angle glaucoma. Primary open-angle is most common form of glaucoma.

- Secondary open-angle glaucoma— Pseudoexfoliation glaucoma, perhaps the most common form of secondary open angle glaucoma, is associated with the deposition of fibrillar material of varying composition throughout the anterior segment.

2. Angle-closure glaucoma—In angle-closure, the peripheral zone of the iris adheres to the trabecular meshwork and physically impedes the egress of aqueous humor from the eye. It further classified as-
   - Primary angle-closure glaucoma— Primary angle-closure glaucoma typically develops in eyes with shallow anterior chambers, often found in individuals with hyperopia.
   - Secondary angle-closure glaucoma— Contraction of various types of pathologic membranes that form over the surface of the iris can draw the iris over the trabecular meshwork, occluding aqueous outflow. For example, chronic retinal ischemia.

Clinical manifestations.

- Blurred vision.
- Redness of the eye.
- Pain in the eyes and the eyebrows.
- High intraocular pressure.
- Headache, vomiting nausea.

Pharmacological managements.

- Beta- adrenergic blockers— timolol, betaxolol, levobunolol.
- Alpha-adrenergic agonists— dipivefrine, apraclonidine, brimonidine.
- Prostaglandin analogues— latanoprost, travoprost, bimatoprost.
- Carbonic anhydrase— acetazolamide, dorzolamide.
- Miotics— pilocarpine, physostigmine.

Non-pharmacological managements.

- Take the wholesome food/diet.
- Practice the exercise and yoga because it also helps in reducing the eye pressure.
- Avoid the polluted area because pollutants cause the irritation in eye.

Chapter-2 (I)

Anti-Microbial Resistance

Anti-microbial resistance

Resistance to antimicrobial agents has become a major source of morbidity and mortality worldwide. Its occurs due to the exposure of unspecific chemical or substance on to the microbes and leads to the physiological adaptation against the particular chemical or substance. It also occurs due to the repeatedly uses of the specific substances.

Antimicrobial agents can be divided into groups based on the mechanism of antimicrobial activity.

1. Agents that inhibit cell wall synthesis.
2. Depolarize the cell membrane.
3. Inhibit protein synthesis.
4. Inhibit nucleic acid synthesis.
5. Inhibit metabolic pathways in bacteria.

Antimicrobial are two types.

- Bacteriostatic. Chloramphenicol, macrolides, clindamycin, sulfa, trimethoprim, tetracyclines.
- Bactericidal. Aminoglycosides, beta-lactams, vancomycin, rifampin, metronidazole.

Factors contributing to antibiotic resistance.

1. Environmental factors— Due to the changes in the environmental condition, it modifies or effects the microbial growth and leads to the adaptation in the microbes against the particular environment condition.
    - Population and overcrowding.
    - Poor sanitation.
    - Ineffective infection control program.
    - Widespread use of antibiotics in animal husbandry and agriculture and as medicated cleansing products.

2. Drug related factors.
    - Fake and quality of drugs.
    - Soaring used is antibiotics.
    - OTC availability of antimicrobials.
    - Irrational fixed dose combination of antimicrobials.
3. Patient related factors.
    - Poverty.
    - Poor adhere of dosage regimens.
    - Lack of sanitation concept.
    - Lack of education.
    - Self-medication.
    - Misconception.
4. Physician/prescriber related factors—
    - Inappropriate use of available drugs.
    - Overuse of anti-microbial.
    - Inadequate dosing.
    - Lack of current knowledge and training.

Two types of antimicrobial resistance.

1. Intrinsic or natural— The most common bacterial mechanisms involved in intrinsic resistance are reduced permeability of the outer membrane and the naturel activity of efflux pumps.
    - It always expressed in the species.
2. Acquired— bacteria acquire any genetic material, transformation, transposition, and conjugation, mutations etc.

Mechanism of antimicrobial resistance—

- Limiting uptake of a drug— Certain bacteria modify their cell membrane porin channels, there by preventing the antimicrobials from entering into the cell. There are two main ways in which porin changes can limit drug uptake, a decrease in the number of porins present, and mutations that change the selectivity of the porin channel.
- Modification of a drug target—One mechanism of resistance to the beta-lactam drugs used almost exclusively by G+ bacteria is via alteration in the structure and number of PBPs (penicillin-binding proteins). Change in number decrease in binding ability.

- Inactivation of a drug— it is done by two mechanisms
  1. Actual degradation of the drug.
  2. Transfer of a chemical group to the drug.
- Active efflux of a drug— Bacteria posses chromosomally encoded genes for efflux pumps. Some are expressed constitutively, and others are induced or overexpressed under certain environmental stimuli or when a suitable substrate is present.

Chapter-2 (m)

Women's Health

(m) Women's Health

- **Polycystic Ovary Syndrome**
- **Dysmenorrhea**
- **Premenstrual Syndrome**

Polycystic Ovary Syndrome:

Introduction.

- Polycystic ovarian syndrome (PCOS) is a complex endocrine disorder characterized by hyperandrogenism, menstrual abnormalities, polycystic ovaries, chronic anovulation, and decreased fertility. Formerly called Stein Leventhal syndrome, it affects 6-10% of reproductive age women worldwide. It is also associated with obesity, type 2 diabetes, and premature atherosclerosis, all of which may be indicative of an underlying metabolic disorder.

Etiopathogenesis.

