PHARMACOLOGY

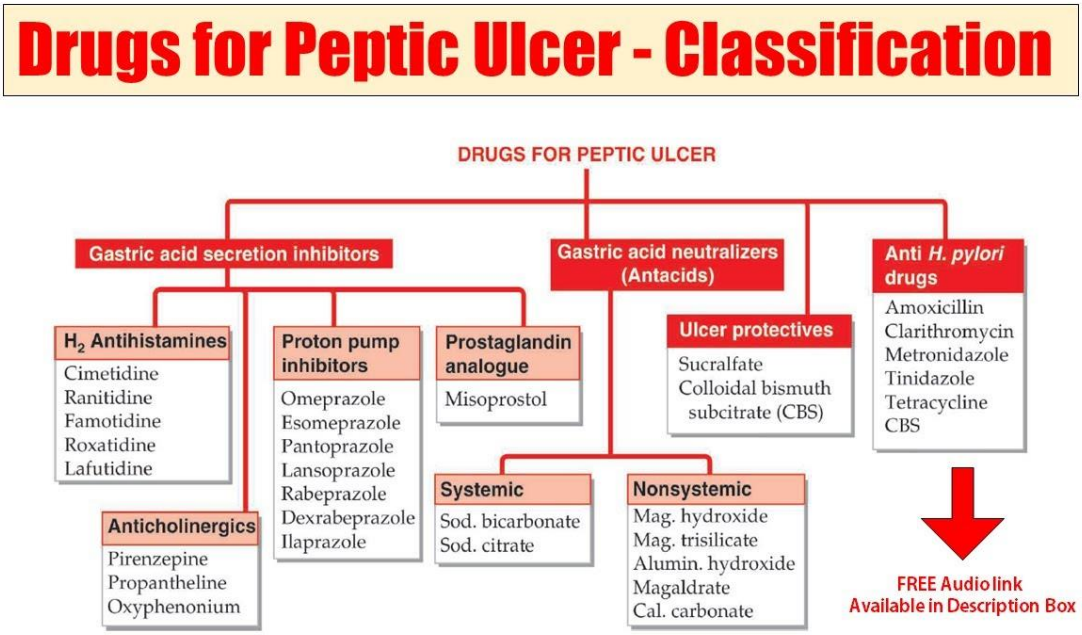
Drugs used for peptic ulcer

Different factors are thought to be the culprits.

- Helicobacter pylori
- NSAIDS
- Increased secretion of HCL
- Inadequate defense against acid secretion

Aim of therapy would be to

- Eradicate H. pylori
- Decrease acid secretion
- Increase mucosal defense mechanism

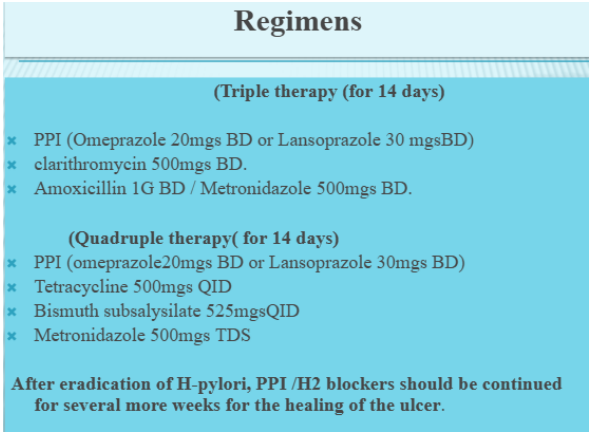


1. **Gastric acid inhibitors**
   a. **Proton pump inhibitors**: Omeprazole, Lansoprazole, Pantoprazole, Rabeprazole
   b. **H2-receptor blockers**: Cimetidine (Cimetidine can also have endocrine effects, because it acts as an **antiandrogen**. These effects include **gynecomastia**, **galactorrhea**, and **reduced sperm count**.), Ranitidine Famotidine, Roxatidine
   c. **Anti-muscrinic agents**: Pirenzipine, Telezipine
   d. **Prostaglandins**: Misoprostol Enprostil
2. **Enhancers of mucosal defense**
   a. **Prostaglandin analogues**: misoprostol
   b. **Ulcer protective**: Sucralfate, Colloidal Bismuth Subcitrate
3. **Drugs that neutralize gastric acid**
   a. **Systemic antacids**: Sodium bicarbonate, Sodium citrate

b. **Non-systemic antacids**: Magnesium hydroxide, magnesium trisilicate, aluminium hydroxide gel, calcium carbonate

4. **Anti- H. pylori drugs**

a. Amoxicilin, tetracycline, clarithromycin, metronidazole, bismuth subsalicylate, H2 antagonist & proton pump inhibitors



The **half-life of omeprazole** is approximately **0.5 to 1 hour** in healthy individuals.

Important Notes:

- **Duration of Action:** Despite its short half-life, the effect lasts up to **24 hours** because it **irreversibly inhibits the proton pump (H+/K+ ATPase)** in gastric parietal cells. New pumps need to be synthesized for acid production to resume.

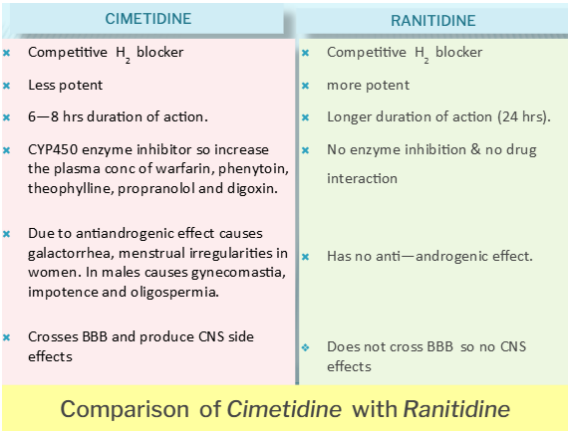
Therapeutic Uses

- **Peptic ulcer**: Patients with ***NSAID-induced ulcers*** should be treated with **PPIs**, because these agents heal and prevent future ulcers better with **H2 antagonists**
- **Acute stress ulcers**: These drugs are useful in managing acute stress ulcers associated with major physical trauma in high-risk **patients in intensive care units**. They are usually injected **intravenously**.
- **GERD**: They are used for the treatment **heart burn**. They relieve the heart burn in 50% of the patients. Since they relieve these symptoms of GERD by inhibiting the release of acid by blocking the H2-receptors therefore it requires a minimum of **45 minutes.**
- **Zollinger Ellison syndrome (ZES)**: definitive treatment is **surgery**, but PPI's and H2 control the hypersecretion of acid. The drug of choice being the **PPI's**.
- **(Preanesthetic medication):** are used preoperatively before anesthesia when the surgery is to be performed in **emergency** to **avoid acid aspiration pneumonia**.

PPI's should always be taken **30-60minutes prior to intake of breakfast or largest meal**.
Side effects=

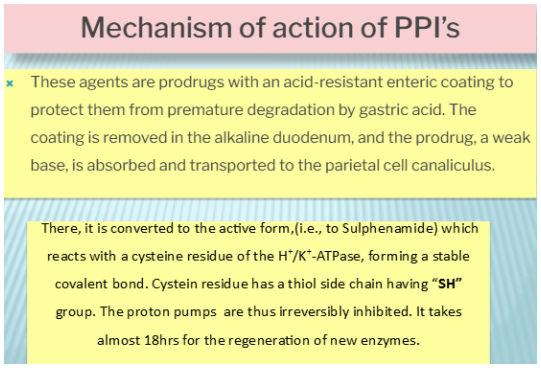
- risk of **fractures** as PH increases so calcium absorption in the bones decreases,
- Vit B12 absorption decreases,
- can cause **diarrhea** (c. difficile colitis),
- PPIs inhibit CYP2C19, **reducing clopidogrel effectiveness**.
- Inhibit CYP450, affecting **warfarin, phenytoin, diazepam, cyclosporine** metabolism (except **pantoprazole**).
- **Increase gastric pH**, reducing absorption of **ketoconazole, digoxin, iron**.

- Compared to Cimetidine, ranitidine is longer acting and is five- to ten-fold more potent. **Ranitidine** has minimal side effects and does not produce the antiandrogenic or prolactin-stimulating effects



CIMETIDINE	RANITIDINE
Competitive H_2 blocker	Competitive H_2 blocker
Less potent	more potent
6—8 hrs duration of action.	Longer duration of action (24 hrs).
CYP450 enzyme inhibitor so increase the plasma conc of warfarin, phenytoin, theophylline, propranolol and digoxin.	No enzyme inhibition & no drug interaction
Due to antiandrogenic effect causes galactorrhea, menstrual irregularities in women. In males causes gynecomastia, impotence and oligospermia.	Has no anti—androgenic effect.
Crosses BBB and produce CNS side effects	Does not cross BBB so no CNS effects

Comparison of *Cimetidine* with *Ranitidine*



- Contraindication of misoprostol= Pregnancy, IBD

Sucralfate – (Aluminum hydroxide + Sulphated sucrose.)
Mechanism of Action:

- Forms a **sticky gel** in **acidic pH (< 4),** adhering to ulcers and protecting them. **Binds to proteins** at the ulcer base, acting as a **physical barrier** against **acid and pepsin**. **Stimulates prostaglandins, epidermal growth factor**, increasing mucus and bicarbonate secretion.
- **Administration**: Take **1 hour before meals**.
- **Pharmacokinetics**: Requires an **acidic medium**, avoid with **PPIs, H2 blockers, antacids**.

- **Properties of an ideal antacid (weak bases) -- IMPORTANT**
  - Should be insoluble & non absorbable.
  - Should be capable of neutralizing acidity
  - Should not liberate CO2
  - Should not disturb acid base balance
- Aluminium antacids cause **constipation** and hypophosphatemia
- Magnesium antacids cause **diarrhoea**
- Calcium containing antacids lead to hypercalcemia and hypercalcaemia.

Benefits of Combination Antacids (non-systemic)

- **Balanced Effect**: Aluminium causes constipation; magnesium causes diarrhoea—**counteract each other**. **Rapid & Sustained Relief**: Magnesium acts fast; aluminium acts slow. **Reduced Toxicity**: Lower individual doses minimize systemic effects.

Difference between PPI and PCABs?

Feature	Proton Pump Inhibitors (PPIs)	Potassium-Competitive Acid Blockers (PCABs)
Examples	Omeprazole, Pantoprazole, Esomeprazole	Vonoprazan, Revaprazan
Mechanism of Action	**Irreversibly** inhibits H+/K+ ATPase	**Reversibly** blocks H+/K+ ATPase at K+ site

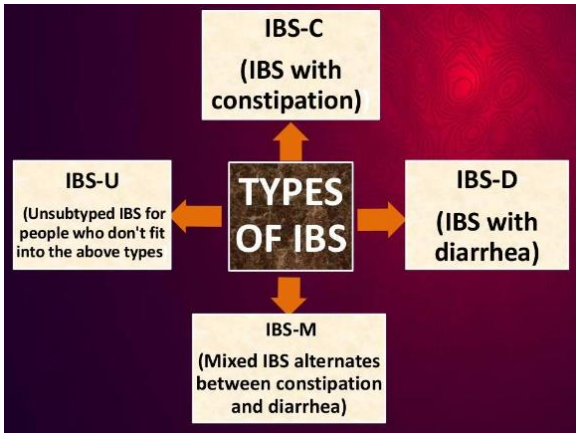
Onset of Effect	Takes hours to start, full effect in 1-4 days	**Rapid** onset, significant effect within hours
Duration of Action	Long-lasting (24-48 hrs), requires new enzyme synthesis	Sustained (~24 hrs), reversible binding
Activation Requirement	Needs acidic activation, affected by meal timing	Works regardless of acidity or meals
Advantages	Prolonged acid suppression	Faster, more consistent acid control

Summary

- PPIs: Irreversibly inhibit the proton pump, with a slower onset (1-4 days for full effect) and long-lasting acid suppression.
- PCABs: Reversibly block the proton pump with a faster onset (within hours) and consistent acid control, regardless of meal timing.

Drugs for irritable bowel syndrome

IRRITABLE BOWEL SYNDROME: It is a functional GI disorder characterized by recurrent abdominal pain or discomfort, associated with abnormal stool frequency or consistency, and **not** related to **structural** abnormalities of the GI tract.



Predominant symptoms	First line drugs	Second line drugs
Pain	Antispasmodic agents	Tricyclic antidepressives, Hypnosis, Psychological treatments
Diarrhoea	Loperamide	5-HT 3 antagonist
Constipation	Ispaghula	5-HT 4 agonist
Bloating with distension	Dietary manipulation Polyethylene glycols	Probiotics 5-HT 4 agonist
Bloating without distension	Antispasmodic agents	Probiotics Tricyclics

1. Antispasmodics (Anticholinergics)

- **Drugs: Dicyclomine, Hyoscyamine**
- **Mechanism:** Blocks muscarinic receptors, reducing gut spasms.
- **Use:** Relieves abdominal cramps in **IBS-D (diarrhea-predominant IBS)**.
- **Side Effects:** Dry mouth, blurred vision, constipation, urinary retention.

2. Antidiarrheal Agents *(For IBS-D)*

- **Drugs: Loperamide, Diphenoxylate**
- **Mechanism:** Slows intestinal motility via opioid receptor activation.
- **Use:** Reduces diarrhea in **IBS-D**.
- **Side Effects:** Constipation, bloating.

3. Laxatives *(For IBS-C)*

- **Drugs: Psyllium, Methylcellulose (fiber), Polyethylene glycol (PEG), Lactulose**
- **Mechanism:** Increases stool bulk or water retention.

- **Use:** Treats **IBS-C (constipation-predominant IBS)**.
- **Side Effects:** Bloating, gas.

4. Secretagogues *(For IBS-C)*

- **Drugs: Lubiprostone, Linaclotide, Plecanatide**
- **Mechanism:** Increases chloride secretion to enhance bowel movements.
- **Use:** Chronic **IBS-C**.
- **Side Effects:** Diarrhea, nausea.

5. Serotonin Modulators

- **For IBS-D: Alosetron (5-HT3 antagonist)** → Slows motility. **Use:** Severe IBS-D.
- **For IBS-C: Tegaserod (5-HT4 agonist)** → Increases motility. **Use:** IBS-C in women.

6. Antibiotics

- **Drug: Rifaximin**
- **Mechanism:** Alters gut microbiota, reducing bloating and diarrhea.
- **Use:** IBS with bloating.

7. Antidepressants (For Pain & Motility)

- **Drugs: Tricyclics (Amitriptyline, Nortriptyline), SSRIs (Fluoxetine, Sertraline)**
- **Mechanism:** Modulates gut-brain axis, reduces pain & motility disturbances.
- **Use:** IBS with chronic pain, depression, or mixed symptoms.

NEWER AGENTS: DOMPERIDONE, BILE ACID SEQUESTRANTS

8.Pharma viva

Drugs used for treatment of IBD

Class	Drugs	Mechanism of Action (MOA)	Use	Side Effects
Aminosalicylates	Mesalamine, Sulfasalazine	**Inhibit COX and LOX**, reducing prostaglandins and leukotrienes	Mild-to-moderate **IBD**	Nausea, headache, rash
Corticosteroids	Prednisone, Budesonide	Suppress inflammation via **inhibition of cytokines** and immune response	Remission induction in **IBD**	Weight gain, osteoporosis
Immunomodulators	Azathioprine, 6-MP, Methotrexate	**Inhibit purine synthesis**, suppress T-cell proliferation	Maintenance therapy	Bone marrow suppression, liver toxicity
Biologics	Infliximab, Adalimumab	TNF-α inhibitors; prevent TNF-α from binding to its receptors	Moderate-to-severe **IBD**	Infections, injection reactions

Antibiotics	Metronidazole, Ciprofloxacin	Inhibit bacterial DNA synthesis and reduce bacterial overgrowth	**Crohn's Disease**	Nausea, neuropathy
Other Agents	Probiotics, Bile acid sequestrants	Modulate gut microbiota or reduce bile acid reabsorption	Symptom relief	Bloating, gas

Pharmacokinetics of Mesalamine (5-ASA)

1. **Absorption**
   - Poorly absorbed in the small intestine.
   - Formulated for delayed or controlled release in the **colon** (site of action).
2. **Distribution**
   - Concentrates in the intestinal mucosa.
   - Minimal systemic distribution.
3. **Metabolism**
   - **In the liver**: Acetylation into N-acetyl-5-ASA (inactive metabolite).
   - Partially metabolized by intestinal bacteria.
4. **Excretion**
   - Mainly excreted in **feces**.
   - A small portion excreted in **urine** (as acetylated metabolite).

Role of Lactulose in Hepatic Coma

2. **Reduces Ammonia Levels**: **Converts ammonia (NH_3) to ammonium (NH_4^+),** which is non-absorbable and excreted in stool.
3. **Acidifies Colon**: Creates an acidic environment that inhibits ammonia-producing gut bacteria.
4. **Laxative Effect**: Promotes bowel movements, reducing ammonia absorption.
5. **Prevents Recurrence**: Used for both treatment and prevention of hepatic encephalopathy.