- PCOS remains incompletely understood. It is marked by a dysregulation of enzymes involved in androgen biosynthesis and excessive androgen production, which is considered to be a central feature of this disorder. In addition, women with PCOS show insulin resistance and altered adipose tissue metabolism, which contribute to the development of both diabetes and obesity.
- The central morphologic abnormality of PCOS is numerous cystic follicles or follicle cysts that enlarge the ovaries. However, polycystic ovaries are detected in 20% to 30% of all women, so this finding is not

specific. In addition, due to an increase in free serum estrone levels, women with PCOS are at risk for endometrial hyperplasia and carcinoma.

Clinical manifestations.

- Obesity.
- Hyperinsulinemia.
- Ovarian cyst.
- Endocrine dysfunction.
- Irregular or missed period.
- Hyperandrogenism.
- Excessive body hair growth.
- Mood swing, fatigue, acne etc.

Pharmacological managements.

- Insulin sensitizing medications. Ex- metformin, pioglitazone etc.
- Androgen blocking medication. Ex- bicalutamide, nilutamide, leuprolide.
- Birth control pills to regulate menstrual cycle. ex- progesterone and norethindrone, levonorgestrel.
- Ovulation induction medications. Ex- clomiphene citrate.
- Others antibiotic and anti-acne drugs are also used.

Non-pharmacological managements.

- Any symptoms appear then consult with the gynaecologist, and change their life style (sleep and wake up pattern) as per the instruction.
- Diet pattern is very essential because it maintain the BMR and maintain the body weight.
- Behavioural changes like (anger, sadness, anxiety) also cause the hormonal balancing, so try to make happy and cheerful.
- Less consumption of tobacco, alcohol, and caffeine because it increases the production of androgens.
- Physical activity, yoga and meditation improve the body activity (Physically and mentally). It helps in reducing the stress and anger and maintain the hormonal level.

Dysmenorrhea.

Introduction.

- Dysmenorrhea term is defined as the menstruation with pain. Dysmenorrhea is pain, typically cramping in character and lower abdominal in location, occurring in the days just before and during menstrual flow. Dysmenorrhea can occur as a primary disorder in the absence of identifiable pelvic disease, or it may be secondary to an underlying pelvic disease such as endometriosis or leiomyomas

Etiopathogenesis.

- Primary dysmenorrhea is thought to be due to disordered prostaglandin production by the secretory endometrium. Prostaglandin F2α (PGF2α) stimulates myometrial contractions of the nonpregnant uterus, whereas prostaglandins of the E series tend to inhibit its contraction. It appears that patients with severe dysmenorrhea generally experience excessive PGF2α production rather than increased sensitivity to this prostaglandin. Unabated contractions of the myometrium result in uterine muscle ischemia, which stimulates the uterine pain fibres of the autonomic nervous system.
- The secondary causes of dysmenorrhea are endometriosis, a disorder in which extrauterine implants of ectopic endometrial tissue respond cyclically to estrogen and progesterone production (see Table 22–5). This is a common disorder, affecting 10–25% of women of reproductive age. The presenting symptoms of patients with endometriosis can range from pain and cramping during menstruation to adhesions with bowel obstruction in severe cases.

Clinical manifestations.

- Sweating.
- Weakness, Fatigue.
- Insomnia.
- Nausea, Vomiting, Diarrhoea.
- Back pain.
- Headache, migraine and tension headaches.
- Dizziness, and syncope.

- Including a sensation of bloating, weight gain, oedema of the hands and feet, breast tenderness, acne, anxiety, aggression, mood irritability, food cravings, and change in libido.

Pharmacological managements.

For reducing the pain and any infection drugs used as-

- NSAIDs. Ex- Ibuprofen, mefenamic acid, naproxen, celecoxib, nimesulide.
- Oral contraceptive. Ex- Norethindrone, levonorgestrel.

Non-pharmacological managements.

- Any symptoms appear then consult with the gynaecologist, and change their life style (sleep and wake up pattern) as per the instruction.
- Diet pattern is very essential because it maintain the BMR and maintain the body weight.
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Premenstrual Syndrome.

Introduction.

- Premenstrual syndrome (PMS) is the symptom of stress that appears before the onset of menstruation. It also called premenstrual stress syndrome, premenstrual stress or premenstrual tension. It is last for about 4 to 5 days prior to menstruation.

Etiopathogenesis.

- It is cause by any hormonal imbalance in the body mainly progesterone and estrogen. It may be happened due to any stress, emotional behaviour and nutrient deficiency etc.

- The neurotransmitter that is most implicated in the manifestations of PMS is serotonin, although there is evidence to implicate beta-endorphin, gamma-aminobutyric acid (GABA), and the autonomic nervous system.
- It is cause by the salt and water retention by estrogen.

Clinical manifestations.

- Mood swings.
- Irritability, anger, anxiety.
- Less interested in sex wanting to be alone.
- Join pain, muscle pain.
- Emotional instability.
- Headache, indecision, insomnia.
- Constipation, and gastric abnormality.
- Abdominal cramping.
- Bloating (abdominal swelling).

Pharmacological managements.

- Antidepressants. Ex- Escitalopram, citalopram, fluoxetine.
- Diuretics. Ex- Furosemide, torsemide, spironolactone.
- Oral contraceptives. Ex- Norethindrone, levonorgestrel.
- NSAIDs. Ex- Ibuprofen, mefenamic acid, naproxen, celecoxib, nimesulide.

Non-pharmacological managements.

- Any symptoms appear then consult with the gynaecologist, and change their life style (sleep and wake up pattern) as per the instruction.
- Diet pattern is very essential because it maintain the BMR and maintain the body weight.
- Behavioural changes like (anger, sadness, anxiety) also cause the hormonal balancing, so try to make happy and cheerful.
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