Anti-Integrin MOA (Mechanism of Action):

Blocks **integrins** on leukocytes, preventing adhesion and migration into inflamed tissues → reduces inflammation in autoimmune diseases like **IBD** and **MS**. **Example:** Natalizumab (targets α4-integrin).

Drugs used for hepatitis B

ACUTE HBV INFECTION TREATMENT

Requires no anti-viral therapy

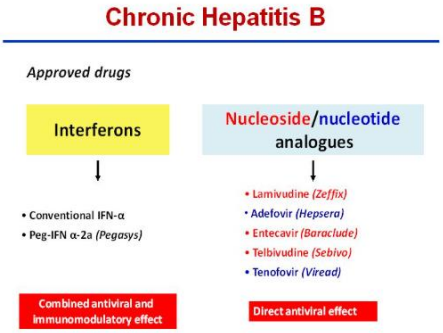
- **Bed rest**
- **10% Dextrose infusion**
- **Gastoprotective agents**
- **Antiemetics**
- **Oxazepam**

- **Detoxicants**

CHRONIC HBV INFECTION TREATMENT

Hepatitis B Drugs

1. **First-Line Antiviral Therapy**
   - **Tenofovir**: Inhibits reverse transcriptase, preventing the replication of hepatitis B virus (HBV) DNA.
   - **Entecavir**: Inhibits reverse transcriptase, blocking DNA replication, transcription, and synthesis.
2. **Alternative Antiviral**
   - **Lamivudine**: Inhibits reverse transcriptase, preventing the conversion of RNA to DNA.
   - **Adefovir**: Inhibits HBV DNA polymerase, blocking viral replication.
3. **Interferon Therapy**
   - **Pegylated Interferon-alpha**: Stimulates the immune system, enhancing antiviral activity and inhibiting HBV replication.
4. **For Acute Hepatitis B**
   - Supportive care, no antivirals unless severe.



Interferon Mechanism of Action (MOA):

Interferons are **cytokines** that enhance the immune response to fight **viral infections, tumors, and regulate immune activity**.

1. **Antiviral Action:**
   - Induce synthesis of antiviral proteins that inhibit viral replication by degrading viral RNA and blocking protein synthesis.
2. **Immunomodulatory Action:**
   - Increase **MHC I and II expression** → enhance antigen presentation to T cells.
   - Activate **natural killer (NK) cells** and macrophages to destroy infected or malignant cells.
3. **Antiproliferative Action:**
   - Inhibit cell growth and promote apoptosis in tumor cells.

Types:

- **IFN-α:** Used in Hepatitis B, C, and some cancers (e.g., melanoma, CML).

- **IFN-β:** Used in multiple sclerosis.
- **IFN-γ:** Enhances macrophage activity, used in chronic granulomatous disease.

The **Hepatitis B vaccine** is an **active immunization**. It stimulates the body to produce **protective antibodies (anti-HBs)** against the **Hepatitis B surface antigen (HBsAg)**.

Passive Immunization

In contrast, **Hepatitis B Immune Globulin (HBIG)** provides **passive immunity** by giving pre-formed antibodies, offering immediate but short-term protection. It's used in:

- **Post-exposure prophylaxis** (e.g., after needlestick injury)
- **Newborns of HBsAg-positive mothers** (along with the HBV vaccine)

Oral Drugs for Hepatitis C (HCV)

1. **Sofosbuvir**
    - **MOA**: Inhibits the HCV RNA-dependent RNA polymerase, preventing viral replication.
2. **Ledipasvir**
    - **MOA**: Inhibits the HCV NS5A protein, which is essential for viral RNA replication and virion assembly.
3. **Velpatasvir**
    - **MOA**: Inhibits the HCV NS5A protein, impairing viral replication and assembly.
4. **Glecaprevir**
    - **MOA**: Inhibits the HCV NS3/4A protease, preventing the cleavage of the viral polyprotein.
5. **Pibrentasvir**
    - **MOA**: Inhibits the HCV NS5A protein, impairing viral replication.
6. **Dasabuvir**
    - **MOA**: Inhibits the HCV NS5B polymerase, blocking RNA replication.

- **NS3/4A Protease Inhibitors**: (-**previr** suffix = *Protease inhibitors*)

Grazoprevir, Voxilaprevir

- **NS5A Inhibitors**: (-**asvir** suffix = *NS5A inhibitors*)

Ledipasvir, Velpatasvir, Pibrentasvir

- **NS5B Polymerase Inhibitors**: (-**buvir** = *Polymerase inhibitors*).

Sofosbuvir, Dasabuvir

Q. Mechanism of Action (MOA)

1. **Sofosbuvir** (Used for Hepatitis C)
    - **Nucleotide analog** that inhibits **NS5B RNA-dependent RNA polymerase**, preventing viral RNA replication.
2. **Lamivudine** (Used for Hepatitis B and HIV)
    - **Nucleoside reverse transcriptase inhibitor (NRTI)**
    - Incorporates into viral DNA and causes **chain termination**, inhibiting reverse transcription in HIV and HBV replication.

9.Pharma viva

How metoclopramide acts as prokinetic

Metoclopramide as a Prokinetic (increases GI motility)

- **5HT4 agonist** → ↑ Ach release → enhances motility.
- **D2 antagonist** → removes inhibitory effect on Ach.
- **Direct smooth muscle action** in the upper GIT.

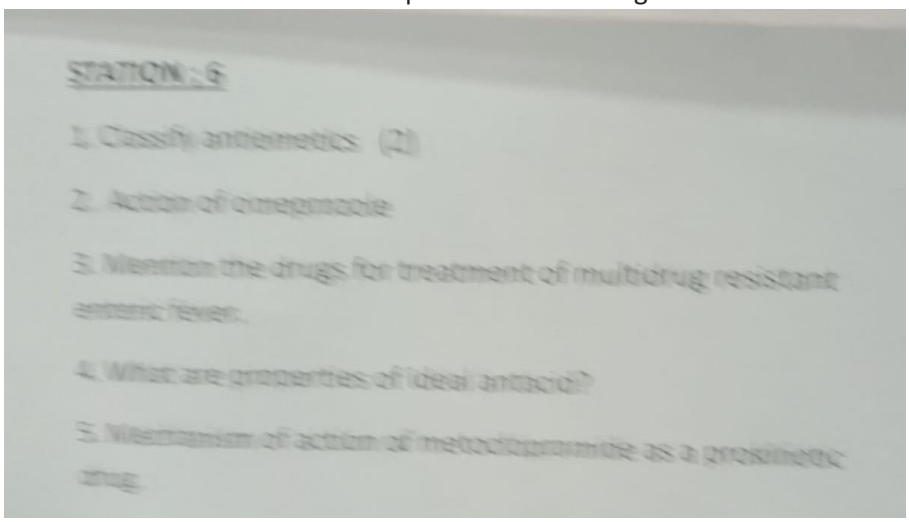
➢ Uses= anti-emetic, GERD, Gastroparesis

➢ SE=

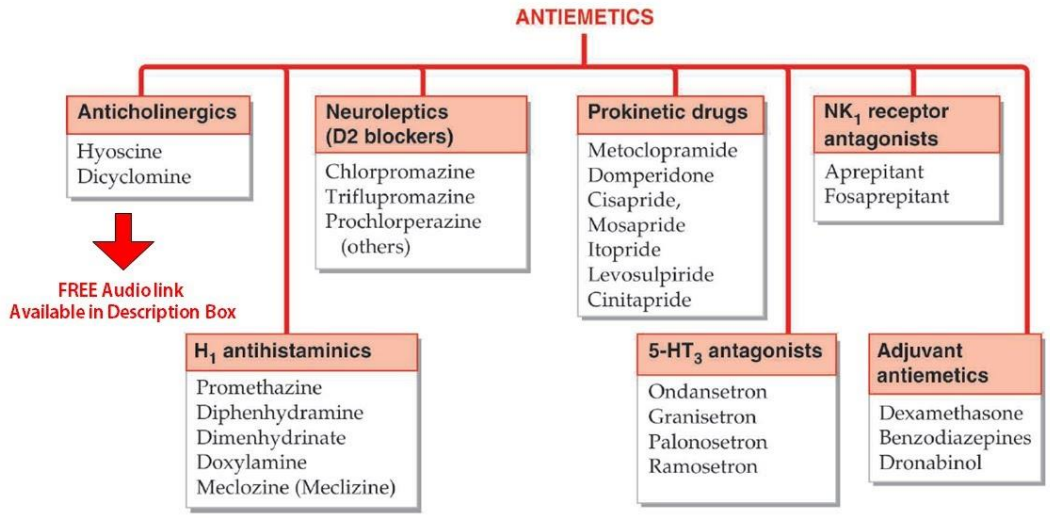
- **(Drug induced parkinsonism) = D2 BLOCKADE!**
- **Long term use** may lead to
  - Gynecomastia,
  - Galactorrhea
  - Menstrual Irregularities. (blockade of inhibitory effect of dopamine on prolactin release)

WITH LEVODOPA:

- Metoclopramide cross BBB>> blocks d2 receptor in basal ganglia>>>> interfere with antiparkinsonian effect of levodopa
- Do not use for levodopa induced vomiting.



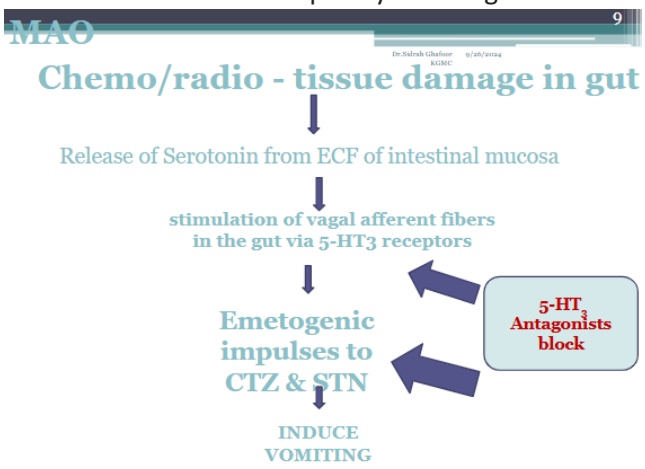
Antiemetic Drugs - Classification



Classify antiemetics

1. **Anti-cholinergic:**
    - *Scopolamine*—DOC for motion sickness
2. **Anti-histamines (H_1 blockers):** ---mainly in motion sickness
    - *Dimenhydrinate,*
    - *Diphenhydramine*
    - *Cyclizine, Meclizine*
    - *Promethazine, Hydroxazine***.**
3. **5-HT_3 -Receptor antagonists:** (Blocks 5HT3 receptors in vagal afferent fibers (**gut**), chemoreceptor trigger zone (**CTZ**), and **solitary tract nucleus**.)—mainly for CINV, pregnancy
    - *Ondansetron, Granisetron (more potent), Dolasetron (QT prolongation), palonosetron (longer duration)*
4. **Prokinetic drugs:**
    - *Metaclopromide (***D2-receptor blocker** with central (CTZ) and **peripheral prokinetic** effects via 5HT4 agonism, D2/5HT3 antagonism, and Ach release.*),*
    - *Domperidone, Cisapride, Mosapride.*
5. **Neuroleptics*:***
    - *Cholpromazine, fluphenazine, Prochlorperazinem, Haloperidol***.**
6. **Cannabinoids*:***
    - *Dronabinol.*
7. **Neurokinin NK1 receptor antagonists;** Blocks action of substance P in CTZ & NTS
    - *Aprepitant, Fosaprepetant*
8. **Adjuvant anti-emetics:**

- **Glucocorticoids:** *Betamethasone, Dexamethasone, Methylprednisolone.*
- **Benzodiazepines:** *Lorazepam, Alprazolam.* –mainly used for Psychogenic and anticipatory vomiting.



CINV; Emesis not only affects the quality of life but can lead to rejection of potentially curative antineoplastic treatment.

IMP

- **Domperidone** is favored especially in **chronic GI motility disorders**.
- **Metoclopramide** is the go-to for **acute nausea/vomiting**, but should be used **cautiously due to its CNS effect**s, especially in long-term use.

Interaction: Metoclopramide and Levodopa

- **Mechanism**: Metoclopramide, a dopamine antagonist, reduces the effectiveness of levodopa by blocking dopamine receptors.

For Morning Sickness:

- **Ondansetron**
- **Promethazine**
- **Doxylamine** + **Vitamin B6**

For Chemotherapy-Induced Vomiting:

- **Ondansetron**
- **Granisetron**
- **Dexamethasone**

Anti-salmonellosis Drugs:

1. **Selection of Antibiotics Based on Age, Pregnancy, and Resistance:**
    - **Age**:
        - **Children**: Typically treated with **Ceftriaxone** or **Ciprofloxacin** if severe; avoid tetracyclines in young children.

    - **Pregnancy**:
        - **First-Line**: **Ceftriaxone** or **Azithromycin** are preferred due to safety in pregnancy.
        - Avoid **Tetracyclines**, **Fluoroquinolones**, and **Aminoglycosides** during pregnancy.
    - **Resistance**:
        - **Extended-spectrum beta-lactamase (ESBL)-producing strains**: Use **Ceftazidime**, **Cefotaxime**, or **Carbapenems**.
        - **Multidrug-resistant strains**: Consider **Meropenem**, **Azithromycin**, or **Fluoroquinolones** based on sensitivity.
2. **Common Antibiotics**:
    - **Ciprofloxacin**
    - **Azithromycin**
    - **Ceftriaxone**
    - **Trimethoprim-sulfamethoxazole (TMP-SMX)**

Treatment for Multidrug-Resistant (MDR) Enteric Fever:

1. **Azithromycin** – First-line for uncomplicated cases.
2. **Carbapenems** (e.g., **Meropenem**) – Severe cases or complicated infections.
3. **Tigecycline** – Alternative for resistant cases.
4. **Ceftriaxone** – Extended-spectrum β-lactam for serious infections.

Prostaglandin (PG) Functions (Short):

- **PGE2:** Fever, pain, vasodilation, gastric protection
- **PGF2α:** Uterine contraction, bronchoconstriction
- **PGI2 (Prostacyclin):** Vasodilation, inhibits platelets
- **TXA2 (Thromboxane):** Vasoconstriction, platelet aggregation

Anti-diarrheal Agents:

1. **Opioids**
    - **Examples**: Loperamide, Diphenoxylate
    - **MOA**: Activate opioid receptors in the gut, decreasing motility and increasing transit time, leading to more water absorption.
2. **Colloidal Bismuth Compounds**
    - **Example**: Bismuth subsalicylate
    - **MOA**: Coats the gastrointestinal lining, reduces inflammation, and has mild antibacterial effects.
3. **Kaolin & Pectin**
    - **MOA**: Absorb toxins and excess fluid in the intestines, helping to bulk up stools and reduce diarrhea.

Laxatives:

1. **Bulk-forming Laxatives**

    - **Examples**: Psyllium, Methylcellulose
    - **MOA**: Absorb water, increase stool bulk, and stimulate peristalsis.
2. **Stool Softeners**
    - **Examples**: Docusate sodium
    - **MOA**: Lower surface tension, allowing water to enter stool and soften it.
3. **Osmotic Laxatives**
    - **Examples**: Lactulose, Polyethylene glycol (PEG)
    - **MOA**: Draw water into the intestines, increasing stool volume and promoting bowel movement.
4. **Stimulant Laxatives**
    - **Examples**: Senna, Bisacodyl
    - **MOA**: Stimulate the intestinal muscles to increase motility and promote defecation.

Pigmentation of the Colon (Melanosis Coli)

- **Definition:** A benign condition where the colonic mucosa becomes **brown to black** due to the accumulation of **lipofuscin** in macrophages within the lamina propria.
- **Cause:** Most commonly associated with prolonged use of **stimulant laxatives** (e.g., Senna, Bisacodyl).
- **Clinical Significance:** Harmless, no increased risk of cancer, and reversible upon stopping laxatives.

Erythromycin as a Prokinetic Agent

- **MOA:** Acts as a **motilin receptor agonist** → stimulates gastrointestinal smooth muscle contractions, particularly in the **stomach and duodenum**.
- **Uses:**
    - Gastroparesis
    - Delayed gastric emptying
    - Post-surgical ileus

MOA OF Albendazole: It inhibits microtubule synthesis in parasites by binding to β-tubulin, disrupting cell division, glucose uptake, and motility, leading to parasite death.

Amoebic Dysentery (Amoebiasis)

Infection occurs when mature cysts of *Entamoeba histolytica* are ingested, and they pass into the colon where they divide into trophozoites.

Two Forms of Treatment

1. Tissue Amoebicidal Drugs

- **Metronidazole (Flagyl)**:
    - **Indication**: DOC for extraluminal amebiasis.
    - **MOA**: Kills **trophozoites** but not cysts. Inhibits enzymes involved in DNA synthesis.
- **Tinidazole**:
    - **Indication**: Same as metronidazole but with a better toxicity profile.
    - **MOA**: Inhibits enzymes similar to metronidazole.
- **Chloroquine**:

    - **Indication**: Effective in **hepatic amoebiasis**.
    - **MOA**: Reaches high concentrations in the liver, effective in killing trophozoites.

2. Luminal Amoebicidal Drugs

- **Diloxanide Furoate**:
    - **Indication**: Used to eradicate trophozoites in the intestinal lumen, thus preventing cyst formation.
    - **Precaution**: Contraindicated in pregnancy and children under 2 years old.
- **Iodoquinol**:
    - **Indication**: Drug of choice for asymptomatic or moderate forms of amoebiasis.
- **Paromomycin**:
    - **Indication**: First-line treatment for amebiasis or giardiasis during **pregnancy**.

Interactions of Metronidazole:

- Increases **Warfarin** levels (enhanced anticoagulation).
- **Alcohol**: Disulfiram-like reaction (severe nausea, vomiting).
- **CYP450** inhibitors or inducers may alter its metabolism

Bowel lumen amoebiasis: Trophozoites (noninfective) and cysts (**infective**) are passed into the faeces.

Treatment is directed at eradicating cysts with a luminal amoebicide;

- **diloxanide furoate** (drug of choice)
- **iodoquinol or**
- **paromomycin** sometimes used.

Anti-helminthic drugs:

1. **Albendazole & Mebendazole**
    - **MOA**: Inhibits microtubule synthesis in worms, leading to paralysis and death.
    - **For**: Roundworms, Hookworms, Whipworms, Pinworms.
2. **Pyrantel Pamoate**
    - **MOA**: Depolarizing neuromuscular blocker that causes paralysis in worms.
    - **For**: Pinworms, Roundworms, Hookworms.
3. **Ivermectin**
    - **MOA**: Binds to glutamate-gated chloride channels in parasites, causing paralysis.
    - **For**: Scabies, Lice, Onchocerciasis, Strongyloidiasis, Filariasis.
4. **Praziquantel**
    - **MOA**: Increases cell membrane permeability to calcium, causing muscle spasms and paralysis in worms.
    - **For**: Schistosomiasis, Tapeworms.
5. **Niclosamide**
    - **MOA**: Inhibits mitochondrial phosphorylation in tapeworms, leading to their death.
    - **For**: Tapeworms.
6. **Diethylcarbamazine**
    - **MOA**: Immobilizes microfilariae, making them susceptible to immune destruction.
    - **For**: Filariasis, Loiasis.
7. **Doxycycline**

- **MOA**: Inhibits *Wolbachia* bacteria (endosymbionts of filarial worms), reducing worm fertility and survival.
- **For**: Filariasis (caused by *Wolbachia*).

Drugs for Variceal Hemorrhage:

1. **Somatostatin & Octreotide**
   - **MOA**: **Inhibit vasodilation** by reducing blood flow to the splanchnic circulation, decreasing portal pressure.
2. **Vasopressin & Terlipressin**
   - **MOA**: **Vasoconstrict** splanchnic vessels, reducing portal blood flow and pressure.
3. **Beta-blockers**
   - **MOA**: Reduce portal hypertension by **decreasing cardiac output**, thereby lowering portal pressure.

First-pass Hepatic Metabolism

- **Definition**: The process where a drug is metabolized in the liver before reaching systemic circulation, reducing its bioavailability.

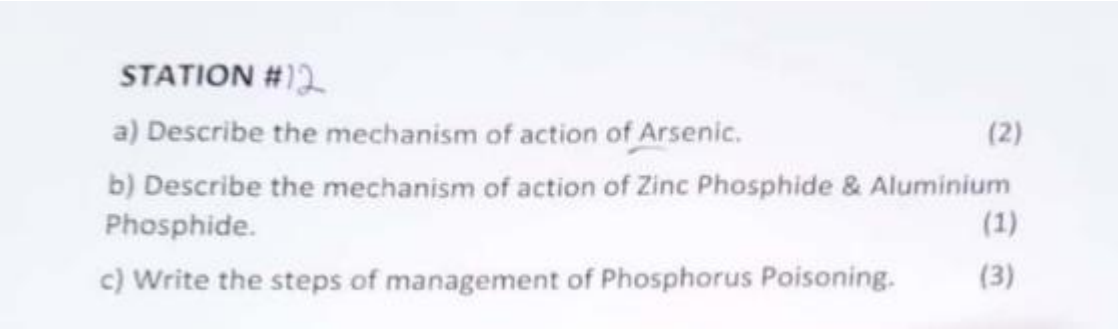
Common Hepatotoxic Drugs

- **Acetaminophen (Paracetamol)**
- **Alcohol**
- **Isoniazid**
- **Methotrexate**
- **Valproic acid**
- **Amiodarone**

Drug Treatment of Paracetamol Poisoning

- **N-acetylcysteine (NAC)**
  - **MOA**: Restores glutathione levels, detoxifying the reactive metabolites of paracetamol.
  - **Administration**: Given orally or intravenously based on severity.

FORENSICS



STATION #12

a) Describe the mechanism of action of Arsenic. (2)

b) Describe the mechanism of action of Zinc Phosphide & Aluminium Phosphide. (1)

c) Write the steps of management of Phosphorus Poisoning. (3)

- Arsenic posion MOA.
- classify vegetable posions.
- Obstructive jundice histroy

Here are the answers:

1. Mechanism of Action of Arsenic Poisoning

- Arsenic combines with **sulphydryl enzymes** and interferes with **cell metabolism**, causing both **local irritation** of mucous membranes and **systemic depression** of the nervous system.

2. Classification of Vegetable Poisons

- **Castor Seeds** – Contains **Ricin** (toxalbumin).
- **Croton Seeds** – Contains **Croton oil** (toxalbumin).
- **Abrus Precatorius (Jequirity Bean)** – Contains **Abrin**.
- **Colocynth (Bitter Apple)** – Contains **Colocynthin** (glycoside).
- **Calotropis (Madar)** – Contains **Calotropin**.

FIG. 37.6: Castor seeds

FIG. 37.5: Castor fruit

FIG. 37.7: Croton seeds

Table 37.2: Showing difference between castor and croton seeds

Features	*Castor seed*	*Croton seed*
Appearance	Grayish brown, mottled, glossy	Dark brown, non-glossy, not mottled
Shape	Flattened-oval in shape	Oval
Cross section at tip	Lumen is almost circular	The lumen is slit like with radiating creases

FIG. 37.2: Abrus seed—black variety

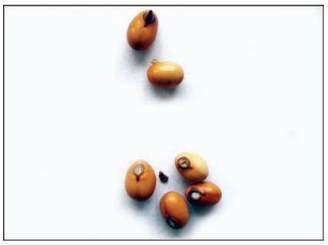


FIG. 37.3: Abrus seeds—white variety

FIG. 37.1: Abrus seeds—red variety

- Present in seed and is toxalbumin
- Abrin is similar to viper snake venom

FIG. 37.12: Marking nut

FIG. 37.10: Calotropis gigantea

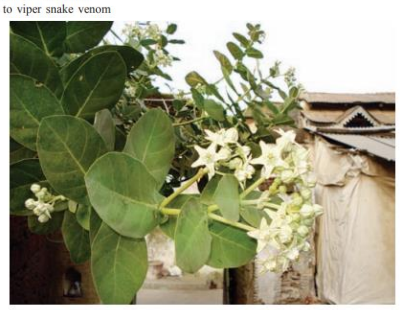


FIG. 37.11: Calotropis procera

Mechanism of action of all poisons almost

1. Sulphuric Acid

- **MOA:** Dehydrates tissues, causing **coagulative necrosis**.
- **Fatal Dose:** 5–10 ml concentrated acid.
- **Special Points:** Causes **black eschar** formation. Death is usually due to shock, perforation, or peritonitis.
- **Antidote:** No specific antidote; give **milk, egg white, or demulcents** to coat the mucosa

2. Nitric Acid

- **MOA:** Causes severe tissue corrosion via **oxidation**.
- **Fatal Dose:** 10 ml.
- **Special Points:** Turns tissues **yellow** due to the xanthoproteic reaction.
- **Antidote: Milk of magnesia or aluminum hydroxide** to neutralize acid

3. Hydrochloric Acid

- **MOA:** Protein denaturation and **liquefactive necrosis**.
- **Fatal Dose:** 10–15 ml.
- **Special Points:** Causes **white eschar** formation and severe gastric perforation.
- **Antidote: Magnesium oxide suspension** or milk

4. Carbolic Acid (Phenol)

- **MOA:** Coagulates proteins and depresses the CNS.
- **Fatal Dose:** 5–15 g.
- **Special Points:** Urine may turn **smoky or greenish-brown**.
- **Antidote: Glycerin or castor oil**, gastric lavage with **olive oil** .

5. Oxalic Acid

- **MOA:** Binds calcium to form **insoluble calcium oxalate**, leading to **hypocalcemia**.
- **Fatal Dose:** 15–30 g.
- **Special Points: Crystalline deposits in kidneys**, convulsions, and tetany occur.
- **Antidote: Calcium gluconate IV**, lime water

6. Cyanides

- **MOA: Blocks cytochrome oxidase**, preventing cellular respiration.
- **Fatal Dose:** 200–300 mg of potassium cyanide.
- **Special Points: Bitter almond odor**, bright red blood due to oxygen retention.
- **Antidote: Amyl nitrite, sodium nitrite, sodium thiosulfate**.

7. Copper

- **MOA: Inhibits cellular enzymes**, causing gastrointestinal and hepatic toxicity.
- **Fatal Dose:** 10 g copper sulfate.
- **Special Points: Greenish-blue vomiting**, jaundice, and hemolysis.
- **Antidote: Penicillamine, EDTA (chelating agent)**

8. Mercury

- **MOA: Inhibits sulfhydryl enzymes**, damaging kidneys and intestines.
- **Fatal Dose:** 1–4 g of mercuric chloride.
- **Special Points: Metallic taste**, **stomatitis**, renal failure.
- **Antidote: Dimercaprol (BAL), penicillamine**.

9. Arsenic

- **MOA:** Arsenic combines with **sulphydryl enzymes** and interferes with **cell metabolism**, causing both **local irritation** of mucous membranes and **systemic depression** of the nervous system.
- **Fatal Dose:** 100–200 mg.
- **Special Points: Garlic odor breath**, Mee's lines on nails, bloody diarrhea.
- **Antidote: Dimercaprol (BAL), penicillamine** .

10. Lead

- **MOA: Inhibits heme synthesis**, leading to anemia and CNS toxicity.
- **Fatal Dose:** Chronic exposure > 100 mg/day.
- **Special Points: Lead line (Burton's line), colic, encephalopathy**.
- **Antidote: EDTA, penicillamine**.

11. Phosphorus

- **MOA:** Causes **hepatocellular necrosis** and **mitochondrial poisoning**.
- **Fatal Dose:** 50–100 mg.
- **Special Points: Garlic odor breath, phosphorescent vomit, "phossy jaw"** (jaw necrosis).
- **Antidote: Copper sulfate (forms insoluble copper phosphide), gastric lavage** .

12. Aluminium Phosphide

- **MOA:** Releases phosphine gas, which **inhibits mitochondrial cytochrome oxidase**.
- **Fatal Dose:** 0.5–1 g.
- **Special Points: Cardiogenic shock, metabolic acidosis, garlic odor breath**.
- **Antidote:** No specific antidote; **magnesium sulfate infusion, supportive care** .

13. Chlorine

- **MOA:** Reacts with water in the respiratory tract to form hydrochloric acid, causing **pulmonary edema**.
- **Fatal Dose:** Prolonged exposure to >1000 ppm.
- **Special Points:** Causes **greenish-yellow gas inhalation injury**.
- **Antidote: 100% humidified oxygen, bronchodilators**

14. Iodine

- **MOA:** Corrosive action on mucosa, protein precipitation.
- **Fatal Dose:** 2–3 g.
- **Special Points:** Causes **brown discoloration of mucosa, burning pain, metabolic acidosis**.
- **Antidote: Starch solution, milk, gastric lavage with sodium thiosulfate** .

15. Snake Venom

- **MOA:**
  - **Neurotoxic (Elapids – Cobra, Krait):** Blocks acetylcholine at NMJ, causing paralysis.
  - **Hemotoxic (Vipers – Russell's Viper):** Causes DIC and hemorrhage.
  - **Myotoxic (Sea Snakes):** Causes muscle necrosis and renal failure.
- **Fatal Dose:** Varies by species.
- **Special Points: Ptosis, paralysis (neurotoxic), bleeding diathesis (hemotoxic)**.

16. Scorpion Venom

- **MOA: Blocks potassium and sodium ion channels**, causing autonomic storm.
- **Fatal Dose:** 100–200 mg (varies by species).
- **Special Points:** Severe **hypertension, pulmonary edema, shock**.
- **Antidote: Scorpion antivenom, calcium gluconate, prazosin**.

Forensic

- Phenolic crystals
- oxalic acid antidote
- Plants poisons
- Zinc phosphate mechanism
- Arsenic mechanism
- Arsenic poison management
- Lead line
- Mees line
- Phossy jaw

1. Phenolic Crystals

- Carbolic acid (phenol) exists as **colorless prismatic needle-like crystals** when pure.

FIG. 34.1: Carbolic acid crystals

FIG. 34.3: Oxalic acid

2. Oxalic Acid Antidote

- **Antidote:** Any **calcium preparation** (e.g., chalk suspension or calcium gluconate) to form **insoluble calcium oxalate**, which reduces toxicity

Zinc phosphate mechanism

- After ingestion zinc phosphide **reacts with gastric acid** to release **phosphine gas**, which is highly toxic, causing dyspnea, pulmonary edema, liver damage, and eventual collapse .

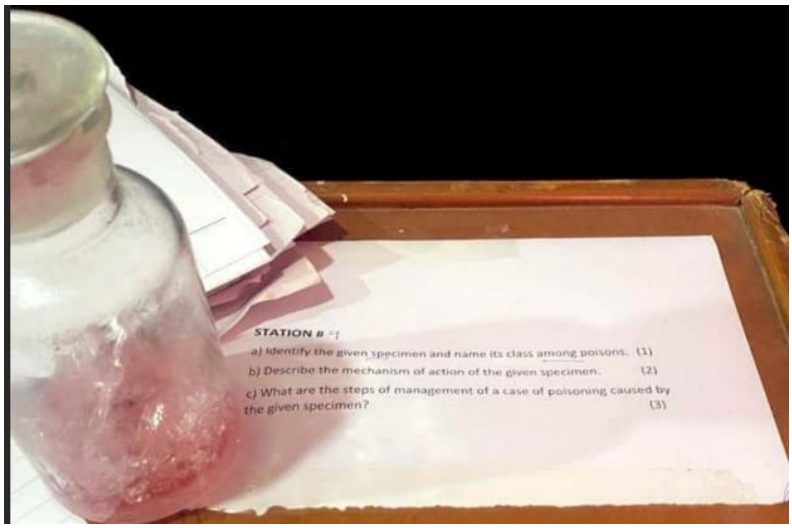
1. **Arsenic Mechanism**:
   Inhibits cellular metabolism by binding to sulfhydryl groups, disrupting enzymes and ATP production.

- **Initial Treatment**: Decontamination (activated charcoal if ingestion is recent).
- **Chelation Therapy**: Use **Dimercaprol** or **DMSA (Dimercaptosuccinic acid)** to bind and remove arsenic from the body.
- **Supportive Care**: Management of symptoms like fluid balance, electrolytes, and organ support.

1. **Lead Line**:
   A blue-black line on gums seen in chronic lead poisoning.
2. **Mees Line**:
   White transverse bands on nails, associated with arsenic poisoning.
3. **Phossy Jaw**:
   Jawbone necrosis from chronic phosphorus exposure, common in matchstick workers.

Table 36.3: Showing difference between arsenic poisoning and cholera

Features	*Arsenic poisoning*	*Cholera*
Pain in throat	Before vomiting	After vomiting
Conjunctiva	Inflamed	Not inflamed
Vomitus	Contains mucus, bile and streaks of blood	It is watery or whey like
Purging	Follows vomiting	Usually precedes vomiting
Stools	Rice watery in early stages, later becomes bloody, discharged with straining and tenesmus	Rice water liquid, involuntary jet
Laboratory examination	1. Radio-opaque shadow on X-ray of abdomen in arsenic trioxide poisoning 2. Coproporphyrin in urine 3. Arsenic detected in chemical analysis	Vibrio cholera detected on microscopic examination



A) Phenol crystals, CORROSIVE POISONS

B) Carbolic acid causes **protein coagulation**, **cell membrane disruption**, and **nervous system depression** by denaturing cellular proteins.

C) Treatment

- **Gastric Lavage:** With 10% glycerine or water containing magnesium sulfate until washings are free of phenolic odor.
- **Magnesium Sulfate:** Forms an insoluble **sulphocarbolate** to reduce toxicity.
- **Alcohol:** Given as a 10% solution to reduce cauterizing action but should be removed soon after.
- **Supportive Therapy:** Intravenous saline with sodium bicarbonate, demulcents (milk or egg white), and local treatment for burns .

IMP POINT Carboluria: Urine turns dark smoky green due to oxidation of metabolic products (hydroquinone, pyrocatechol).

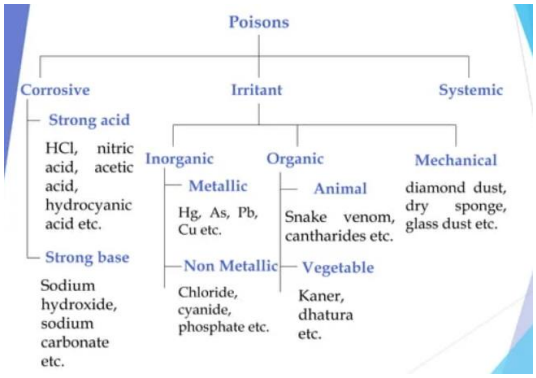
Forensic non-observed...marking nut, it's active principle, fatal dose and medicolegal importance
Marking Nut (Semecarpus Anacardium)

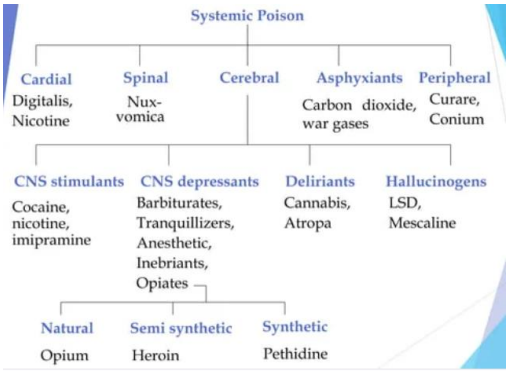
1. **Active Principle:**
   - **Bhilawanol** (a phenolic compound)
2. **Fatal Dose:**
   - About **5 to 10 g** of the nut can be fatal.
3. **Medicolegal Importance:**
   - **Homicidal Use:** Used in homicidal poisoning by mixing with food or applying on the skin to cause vesication.
   - **Suicidal Use:** Occasionally used for self-harm by ingestion or skin application.
   - **Mischief:** Applied to skin to simulate assault or injury due to its vesicant properties, often for false allegations.

Table 37.3: Displaying difference between contusions and marking nut lesion

Features	*Contusion*	*Marking nut lesion*
Shape	Regular	Irregular
Margin	Diffused	Sharp and clear
Color changes	Occurs	Does not occur
Itching	Absent	Present
Extravasation of blood	Present	Absent
Blisters	Absent	Present
Nail beds	Not significant	Similar lesions due to itching
Caused by	Rupture of capillaries	Chemical damage to skin

5.Forensic viva





Classify corrosives

Corrosive poisons are classified into the following categories:

1. **Mineral Acids**
   a. Sulphuric acid
   b. Nitric acid
   c. Hydrochloric acid
2. **Organic Acids**
   a. Oxalic acid
   b. Carbolic acid

c. Acetic acid
d. Salicylic acid

3. **Vegetable Acid**
   a. Hydrocyanic acid
4. **Alkalis**
   a. Caustic potash and soda
   b. Ammonium hydroxide.

Mechanism of action of mineral acids

The **mechanism of action (MOA)** of mineral acids is primarily local and involves:

1. **Extraction of Water from Tissues** – This leads to tissue dehydration.
2. **Coagulation of Cellular Proteins** – Resulting in tissue necrosis and the formation of eschars.
3. **Conversion of Hemoglobin into Hematin** – Causing further damage to the blood and tissues.

Difference between action of mineral acids and vegetable acids!

Aspect	**Mineral Acids**	**Vegetable Acids**
Examples	Sulphuric, Nitric, Hydrochloric Acid	Oxalic Acid, Acetic Acid
Action	Strong corrosive, coagulation necrosis	Mild corrosive, combines with calcium
Local Effects	Severe tissue destruction, discoloration	White mucous membrane, less tissue damage
Systemic Effects	Rare unless large quantity absorbed	Hypocalcemia, kidney damage (oxaluria)
Postmortem Findings	Perforation common, extensive corrosion	Perforation rare, dark brown gelatinous stomach content
Toxicity	Local destruction and shock	Remote toxic effects on calcium and kidneys

Toxic compounds of mercury...mercuric and mercurous chloride....which is more toxic..mercuric... (The poison which is absorbable, soluble is more toxic) Mercuric chloride is significantly more toxic due to its better absorption and potent corrosive effects.

Is mercury metal toxic...no ...when fumes inhaled,they are toxic

- **Mercury Compounds**:
  Mercuric chloride ($HgCl_2$) is more toxic than **Mercurous chloride (Hg_2Cl_2)** due to better absorption and potent corrosive effects.
- **Mercury Metal**:
  Mercury metal is **not toxic** in its elemental form, but **fumes** inhaled can be highly toxic.

Treatment of hydrocyanic acid and antidote

Treatment and Antidote for Hydrocyanic Acid Poisoning:

1. Immediate Treatment:

- Goal: Reverse cyanide-cytochrome combination by converting hemoglobin into methaemoglobin, which binds with cyanide to form nontoxic cyanomethemoglobin.
- Cyanide can also be converted to **non-toxic thiocyanate** for excretion.

2. Antidotes:

- **Dicobalt Tetracemate (Kelocyanor)**: Two 20 ml ampoules of 1.5% solution IV, followed by 20 ml of 50% glucose.
- **Methylene Blue**: 50 ml of 1% solution IV (converts hemoglobin into methaemoglobin).
- **Hydroxocobalamin**: 50 mg/kg IV infusion (combines with cyanide to form cyanocobalamin, which is excreted in urine).

3. Supportive Treatment:

- **Stomach Lavage**: With a 5–10% solution of **sodium thiosulphate** or a mixture of ferrous and ferric sulphates, followed by potassium carbonate to form **Prussian blue** (inert).
- **Inhalation Poisoning**: Remove the patient from the source and administer **100% oxygen**.
- **Intravenous Sodium Nitrite and Sodium Thiosulphate**: Repeat if symptoms reappear.

6.Forensic viva

Uses of phosphorus

Phosphorus mechanism of action

Types of burns caused by phosphorus

1. Uses of Phosphorus:

- **Match Industry:** Used in making match heads and matchbox striking surfaces.
- **Pesticides and Rodenticides:** Yellow phosphorus is used in rat poison.
- **Fireworks and Pyrotechnics:** Provides illumination and creates colorful sparks.
- **Alloy and Metallurgical Industry:** Component of special alloys and phosphor bronze.
- **Fertilizers:** Phosphorus compounds are essential in agriculture for fertilizers.

2. Mechanism of Action of Phosphorus:

Phosphorus is a **protoplasmic poison** that affects cellular oxidation. Its action is comparable to ischemia, causing reduced cellular metabolism under anoxic conditions. This leads to:

- Inhibition of glycogen deposition in the liver.
- Increased fat deposition (fatty degeneration).
- Necrobiosis in organs, especially the liver.

3. Types of Burns Caused by Phosphorus:

- **Slow Healing Burns:** Contact with phosphorus causes deep, slow-healing burns.
- **Vesication (Blister Formation):** When phosphorus is dissolved in carbon disulfide, it gets oxidized by air and ignites when the solvent evaporates, leading to vesication.
- **Keloid Scarring and Disfigurement:** Burns may be followed by keloid scar formation and disfigurement.

VIVA

1. Stages of Chronic Arsenic Poisoning:

- **First Stage: Nutritional and Gastrointestinal Disturbances**
  Gradual emaciation, loss of appetite, nausea, intermittent vomiting, and diarrhea.
- **Second Stage: Catarrhal Changes**
  Inflammation of mucous membranes, conjunctivitis, coughing, and bronchial catarrh.
- **Third Stage: Skin Rashes**
  Vesicular eruptions, nettle-like rash, patchy brown pigmentation, hyperkeratosis of palms and soles, Mee's lines on nails.
- **Fourth Stage: Nervous Disturbances**
  Tingling and numbness, muscle tenderness, paresis, headache, and impaired vision.

2. Abrus Precatorius (Jequirity Bean):

Seed Types:

- Red seeds with a black spot at one pole.
- Black seeds
- Brown seeds
  Small pea-sized, tasteless, and odorless.

Medicolegal Importance:

- Used as an arrow poison.
- Poisoning of cattle through fine needles (sui) containing powdered seeds mixed with toxins.
- Occasionally used in homicide by contaminating wounds or producing conjunctivitis.

3. Castor Oil Active Compound:

The active compound in castor seeds is **ricin**, a toxalbumin. Castor oil itself does **not contain ricin** and is therefore non-toxic.

4. Xanthoproteic Reaction:

This test is used to detect proteins. **When nitric acid reacts with proteins containing aromatic amino acids (like tyrosine or tryptophan), a yellow color develops, indicating the presence of proteins.**

5. Contraindications in Mineral Acid Poisoning Treatment:

Sodium Carbonate and Strong Alkalis are contraindicated because they can cause **exothermic** reactions, leading to further tissue damage.

7.Forensic non-observed...
5 vegetable poisons with active principle, what is smoky stool syndrome

1. Five Vegetable Poisons with Active Principles

1. **Ricinus Communis (Castor Oil Plant)** – Active Principle: **Ricin** (toxalbumin)
2. **Croton Tiglium (Jamalgota)** – Active Principle: **Crotin** (toxalbumin)
3. **Abrus Precatorius (Jequirity Bean)** – Active Principle: **Abrin** (toxalbumin)
4. **Calotropis (Madar)** – Active Principles: **Uscharin, Calotoxin, Calactin, Calotropin**
5. **Cerbera Odollam (Dabur)** – Active Principle: **Cerberin** (glycoside)

2. Smoky Stool Syndrome

It refers to a condition where the patient's urine appears clear initially but later turns **smoky-colored** due to the presence of **hydroquinone and pyrocatechol**, which are metabolic products of **carbolic acid**.

HCN treatment and antidote

Hydrogen Cyanide (HCN) Poisoning Postmortem Findings

1. **External Findings:**
    - Bright **cherry red skin color** due to high oxygen content in the blood.
    - **Cyanosis** may be present around the lips and extremities.
    - Bitter almond smell (not detectable by all).
2. **Internal Findings:**
    - **Blood:** Bright red and fluid.
    - **Lungs:** Congested with petechial hemorrhages.
    - **Stomach:** Erosions if ingested, with inflamed mucosa.
    - **Brain:** Congestion and petechial hemorrhages in meninges.
    - **Heart:** Flaccid, congested, and filled with bright red blood
3. **Immediate Actions:**

- **Remove from exposure** to fresh air.
- **Ensure airway, breathing, and circulation** (ABC).

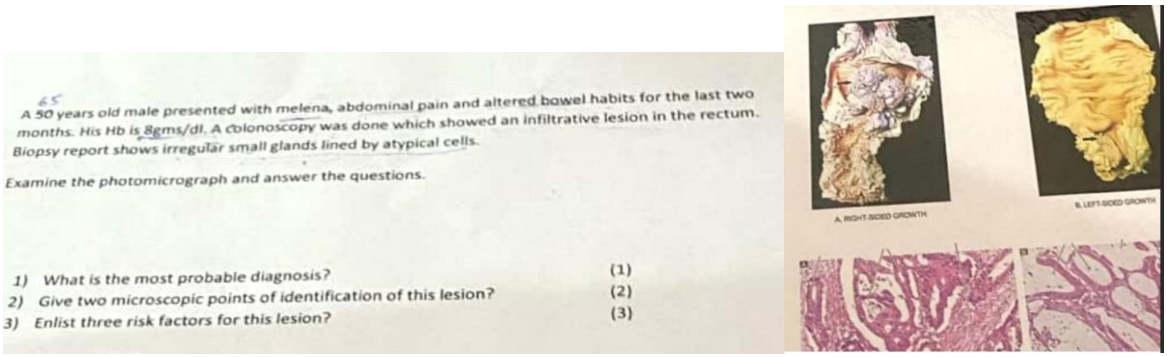
4. **Antidotes:**

- **Sodium Nitrite (3% solution, IV):** Converts hemoglobin to methemoglobin, which binds cyanide to form cyanomethemoglobin.
- **Sodium Thiosulfate (25% solution, IV):** Converts cyanide to nontoxic thiocyanate, which is excreted in urine.
- **Hydroxocobalamin (5 g IV):** Binds cyanide to form cyanocobalamin (Vitamin B12), which is excreted.

5. **Supportive Care:**

- 100% **oxygen** administration.
- **IV fluids** to maintain blood pressure.
- **Seizure control** with benzodiazepines (if needed).

PATHOLOGY



1) **COLORECTAL ADENOCARCINOMA**
2) **IRREGULARLY ARRANGED SMALL GLANDS**
3) **ATYPICAL EPITHELIAL CELLS WITH HYPERCHROMATIC NUCLEI, INCREASED MITOTIC FIGURES, LOSS OF NORMAL ARCHITECTURE**
4) **1. DIETRY FACTORS (HIGH FAT, LOW FIBER), 2. GENETIC PREDISPOSTITIN (FAP, LYNCH SYNDROME, FAMILY HISTROY), 3. IBD (UC/CD)—CHRONIC INFLAMATION**

Difference between Mallory-Weiss Syndrome and Boerhaave Syndrome

Mallory-Weiss Syndrome and Boerhaave Syndrome are both conditions involving damage to the esophagus, but they differ in the extent of the tear and associated causes.

Mallory-Weiss Syndrome

- Cause: Often results from severe vomiting, retching, or coughing, leading to tears in the mucosal lining of the esophagus, usually at the gastroesophageal junction.
- Tear Extent: Partial-thickness tear, affecting only the mucosal layer.
- Symptoms: Typically presents with hematemesis (vomiting blood) and sometimes mild pain.
- Treatment: Often managed conservatively with acid suppression and antiemetics; most cases resolve without surgery.

Boerhaave Syndrome

- Cause: Often results from forceful vomiting or intense pressure (e.g., from coughing, childbirth, or seizures), causing a full-thickness rupture of the esophagus.
- Tear Extent: Full-thickness tear, meaning the rupture extends through all layers of the esophagus.
- Symptoms: Severe chest pain, subcutaneous emphysema (air under the skin), and may develop into mediastinitis (inflammation in the chest cavity) due to leakage of esophageal contents.
- Treatment: Considered a medical emergency, typically requires surgical repair and intensive care to prevent complications like infection and sepsis.

Summary

- Mallory-Weiss: Partial tear at the mucosa, managed conservatively.
- Boerhaave: Full-thickness rupture, requires emergency surgical intervention.

Common cause of esophagitis and types of esophagitis

Common Causes of Esophagitis:

1. **GERD** – Most common cause
2. **Infectious Esophagitis** – Common in immunocompromised patients (e.g., **Candida, HSV, CMV**)
3. **Drug-induced Esophagitis** – From medications like NSAIDs, bisphosphonates, doxycycline
4. **Eosinophilic Esophagitis** – Allergic reaction, associated with atopy
5. **Caustic Esophagitis** – Ingestion of corrosive substances (acid or alkali)

. Esophagitis Types

1. **Reflux Esophagitis (GERD):** Acid reflux causing inflammation
2. **Infectious Esophagitis:** Common in immunocompromised patients
    1. *Pathogens:* Candida, HSV, CMV

3. **Eosinophilic Esophagitis:** Allergic inflammation, associated with atopy
4. **Drug-Induced Esophagitis:** Pill-induced (bisphosphonates, NSAIDs)
5. **Radiation Esophagitis:** Post-radiation therapy
6. **Caustic Esophagitis:** Chemical injury (alkali or acid ingestion)

- Alcoholic liver disease
- Esophagitis types
- Colorectal carcinoma genes mutation step wise
- Consequences of portal hypertension

1. Alcoholic Liver Disease

- **Pathogenesis:** Chronic alcohol intake → steatosis (fatty liver) → alcoholic hepatitis → cirrhosis.
- **Clinical Features:**
    - Hepatomegaly, jaundice, ascites
    - Signs of cirrhosis: spider angiomas, palmar erythema, gynecomastia
- **Histological Stages:**
    1. **Steatosis:** Fat accumulation in hepatocytes
    2. **Alcoholic Hepatitis:** Inflammation, Mallory bodies, neutrophilic infiltration
    3. **Cirrhosis:** Fibrosis, regenerative nodules, liver dysfunction
- **Complications:** Cirrhosis, hepatocellular carcinoma, portal hypertension.

3. Colorectal Carcinoma Gene Mutations (Stepwise Progression)

- **Stepwise Genetic Model (Vogelstein model):**
    1. **APC Mutation (Adenoma Formation)** – Early event
    2. **KRAS Mutation (Adenoma Growth)**
    3. **p53 Mutation (Malignant Transformation)**
    4. **DCC Mutation (Invasion and Metastasis)**
- **Pathways:**
    - **Chromosomal Instability Pathway (85%)** – APC/KRAS/p53
    - **Microsatellite Instability Pathway (15%)** – Mismatch repair genes (MLH1, MSH2)

4. Consequences of Portal Hypertension

1. **Variceal Bleeding:** Esophageal, gastric, and rectal varices
2. **Ascites:** Accumulation of fluid in the peritoneal cavity
3. **Splenomegaly:** Hypersplenism causing pancytopenia
4. **Hepatic Encephalopathy:** Ammonia accumulation → neurotoxicity
5. **Caput Medusae:** Distended abdominal veins
6. **Hepatorenal Syndrome:** Renal failure secondary to severe liver disease

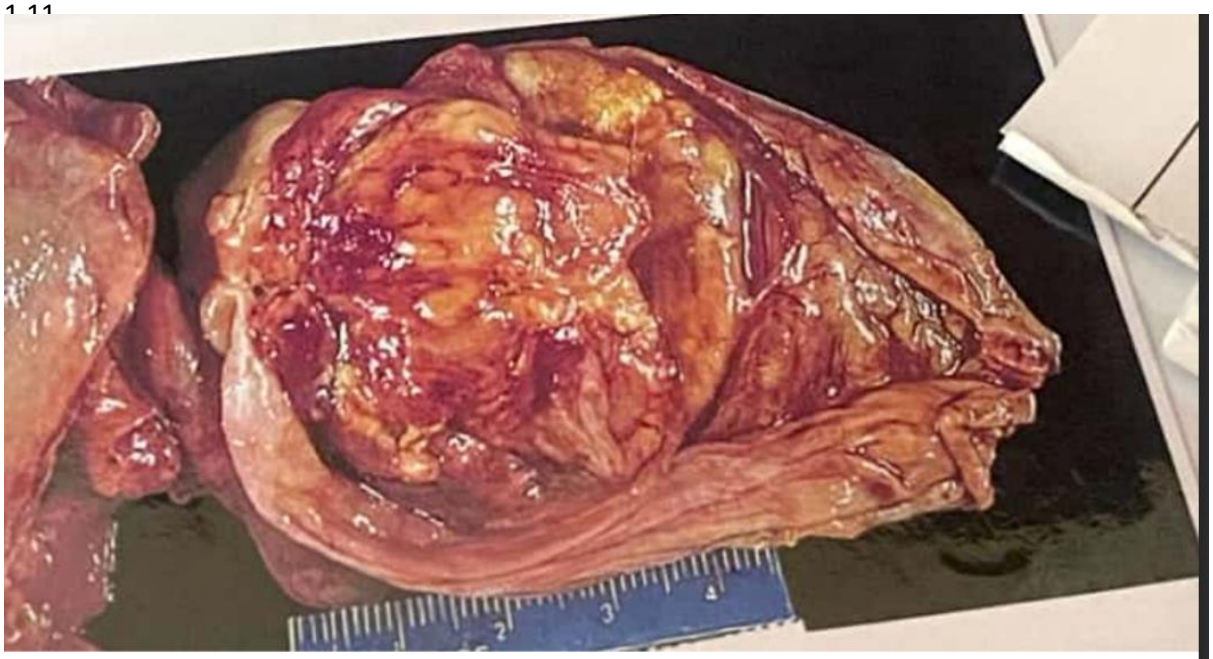
MARKS : 06

This photograph is from a 54-year-old female presented to the emergency department with severe, constant and sharp pain in right upper quadrant of abdomen for 2 days. Initially, the pain was not constant but has become so with several episodes of vomiting. Pain is radiating to the back to the tip of right shoulder. She has experienced similar, but much less severe abdominal pain for the last 3 years. She does not report any other remarkable feature. Her past medical history is significant for obesity, hypertension, and diabetes.

By looking at scenario and photograph, answer following questions.

1. What is your diagnosis? 1
2. What is the most common risk factor for this condition? 1
3. Name two growth patterns of this condition on gross and microscopic examination 2
4. What condition can be considered in differential diagnosis of this lesion? 1
5. What is prognosis of this condition? 1

1 11



1. GALLBLADDER CARCINOMA
2. GALLSTONES (CHOLELITHIASIS)
3. GROSS= 1. INFITRATIVE, 2. EXOPHYTIC, MICROSCOPIC= 1. ADENOCARCINOMA, 2. PAPILLARY CARCINOMA
4. METASTASIS FROM OTHER ORGAN, CHRONIC CHOLECYSTITS WITH XANTHOGRANULOMATOUS INFLAMMATION, GB ADENOMOMATOSIS
5. POOR PROGNOSIS

Adenocarcinoma of the GIT

Histology

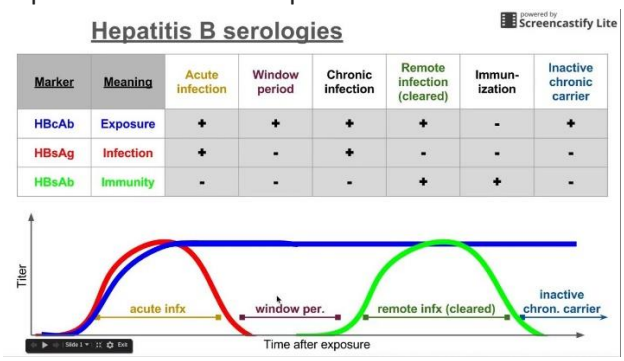
- **Well to poorly differentiated glands** forming mucin-producing cells.
- **Signet ring cells** (in diffuse gastric adenocarcinoma).
- **Invasive growth** into surrounding tissues.

Risk Factors

1. **Gastric Adenocarcinoma**
    - **H. pylori** infection
    - Chronic gastritis, smoking, high-salt diet
    - Genetic mutations (CDH1 mutation in diffuse type)
2. **Colorectal Adenocarcinoma**
    - **APC gene mutation, KRAS mutation, p53 mutation**
    - Low-fiber, high-fat diet
    - Family history, IBD (ulcerative colitis, Crohn's disease)

19.Patho non-observed

Hepatitis-B markers etc..pic shared



	Acute HBV	Chronic HBV	Cleared HBV	Vaccination
HBcAb IgM	+	-	-	-
HBcAb IgG	+	+	+	-
HBsAg	+	+	-	-
Anti-HBs	-	-	+	+
HBeAg	+	+/-	-	-
Anti-HBe	-	+/-	+/-	-
HBV DNA	High/Low	Low/High	-	-

Stage	ALT	HBeAg	HBV DNA
Immune tolerant	Normal	Positive	High
Immune active	High	Positive/Negative	Low
Immune surveillance	Normal/Slightly raised	Mostly negative	Low
Immune escape	High	Negative	High

Patho viva

Types of Gallstones:

1. **Cholesterol Stones**
    - Yellow-green, mostly cholesterol.
    - Associated with obesity, pregnancy, and hyperlipidemia.
2. **Pigment Stones**
    - **Black**: Calcium bilirubinate, seen in chronic **hemolysis** (e.g., sickle cell anemia).
    - **Brown**: Associated with biliary **infections** and **parasites** (e.g., Clonorchis).
3. **Mixed Stones**
    - Combination of cholesterol, calcium, and bile pigments.

Morphological Features of Chronic Cholecystitis:

1. **Thickened gallbladder wall**
2. **Fibrosis** and **mononuclear cell infiltration** (lymphocytes, plasma cells)
3. **Rokitansky-Aschoff sinuses** (mucosal outpouchings into the muscle layer)
4. **Mucosal atrophy**
5. **Serosal fibrosis and adhesions**

In severe cases, the gallbladder may be **contracted** or show **calcification** (porcelain gallbladder).

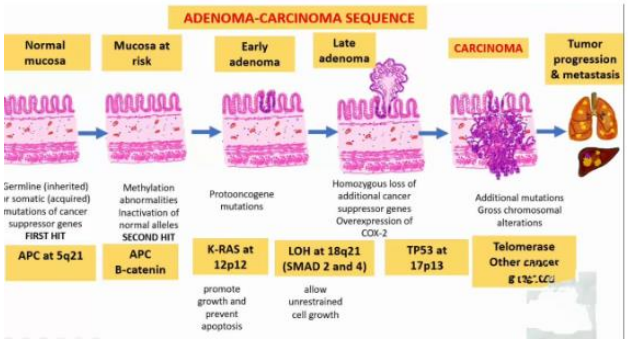
-4 Causes of gall stones(Mention Ca pancreas head must)
Causes of Gallstones:

1. **Increased Cholesterol Secretion**
   - Obesity, high-fat diet, pregnancy, oral contraceptives
2. **Bile Stasis**
   - Prolonged fasting, spinal cord injury, total parenteral nutrition (TPN)
3. **Increased Bilirubin Secretion**
   - Hemolysis (sickle cell anemia, thalassemia), liver diseases
4. **Infections**
   - Biliary infections (e.g., E. coli, parasites like Clonorchis sinensis)
5. **Reduced Bile Salts**
   - Ileal diseases (Crohn's), resection, or bile acid malabsorption
6. carcinoma of the pancreas (especially of the **head** of the pancreas) can cause **gallstones** by:

- **Biliary Obstruction** – Tumor compresses the common bile duct, leading to stasis of bile and stone formation.
- **Chronic Inflammation** – Biliary stasis increases the risk of pigment and mixed gallstones.

_gall stones types
_microscpic and macroscopic histological feautres of crohns disease
_hallmark of autoimmune gastritis histology
_most common type of stomach ca.....adeno.ca(intestinal type)
Ulcerative colitis picture identification
extraintestinal symptoms +most common
complications

13.Patho viva
-Adenoma carcinoma sequence...genes



APC (Early event) – Tumor suppressor gene mutation → adenoma formation
KRAS – Oncogene mutation → promotes adenoma growth
TP53 – Tumor suppressor mutation → transition from adenoma to carcinoma
SMAD4 (18q loss) – Disrupts TGF-β signaling → promotes cancer progression
PIK3CA – Activates AKT pathway → enhances tumor growth

CRC (98%) are Adenocarcinoma, Almost always arise in adenomatous polyps, 60-70 yrs, Adenomas are the presumed precursor lesion;

Aspirin and other NSAIDS exerts a protective effect

- Induction of apoptosis in tumour cells and inhibition of angiogenesis.
- Inhibition of cyclooxygenase-2.

➢ **Genetic** ab (APC/ beta catenin pathway {ADENOCARCINOMA SEQUENCE} OR MSI pathway {DEFECTS IN MISMATCH REPAIR OF DNA}),
➢ **Epigenetic** ab (Methylation induced gene silencing)

Adenoma-Carcinoma Sequence: Stepwise Process

The sequence shows how **colorectal cancer (CRC)** develops due to multiple genetic mutations.

1. **APC Gene Mutation (Early Event)**
    - **APC** is a **tumor suppressor gene**. Loss of both copies leads to uncontrolled cell growth.
    - **β-catenin** accumulates, enters the nucleus, and triggers genes like **MYC** and **cyclin D1**, promoting cell proliferation.
    - Found in **80% of sporadic CRC cases**.
2. **KRAS Mutation** (Intermediate Stage)
    - **KRAS** is an **oncogene**. Mutation locks it in an **active state**, continuously sending growth signals and blocking apoptosis.
    - Occurs in **50% of adenomas >1 cm and 50% of carcinomas**.
3. **18q21 Deletion (Late Stage)**
    - Loss of **SMAD4/DPC4** (part of the **TGF-β signaling pathway**), which normally inhibits cell growth.
    - Loss allows **unrestrained cell proliferation**.
4. **TP53 Mutation (Final Step)**
    - Found in **70-80% of CRC cases**.
    - Mutation in **TP53** (tumor suppressor gene) occurs late, enabling the transition to invasive carcinoma.

Second Pathway: Microsatellite Instability (MSI)—usually right sided CAs

- Caused by defective **DNA mismatch repair (MMR) genes**: **MLH1, MSH2, MSH6, PMS2**.
- Leads to **accumulation of mutations** in microsatellite regions, affecting genes like **TGF-β receptor and BAX** (promotes apoptosis).
- Associated with **HNPCC (Lynch Syndrome)** and has a **better prognosis**.

Carcinoid tumors; NE cells => Serotonin => smooth muscle stimulation => abdominal cramps, diarrhea, vasodilation causes flushing of skin and bronchospasm; **MC** tip of appendix, next ileum
-**Most common extra intestinal manifestation of IBD** (MC: ARTHRITIS), rest Erythema nodosum, uveitis, primary sclerosing cholangitis.
-**Role of H pylori in development of gastric cancer**...(repeated inflammations...intestinal metaplasia...dysplasia..) **H. pylori → Chronic gastritis → Atrophic gastritis → Intestinal metaplasia → Dysplasia → Gastric adenocarcinoma**
-**Most common type of gastric cancer**... **ADENOCARCINOMA** (intestinal type)
-Crohn's disease...characteristic morphological features

Crohn's Disease – Morphological Features

1. **Transmural inflammation**
2. **Skip lesions**
3. **Cobblestone appearance**
4. **Non-caseating granulomas**
5. **Fissures and fistulas**
6. **Strictures and fibrosis**
7. **Creeping fat**

Crohn's Disease vs Ulcerative Colitis (Pathoma Summary)

Feature	Crohn's Disease	Ulcerative Colitis (UC)
Location	Anywhere from **mouth to anus**	**Rectum** → Colon only
Inflammation	**Transmural** (full thickness)	**Mucosal and submucosal**
Gross Appearance	**Cobblestone mucosa**, skip lesions	**Pseudopolyps**, continuous
Complications	Fistulas, strictures, abscesses	Toxic megacolon, carcinoma
Histology	**Non-caseating granulomas**	**Crypt abscesses** with neutrophils
Symptoms	Right lower quadrant pain + diarrhea (non-bloody)	Left lower quadrant pain + **bloody diarrhea**
Smoking	**Worsens Crohn's**	**Protective in UC**

-Autoimmune gastritis... characteristic morphological features.

Autoimmune Gastritis – Morphological Features

1. **Atrophy of Gastric Mucosa**
   - Mainly affects the **body and fundus** (spares the antrum).
2. **Lymphocytic and Plasma Cell Infiltration**
   - Infiltration in the **lamina propria**.
3. **Loss of Parietal and Chief Cells**
   - Reduced **acid (HCl)** and **intrinsic factor** production → **pernicious anemia** (due to B12 deficiency).
4. **Intestinal Metaplasia**
   - Replacement of gastric epithelium with **goblet cells** (intestinal-type cells), **increasing cancer risk**.
5. **ECL Cell Hyperplasia**
   - Due to increased **gastrin** levels → may lead to **carcinoid tumors**.

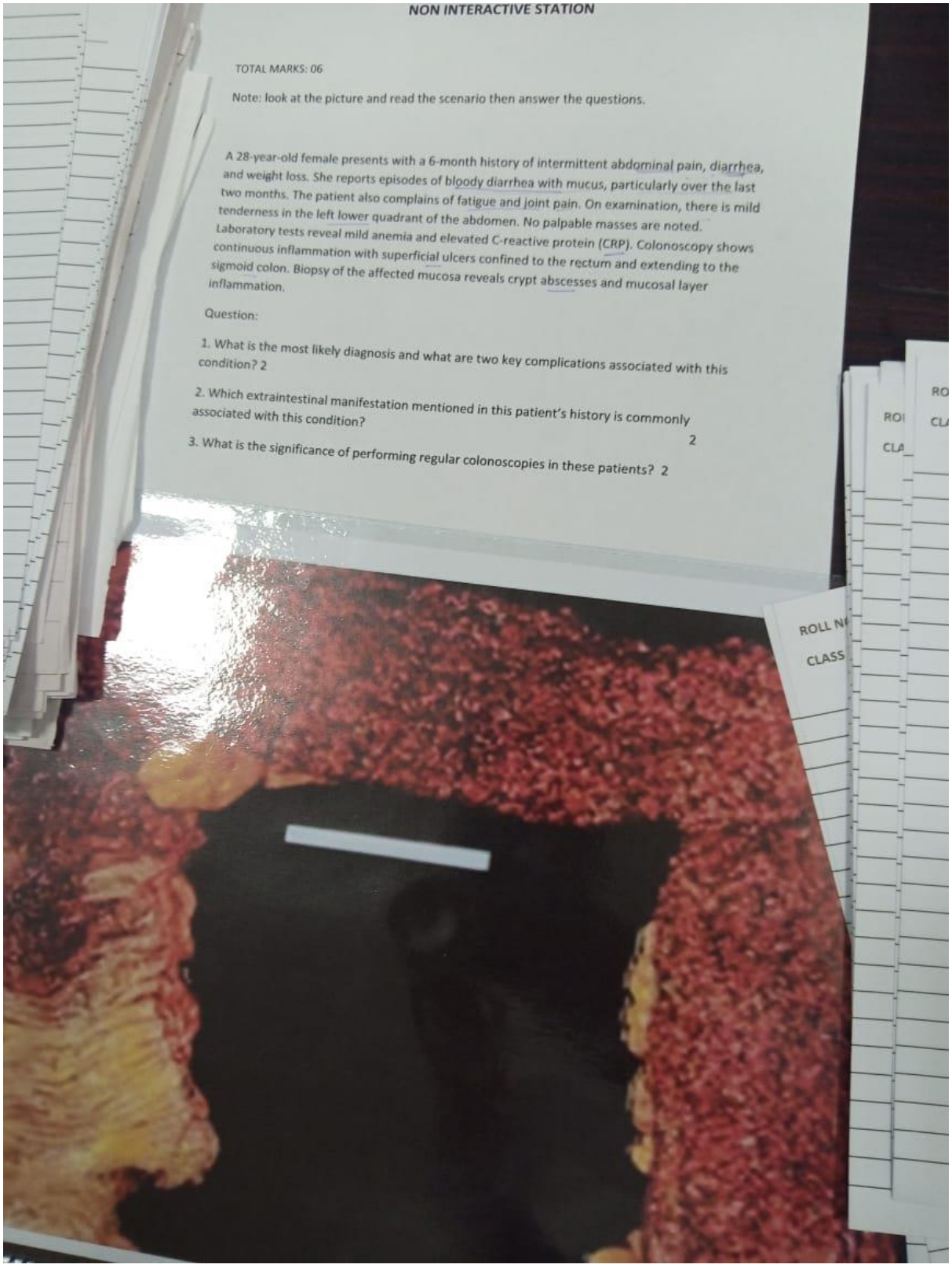
15.Pharma prescription...H.pylori peptic ulcer

16.Patho non-observed..
Ulcerative colitis
Most common extra intestinal manifestations:

- **Arthritis** (Peripheral and Axial) – Most common
- **Primary Sclerosing Cholangitis (PSC)**
- **Erythema Nodosum**
- **Uveitis**

Why regular colonoscopy is recommended

 High Risk of Colorectal Cancer: Long-standing UC (≥ 8–10 years) increases cancer risk.
 Early Detection of Dysplasia: Helps detect precancerous changes.
 Monitor Disease Progression: To assess inflammation and complications like strictures.



1. **ULCERATIVE COLITIS,**

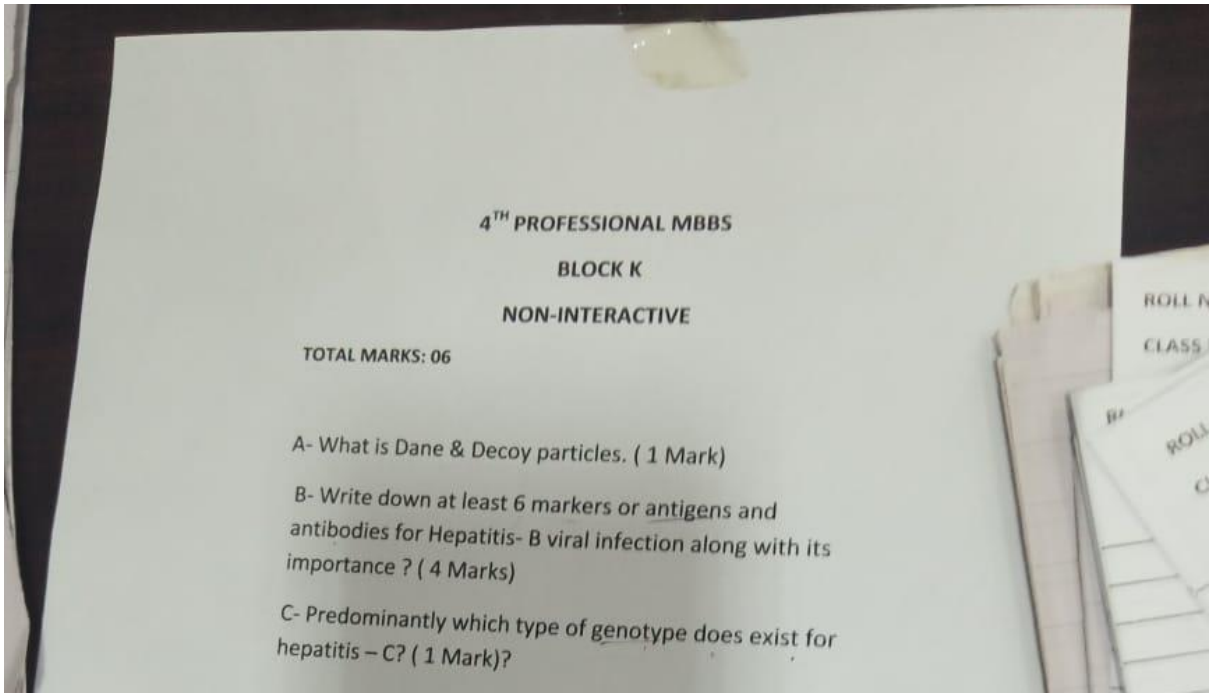
   Two Key Complications of Ulcerative Colitis

- **Toxic Megacolon:** Acute colonic distension with risk of perforation and sepsis.

- **Colorectal Cancer:** Increased risk, especially with long-standing disease (>8-10 years).

2. JOINT PAIN (ARTHRITIS)
3. **Significance of Performing Regular Colonoscopies**:

- Early detection of dysplasia or colorectal cancer
- Monitoring disease progression and inflammation

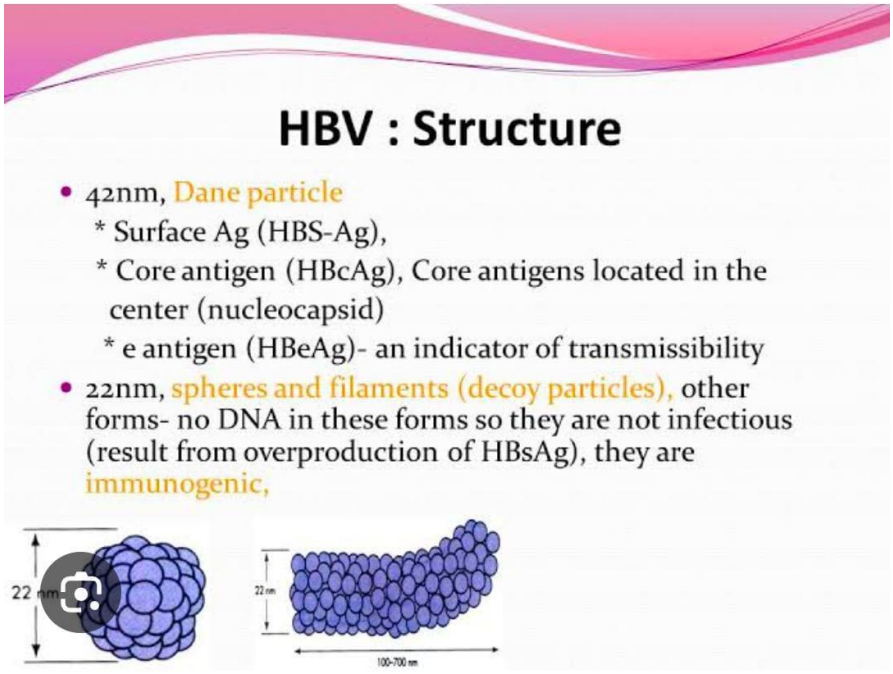


- **DANE** particles: Complete and infectious Hepatitis B virus (HBV) particles containing viral DNA, core, and envelope proteins.
- **Decoy** particles: Non-infectious, smaller particles made only of HBV surface proteins (no viral DNA), produced in excess to evade the immune system.

- **HBsAg**: Active infection.
- **Anti-HBs**: Immunity (recovery/vaccine).
- **HBeAg**: High infectivity.
- **Anti-HBe**: Low infectivity.
- **Anti-HBc (IgM/IgG)**: Past or current infection.
- **HBV DNA**: Viral load, infectivity, treatment response.

The **predominant HCV genotype** globally is **Genotype 1**.

In **Pakistan**, the most common is **Genotype 3**.



Achalasia Definition

Achalasia is a **motility disorder** of the esophagus caused by **failure of the lower esophageal sphincter (LES) to relax** and **loss of esophageal peristalsis**, leading to difficulty swallowing, regurgitation, and chest pain.

Types of Achalasia

1. **Primary Achalasia** – No known cause, loss of nerve cells in the esophagus.
2. **Secondary Achalasia** – Due to other diseases (e.g., **Chagas disease**, cancer).
3. **Vigorous Achalasia** – Less severe, strong esophageal contractions.
4. **End-Stage Achalasia** – Advanced form, esophagus becomes very dilated.

Main Features

- Difficulty swallowing (solids and liquids)
- Food regurgitation
- Chest pain
- Weight loss
- Risk of cancer if untreated

Diagnosis of Achalasia

1. **Barium Swallow** – Bird's beak appearance.
2. **Manometry** – Gold standard; shows incomplete LES relaxation and no peristalsis.
3. **Endoscopy** – To rule out secondary causes like cancer.

Treatment of Achalasia

1. **Lifestyle Changes** – Eat slowly, avoid large meals.
2. **Medications** – Calcium channel blockers, nitrates (temporary relief).
3. **Botulinum Toxin Injection** – For temporary relief (elderly or high-risk patients).
4. **Pneumatic Dilation** – Stretching the LES with a balloon.
5. **Heller Myotomy** – Surgery to cut LES muscles.
6. **Peroral Endoscopic Myotomy (POEM)** – Minimally invasive endoscopic surgery.

Difference Between H. Pylori Gastritis and Autoimmune Gastritis

Feature	H. Pylori Gastritis	Autoimmune Gastritis
Cause	Infection with **H. pylori**	Autoantibodies against parietal cells and intrinsic factor
Location	**Antrum** (initially)	**Body and Fundus** of the stomach
Gastric Acid	Increased (early), decreased (late)	Decreased (hypochlorhydria)
Risk of Cancer	**Gastric adenocarcinoma** and MALT lymphoma	**Gastric adenocarcinoma**
Associated Diseases	Peptic ulcer disease, MALT lymphoma	Pernicious anemia, other autoimmune diseases (e.g., thyroiditis)
Histology	Neutrophil infiltration, lymphoid aggregates	Lymphocytes, gland atrophy, intestinal metaplasia
Antibodies	None	Anti-parietal cell, anti-intrinsic factor antibodies
Treatment	Antibiotics and proton pump inhibitors	B12 supplementation (lifelong)

Types of Gastric Cancer

1. **Intestinal Type**
   - Well-differentiated, glandular structures.
   - Linked to chronic gastritis (H. pylori infection).
   - Common in high-risk populations.
2. **Diffuse Type**
   - Poorly differentiated, signet ring cells.
   - No gland formation, infiltrates stomach wall (linitis plastica).
   - Worse prognosis.

Genes Associated with Gastric Cancer

1. **CDH1** (E-cadherin gene) – Diffuse type
2. **TP53** – Both types (tumor suppressor gene)
3. **APC** – Intestinal type (linked to familial adenomatous polyposis)
4. **HER2/neu** – Intestinal type (overexpression in some cases)
5. **KRAS** – Intestinal type

MSI (Microsatellite instability) – Intestinal type, better prognosis

MEDICINE

- Ascites diagnosis..
- 3 clinical features of ascities
- Drugs given for ascite(potassium sparing diuretics, furesomide and beta blocker[carvidolol])
- Difference betwen mild moderate and intense ascties or what are they
- Acsties can occurs becausr of?

Ascites Diagnosis

Ascites is typically diagnosed through **clinical examination** and **imaging**:

- **Physical examination**: Percussion may reveal shifting dullness and a fluid wave, which are suggestive of fluid in the abdominal cavity.
- **Ultrasound**: Confirms the presence and amount of fluid.
- **Paracentesis**: Analysis of ascitic fluid can help identify the cause (e.g., infection, malignancy) and includes measurements of serum-ascites albumin gradient (SAAG) to determine if the ascites is due to portal hypertension.

3 Clinical Features of Ascites

1) **Abdominal distension**: Swelling due to fluid accumulation.
2) **Shifting dullness**: Dullness on percussion that shifts when the patient changes position.
3) **Shortness of breath**: Due to pressure on the diaphragm from excess fluid.

Drugs for Ascites

1) Potassium-Sparing Diuretics: Spironolactone is commonly used to reduce fluid while preserving potassium levels.
2) Loop Diuretics: Furosemide helps increase fluid excretion.
3) Beta Blockers: Carvedilol can help reduce portal hypertension, thereby reducing ascites in liver disease.

Ascites Severity

1) Mild Ascites: Only detectable via ultrasound, not visually apparent.
2) Moderate Ascites: Visible abdominal distension, noticeable on physical examination.
3) Severe Ascites (Tense Ascites) : Large amount of fluid causing significant abdominal distension and discomfort, potentially affecting breathing.

Causes of Ascites

- Liver cirrhosis (most common cause, due to portal hypertension).
- Heart failure.
- Kidney disease (e.g., nephrotic syndrome).
- Malignancy (cancer-related ascites).
- Infections (e.g., tuberculosis, peritonitis).

Medicine ascites history examination clinical features cause management

Surgery obstructive jaundice history examination clinical features causes mechanism

Important Points for Jaundice History

1. **Presenting Complaint**
    - Onset, duration, and progression of yellow discoloration.
2. **Associated Symptoms**
    - **General**: Fatigue, weight loss, fever.

    - **GI Symptoms**: Nausea, vomiting, abdominal pain, altered bowel habits (pale stools, dark urine).
    - **Pruritus** (itching)
3. **Past Medical History**
    - Liver disease, gallstones, hepatitis, recent infections, blood transfusions.
4. **Drug History**
    - Recent medications (e.g., anti-tubercular, paracetamol overdose).
5. **Social History**
    - Alcohol consumption, travel history, sexual history, intravenous drug use.
6. **Family History**
    - Inherited liver diseases (e.g., Wilson's disease, hemochromatosis).
7. **Occupational History**
    - Exposure to hepatotoxins, healthcare worker.
8. **Differentiation of Types**
    - **Pre-hepatic**: Hemolysis (e.g., dark urine, no pale stools).
    - **Hepatic**: Viral hepatitis, autoimmune liver disease.
    - **Post-hepatic**: Obstructive jaundice (e.g., pale stools, itching).

Obstructive Jaundice

Occurs due to **blockage in bile flow** from the liver to the duodenum.

Causes

1. **Intrahepatic Causes**
    - Primary biliary cholangitis
    - Primary sclerosing cholangitis
    - Liver metastases
2. **Extrahepatic Causes**
    - Gallstones
    - Cholangiocarcinoma
    - Pancreatic cancer
    - Biliary atresia (in children)
    - Strictures (post-surgery or inflammation)

Clinical Features

- **Yellow discoloration** of skin and eyes
- **Pruritus** (intense itching)
- **Dark urine, pale stools**
- Right upper quadrant pain (if due to gallstones)
- Weight loss and anorexia (if malignant cause)

Investigations

- **LFTs**: **Elevated ALP, GGT, bilirubin** (predominantly conjugated)
- **Ultrasound**: First-line for detecting biliary obstruction
- **CT/MRCP/ERCP**: For detailed imaging and diagnosis

Management

1. **Gallstones**: **ERCP**, cholecystectomy
2. **Malignancy**: Surgery, stenting, chemotherapy
3. **Biliary strictures**: Dilatation, stenting

ERCP (Endoscopic Retrograde Cholangiopancreatography)

Definition:
A combined endoscopic and radiologic procedure used to diagnose and treat **biliary** and **pancreatic duct disorders**.

Indications

1. **Biliary obstruction** (gallstones, strictures, tumors)
2. **Cholangitis**
3. **Chronic pancreatitis**
4. **Bile leak** or fistula
5. **Pancreatic duct abnormalities**

Procedure Steps

1. Endoscope is passed through the mouth into the **duodenum**.
2. Contrast is injected into the **biliary and pancreatic ducts**.
3. X-rays are taken to visualize abnormalities.

Therapeutic Uses

- **Stone removal** (common bile duct stones)
- **Stent placement** (malignant obstruction)
- **Dilate strictures**
- **Drain abscesses or pseudocysts**

Complications

1. **Pancreatitis** (most common)
2. Perforation
3. Bleeding
4. Infection (cholangitis)

Liver cirrhosis

Types of Cirrhosis

1. **Alcoholic Cirrhosis**
2. **Post-viral Cirrhosis (Hepatitis B, C)**
3. **Biliary Cirrhosis** (Primary and Secondary)
4. **Cardiac Cirrhosis**
5. **Metabolic Cirrhosis** (e.g., Wilson's disease, Hemochromatosis)

Clinical Features

1. **Early Symptoms**
    - Fatigue
    - Anorexia
    - Weight loss
    - Nausea/vomiting
2. **Advanced Symptoms**
    - Jaundice
    - Ascites
    - Hepatomegaly
    - Splenomegaly
    - Spider angiomas
    - Palmar erythema
    - Caput medusae
3. **Complications**
    - Portal hypertension
    - Hepatic encephalopathy
    - Esophageal varices
    - Hepatocellular carcinoma

Causes

1. **Alcohol consumption** (most common)
2. **Chronic hepatitis B and C infection**
3. **Non-alcoholic steatohepatitis (NASH)**
4. **Autoimmune hepatitis**
5. **Primary and secondary biliary cirrhosis**
6. **Metabolic diseases** (Wilson's disease, Hemochromatosis)

Hallmark Feature

- **Diffuse fibrosis with regenerative nodules**

Morphological Features

1. **Microscopic**
    - Bridging fibrosis
    - Regenerative nodules surrounded by fibrous septa
    - Loss of normal liver architecture
2. **Macroscopic**
    - Small, nodular liver in advanced stages (micronodular/macronodular)
    - Firm and shrunken appearance

Consequences of Portal Hypertension

1. Ascites
2. Congestive Splenomegaly
3. Esophageal Varices
4. Caput Medusae
5. Rectal Varices (Hemorrhoids)
6. Hepatic Encephalopathy

Liver Function Tests (LFTs)

1. **Hepatocellular Injury:**
    - **ALT (Alanine Aminotransferase)** – Specific for liver injury
    - **AST (Aspartate Aminotransferase)** – Elevated in liver, muscle, heart injury
2. **Cholestasis (Biliary Injury):**
    - **Alkaline Phosphatase (ALP)** – Elevated in bile duct obstruction
    - **Gamma-Glutamyl Transferase (GGT)** – Confirms biliary origin
3. **Synthetic Function:**
    - **Albumin** – Low in chronic liver disease
    - **Prothrombin Time (PT)** – Prolonged in severe liver dysfunction
4. **Excretory Function:**
    - **Bilirubin (Total, Direct, Indirect)** – Elevated in jaundice

Key LFT Findings

1. **ALT > AST**
    - Seen in **Viral Hepatitis** and **Non-Alcoholic Fatty Liver Disease (NAFLD)**
2. **AST > ALT (2:1 ratio or more)**
    - Characteristic of **Alcoholic Liver Disease**
3. **Alkaline Phosphatase (ALP)**
    - Elevated in **Obstructive Liver Disease (Cholestasis)**, **Bone Disease**, and **Primary Biliary Cirrhosis**
4. **Gamma-Glutamyl Transferase (GGT)**
    - Elevated in **Alcoholic Liver Disease** and **Cholestasis**
5. **Hyperbilirubinemia**
    - **Unconjugated**: **Hemolysis, Crigler-Najjar, Gilbert Syndrome**
    - **Conjugated**: **Obstructive Jaundice, Dubin-Johnson, Rotor Syndrome**
6. **Prolonged Prothrombin Time (PT)**
    - Indicates **Liver Failure**, **Vitamin K Deficiency**, or **Severe Hepatocellular Damage**

Diagnosis of Cholestasis

1. **Clinical Features:**
    - **Jaundice**, pruritus (itching), dark urine, pale stools, fatigue.
2. **Laboratory Tests:**
    - **Elevated Alkaline Phosphatase (ALP)** and **Gamma-Glutamyl Transferase (GGT)** → hallmark of cholestasis
    - **Conjugated hyperbilirubinemia**
    - **Elevated transaminases (ALT, AST)** in some cases
3. **Imaging Studies:**
    - **Abdominal Ultrasound:** First-line test to detect **bile duct dilation**, stones, or tumors.
    - **Magnetic Resonance Cholangiopancreatography (MRCP):** Non-invasive, detailed view of biliary tree.
    - **Endoscopic Retrograde Cholangiopancreatography (ERCP):** Diagnostic and therapeutic.
4. **Liver Biopsy:**
    - When the cause of intrahepatic cholestasis is unclear (e.g., primary biliary cholangitis or drug-induced liver injury).

Rule of 2s for Meckel's Diverticulum:

1. **2 inches** in length
2. Located **2 feet** from the ileocecal valve
3. Occurs in **2%** of the population
4. **2 types** of ectopic tissue (gastric and pancreatic)
5. Commonly presents before the age of **2 years**
6. **2 times** more common in males

17.Medicine
30 year old male..
Mild scleral icterus..
Especially after fasting
Abdomen soft, non-tender with no hepato or spleenomegaly
No weight loss or change in appetite
No known history of liver disease
Same disease in family
Mild isolated elevation of _unconjugated bilirubin_
Liver enzymes (ALT, AST) and other LFTs are normal
Benign condition!
genetic deficiency in the enzyme UDP-glucuronosyltransferase
Treatment.. counselling..
Diagnosis: Gilbert's Syndrome

Key Features:

- **Mild scleral icterus**, especially **after fasting or stress**.
- **Isolated elevation of unconjugated bilirubin** (≤ 3 mg/dL).
- **Normal liver enzymes (ALT, AST)** and other LFTs.
- **No hepatosplenomegaly, weight loss, or appetite change**.
- **Family history** of similar condition (autosomal recessive inheritance).

Cause:

- Reduced activity of **UDP-glucuronosyltransferase (UGT1A1)** → impaired conjugation of bilirubin.

Management:

- Benign condition, no treatment needed. Avoid prolonged fasting and dehydration.

Here are some **examinations** related to the **GIT** and **Hepatobiliary** systems that may be tested in an OSCE:

GIT Examinations:

1. **Abdominal Inspection**:
   - Look for scars, distension, masses, hernias, or visible peristalsis.
2. **Abdominal Palpation**:
   - Light palpation: Tenderness, guarding, or masses.
   - Deep palpation: Liver, spleen, and kidney palpation.

3. **Percussion**:
    - Check for liver span, tympany, dullness, and shifting dullness (for ascites).
4. **Auscultation**:
    - Bowel sounds: Normal, hyperactive, or absent.
    - Bruits over the abdomen (renal artery stenosis or aortic aneurysm).
5. **Rectal Examination**:
    - Check for masses, tenderness, and blood.
6. **Test for Ascites**:
    - Fluid wave test and shifting dullness.
7. **Abdominal Reflex**:
    - Check for abdominal reflexes (response to stimulation).
8. **Hernia Examination**:
    - Palpate inguinal, femoral, and umbilical regions.

Hepatobiliary Examinations:

1. **Liver Palpation**:
    - Check for liver enlargement (hepatomegaly), tenderness, or a firm edge.
2. **Murphy's Sign**:
    - Check for cholecystitis by palpating the gallbladder area and asking the patient to take a deep breath.
3. **Spleen Palpation**:
    - Check for splenomegaly (enlarged spleen).
4. **Percussion for Liver Size**:
    - Percuss to determine the liver size and any signs of enlargement.
5. **Jaundice Assessment**:
    - Inspect the sclera and skin for yellowing.
6. **Cullen's and Grey-Turner's Signs**:
    - Assess for signs of intra-abdominal bleeding (blue discoloration around the umbilicus or flanks).

18.Surgery station
Right iliac fossa pain...nausea vomiting ..20 year old female
D/Ds ...
Appendicitis, ectopic pregnancy, ruptured ovarian cyst, kidney stones...blood in urine
Investigations
Approach for appendectomy...larpscopic or incision?
If 60 years old female presented with right iliac fossapain....malignancy...Ca Cecum!!!!!!
What is meant by subacute and acute intestinal obstruction
Which appendectomy is preferred in children(laparoscopic is preferred over open one)

Differential Diagnoses for Right Iliac Fossa Pain (20-year-old Female)

1. **Acute Appendicitis** (most common)
2. **Ectopic Pregnancy** (do urine β-hCG)
3. **Ovarian Cyst Rupture or Torsion**
4. **Pelvic Inflammatory Disease (PID)**
5. **Mesenteric Adenitis**
6. **Crohn's Disease (Ileitis)**

Investigations

1. **Blood Tests**: CBC (raised WBC count), CRP
2. **Urine Analysis**: Rule out UTI, β-hCG for pregnancy
3. **Ultrasound Abdomen and Pelvis**: First-line for appendicitis
4. **CT Scan** (if diagnosis is uncertain)

Approach for Appendectomy

- **Laparoscopic Appendectomy** (Preferred)
    - Less postoperative pain, quicker recovery, fewer complications.
- **Open Appendectomy** (If perforation or generalized peritonitis)

Right Iliac Fossa Pain in a 60-Year-Old Female – Differential Diagnosis

1. **Colorectal Cancer**
2. **Diverticulitis**
3. **Appendicitis** (less common at this age)
4. **Ischemic Colitis**
5. **Ovarian or Pelvic Mass**

Subacute vs. Acute Intestinal Obstruction

- **Acute Obstruction**: Sudden onset, severe symptoms (abdominal pain, vomiting, no bowel movement, distension).
- **Subacute Obstruction**: Gradual onset, less severe symptoms, partial obstruction with some bowel movement.

Preferred Appendectomy in Children

- **Laparoscopic Appendectomy** is preferred due to:
    - Less postoperative pain
    - Quicker recovery
    - Fewer wound infections

Psoas abscess it's causes
McBurney point

Psoas Abscess – Causes

2. **Primary (Hematogenous Spread)** – Common in immunocompromised or malnourished individuals.
    - **Staphylococcus aureus** (most common pathogen)
3. **Secondary (Spread from Nearby Structures)** – More common.
    - **Appendicitis**
    - **Crohn's disease**
    - **Osteomyelitis of spine (Pott's disease)**
    - **Diverticulitis**
    - **Infected renal or pelvic organs**

McBurney's Point

- Located **two-thirds of the way from the umbilicus to the right anterior superior iliac spine (ASIS)**.

- **Tenderness at McBurney's point** is a key sign of **acute appendicitis**.
- surgery.....
- gillbert syndrome senario
- common features.....jaundice'yellow discoloration of eyes and urine
- everything else normal of liver tests etc
- only unconjugated billirubin is high
- enzyme involved??¿?
- senario.....
- RIF pain and tenderness
- name the different D/Ds
- what is meant by
- subacute and acute
- intestinal obstruction???
- which appendectomy is preferred in children?? (laproscopic is preferred over open one)

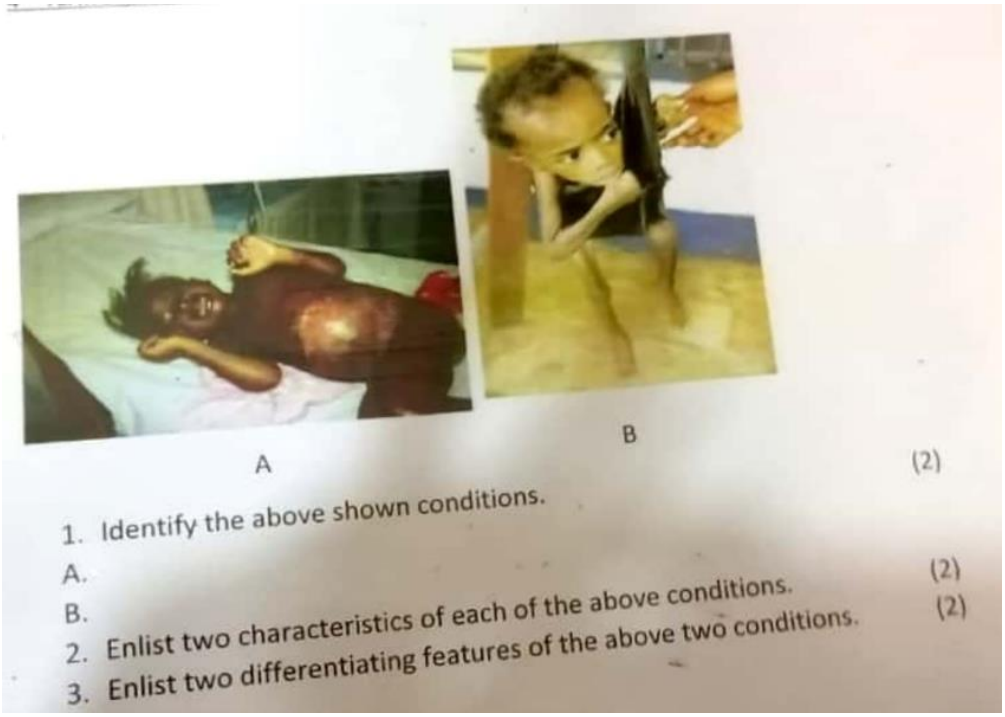
C. MED

SGDS AND MDGS RELATED TO HEALTH?

SGDS AND MDGS RELATED TO ENVIORNMENT= 6, 7, 13

What is safe food? food that is free from contaminants (biological, chemical, and physical) and is handled, prepared, and stored in a way that minimizes the risk of foodborne illnesses.

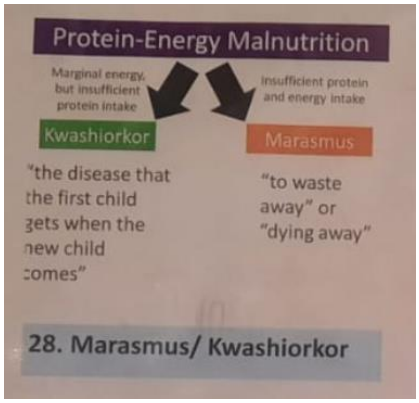
10.Community non-observed
Marasmus and kawashiokor.

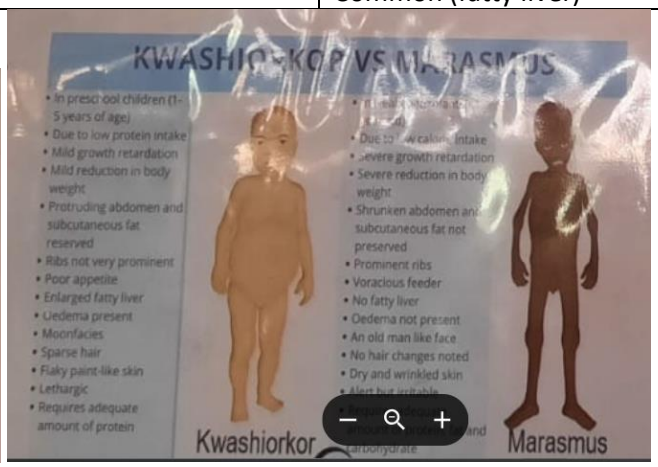


Marasmus vs. Kwashiorkor

Feature	Marasmus	Kwashiorkor

Cause	Severe **calorie deficiency**	Severe **protein deficiency**
Age Group	< 1 year	1–5 years
Body Weight	Markedly reduced (<60% of expected)	Moderately reduced (60–80%)
Edema	Absent	Present (pitting edema)
Muscle Wasting	Severe	Mild to moderate
Subcutaneous Fat	Absent	May be preserved
Hair Changes	Rare	Common (thin, sparse, reddish hair)
Skin Changes	Absent	Common (darkening, peeling)
Mental State	Alert but irritable	Apathy, lethargy
Hepatomegaly	Rare	Common (fatty liver)





11.Community non-observed
Health awareness Karni hai in rural area...no health facility nearby..about awareness of diabetes mellitus in uneducated women
-Write strategies to promote awareness
-common channel?
-which theory or model of health education is used?
-two teaching methods

Strategies to Promote Awareness about Diabetes Mellitus in Rural Areas:

1. **Community Outreach Programs**:
   - Organize **door-to-door visits** by trained community health workers to provide basic information and handouts about diabetes.
2. **Use of Local Media (Radio, TV)**:
   - Broadcast educational programs on **local radio stations** to reach a larger audience. Include simple explanations of diabetes, its symptoms, and prevention tips.
3. **Workshops and Demonstrations**:
   - Conduct **group sessions** where community health workers demonstrate basic prevention and management techniques (e.g., healthy diet, physical activity).
4. **Collaboration with Religious Leaders**:
   - Partner with **local religious leaders** to spread the message during community gatherings (e.g., Friday prayers).
5. **Interactive Health Fairs**:
   - Hold **health fairs** where free screenings, educational materials, and demonstrations are provided.
6. **Peer Education**:

- Train **local women** as health ambassadors to educate their peers within their social groups and families.

Common Channel:

- **Community Health Workers**: These individuals can serve as the **primary channel** for delivering information, utilizing personal interaction in homes, villages, and community centers.

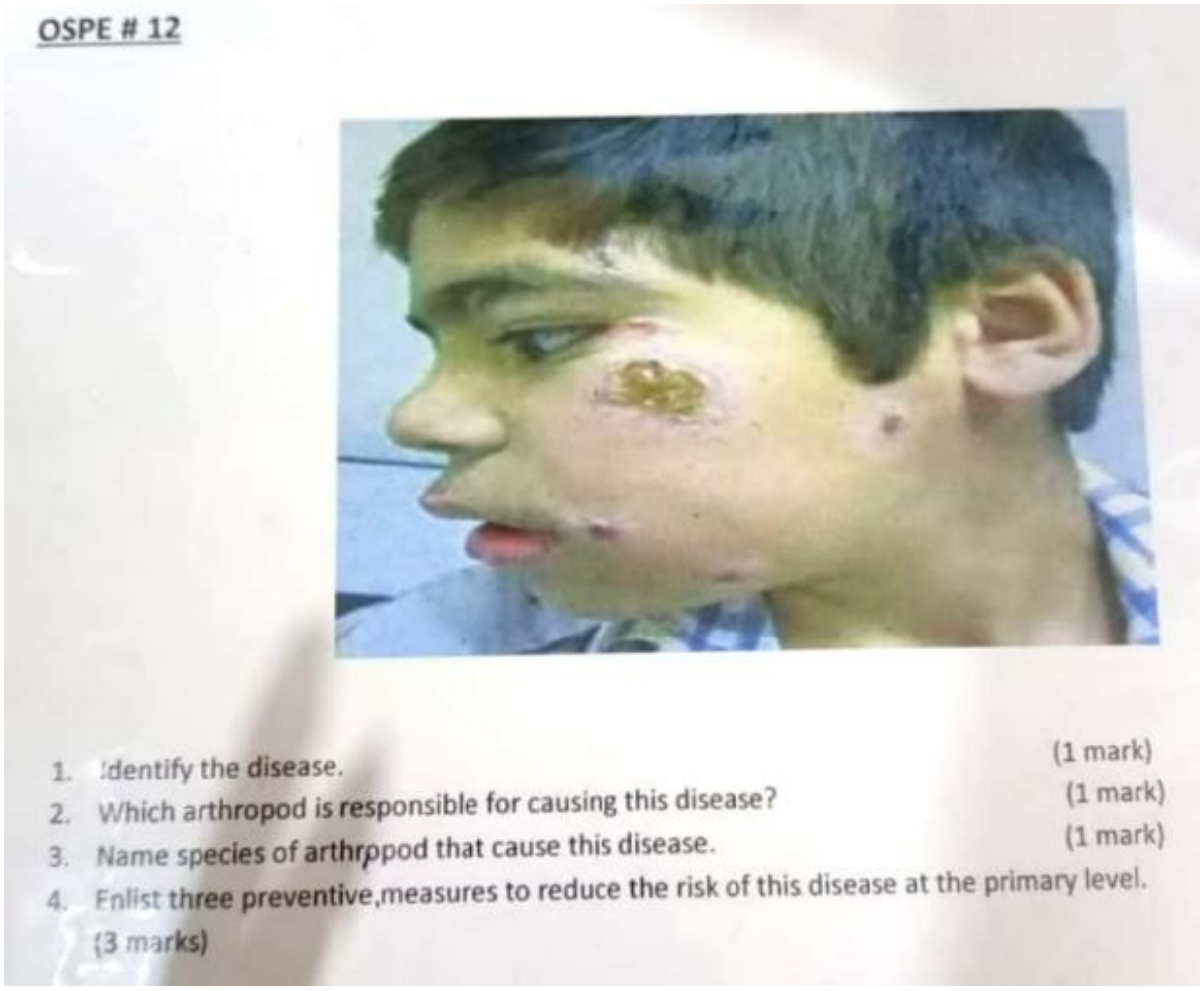
Health Education Theory/Model:

- **Health Belief Model**: This model is effective in rural areas because it emphasizes the **perceived susceptibility**, **severity**, and **benefits** of taking action, which can motivate uneducated women to change their behaviors.

Teaching Methods:

1. **Storytelling/Role Play**: Use **storytelling** or **role-play** techniques to explain the effects of diabetes and its management in simple, relatable terms.
2. **Visual Aids**: Use **posters, charts, and illustrations** showing the importance of healthy eating, exercise, and regular health check-ups to reinforce the message.

2)leishmania case
_identify
_sand fly(transmission) and its biological name
_prevention



Leishmania Case

1. **Identification:**
    - **Causative Agent:** Leishmania species (protozoan parasite).
    - **Types of Leishmaniasis:**
        - **Cutaneous Leishmaniasis**: Skin ulcers or sores.
        - **Visceral Leishmaniasis (Kala-azar)**: Fever, weight loss, hepatosplenomegaly, anemia.
        - **Mucocutaneous Leishmaniasis**: Nasal and oral mucosal destruction.
2. **Sandfly (Transmission)**
    - **Vector:** Phlebotomus (Old World) or Lutzomyia (New World).
    - **Biological Name: Phlebotomus argentipes** (most common vector for visceral leishmaniasis).
    - **Mode of Transmission:** Sandflies inject **promastigotes** into human skin during a blood meal.
3. **Prevention:**
    - **Vector Control:** Use insecticide-treated nets, insect repellents, and indoor spraying.
    - **Avoid Sandfly Bites:** Wear protective clothing and use bed nets in endemic areas.
    - **Environmental Management:** Eliminate breeding sites (damp, dark places).

- **Early Detection and Treatment:** Prompt diagnosis and treatment of infected individuals.

3)my foodplate labelling,components and definition

20.
-Housefly

-Biological name (Musca domestica)

-Mechanism of disease transmission by it
-4 diseases caused by it
-Measures to prevent the spread

House Fly (Musca domestica)

Mechanism of Disease Transmission:

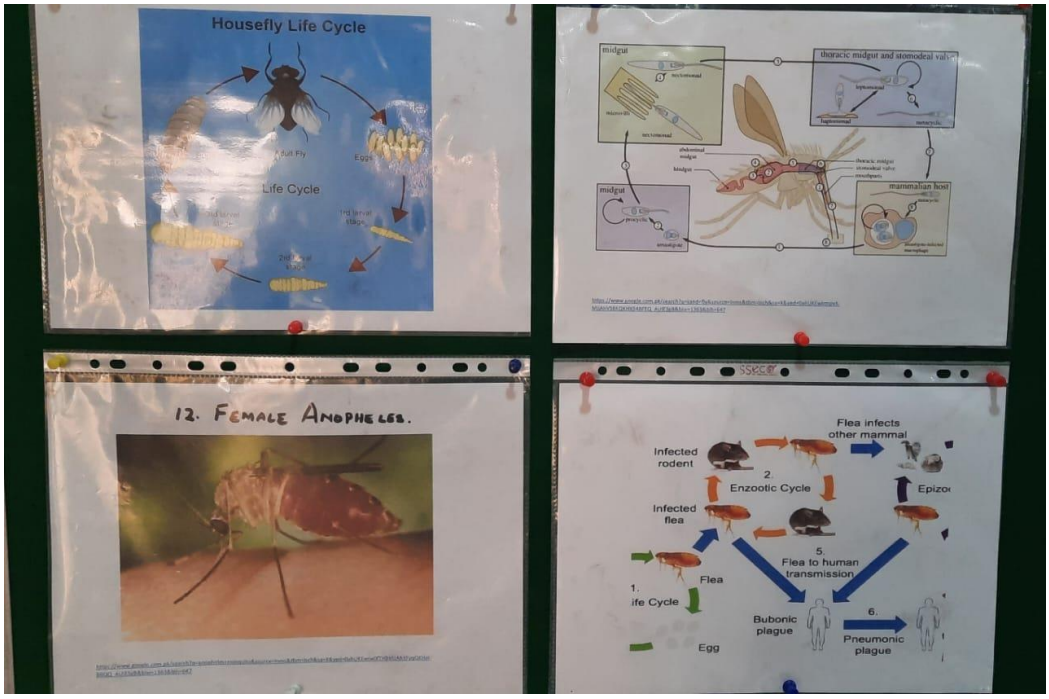
- **Mechanical** vector: **Picks up pathogens** on body surface, legs, and mouthparts from contaminated material (feces, garbage) → **transfers to food and surfaces**.
- **Regurgitation and defecation** on food also spread infections.

4 Diseases Caused:

1. **Typhoid fever**
2. **Cholera**
3. **Dysentery** (Shigellosis)
4. **Trachoma**

Measures to Prevent Spread:

1. **Proper waste management** – Cover garbage, dispose of waste regularly.
2. **Fly-proof homes** – Use window screens and covers.
3. **Food safety** – Cover food and maintain hygiene.
4. **Insecticides and fly traps** – Control fly population.



Aedes aegypti (Yellow Fever Mosquito)

Q399: Disease & Control

- **Diseases Caused**:
    - **Dengue fever**
    - **Zika virus**
    - **Chikungunya**
    - **Yellow fever**
- **Control Measures**:
    1. **Eliminate mosquito breeding sites** (stagnant water)
    2. **Use insecticides** and larvicides
    3. **Wear protective clothing** and use **mosquito repellents**
    4. **Install mosquito nets and screens**

OSPE # 7

A mother brought her four years child to pediatric OPD of HMC with one-week history of perianal itching. She noted that the itching occurs mostly at night. On examination the perianal area was red, irritated and excoriated. Diagnosis with Pin worm infestation was made. Treatment given and mother was warned about possible re infection.

1. Write the confirmatory test for diagnosis of pinworms infestation? [1]
2. Describe four preventive measures for pin worm infestation? [2]
3. Describe differences between reinfection and autoinfection? [2]
4. What is the treatment of choice for pinworms infestation? [1]

Identify the Model

Autoclave – A device used for **steam sterilization** under pressure.

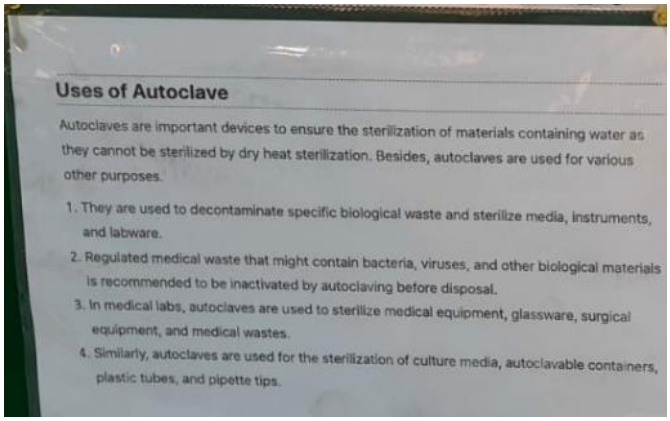
Types of Items for Autoclave Use

1. **Surgical Instruments**
2. **Glassware** (e.g., lab flasks, test tubes)
3. **Culture Media** (for microbiology)
4. **Dressings, gowns, and drapes**
5. **Rubber Items** (catheters, gloves)

Note: Not suitable for heat-sensitive items like plastics or electronics.

Steps of Instrument Sterilization (Autoclave Process)

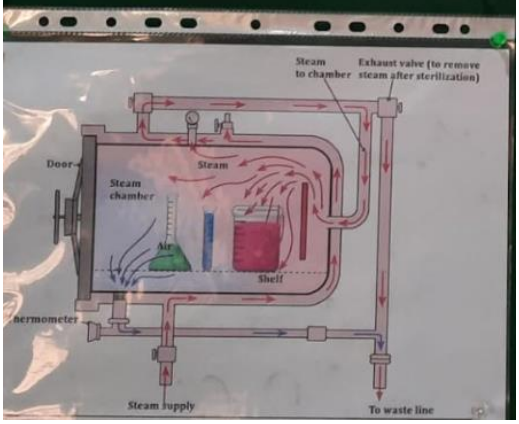
1. **Cleaning** – Wash instruments to remove debris and organic matter.
2. **Packaging** – Wrap in sterilization paper or pouches.
3. **Loading the Autoclave** – Place items to allow steam circulation.
4. **Sterilization Cycle**:
    - **121°C at 15 psi for 15–20 minutes** (standard cycle)
5. **Drying and Cooling** – Ensure items are dry before use.
6. **Storage** – Store sterilized items in a clean, dry area.



Uses of Autoclave

Autoclaves are important devices to ensure the sterilization of materials containing water as they cannot be sterilized by dry heat sterilization. Besides, autoclaves are used for various other purposes.

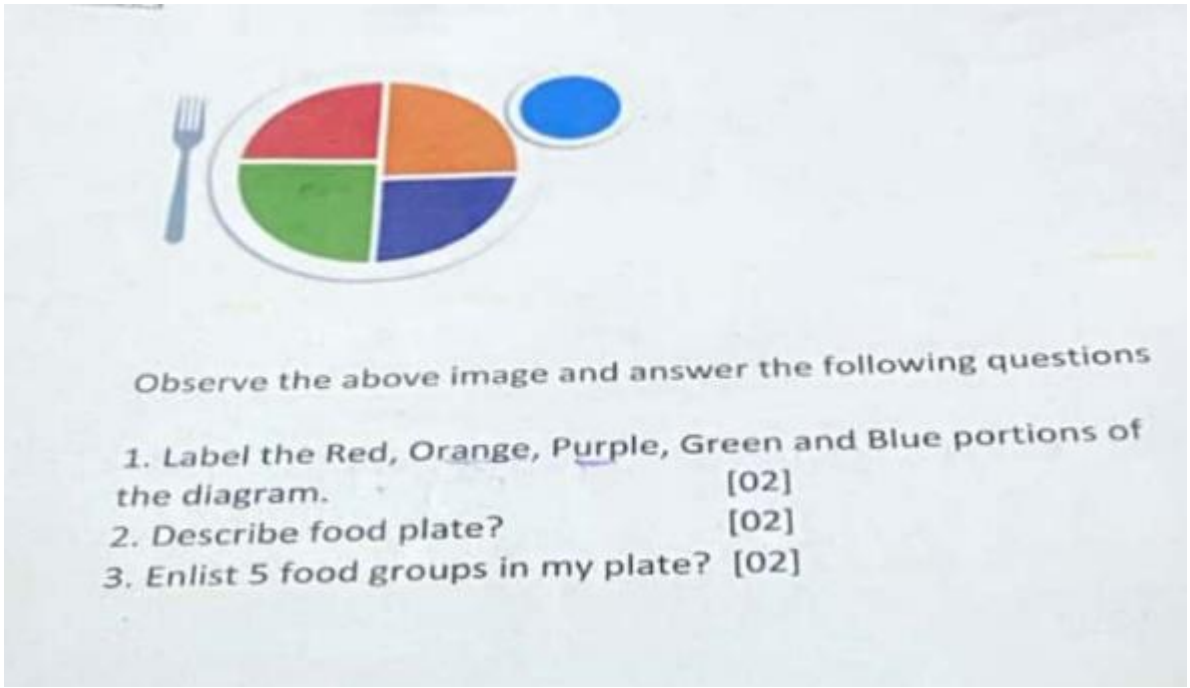
1. They are used to decontaminate specific biological waste and sterilize media, instruments, and labware.
2. Regulated medical waste that might contain bacteria, viruses, and other biological materials is recommended to be inactivated by autoclaving before disposal.
3. In medical labs, autoclaves are used to sterilize medical equipment, glassware, surgical equipment, and medical wastes.
4. Similarly, autoclaves are used for the sterilization of culture media, autoclavable containers, plastic tubes, and pipette tips.



OSPE BLOCK K

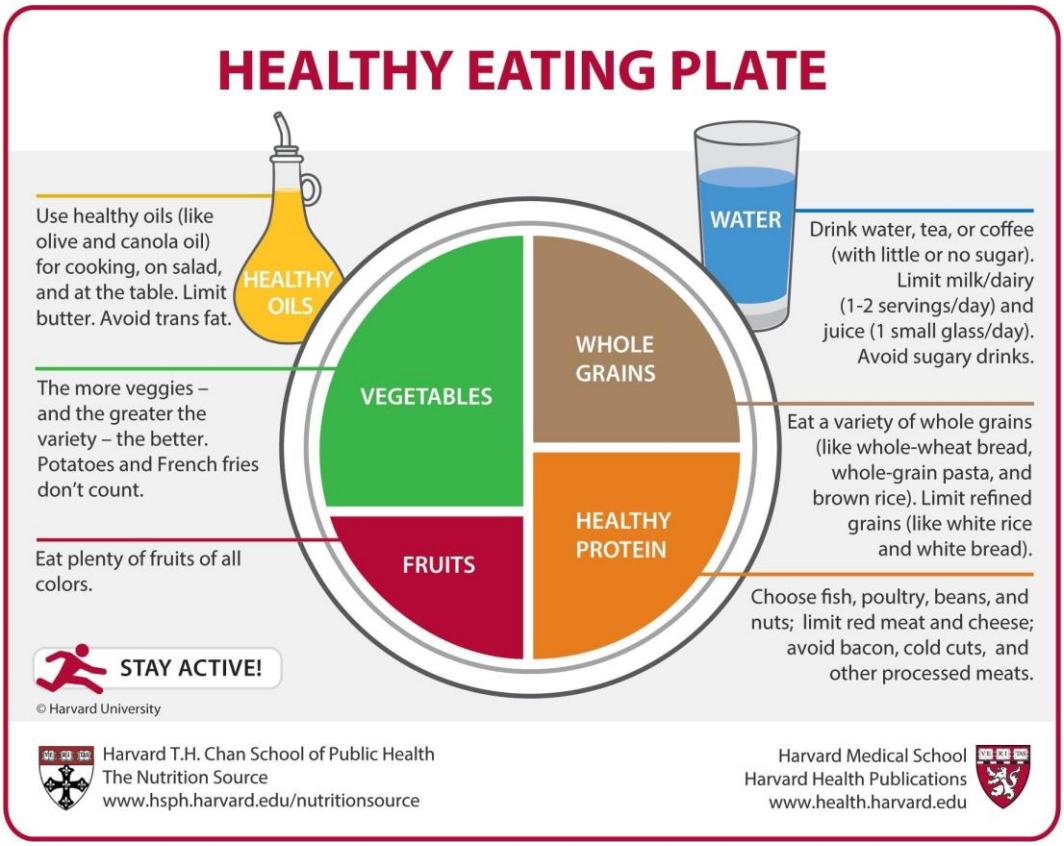
1.Community non-observed...foodplate

- The food plate is a **visual representation of a balanced diet**, focusing on proportions of food groups for healthy eating.



Observe the above image and answer the following questions

1. Label the Red, Orange, Purple, Green and Blue portions of the diagram. [02]
2. Describe food plate? [02]
3. Enlist 5 food groups in my plate? [02]

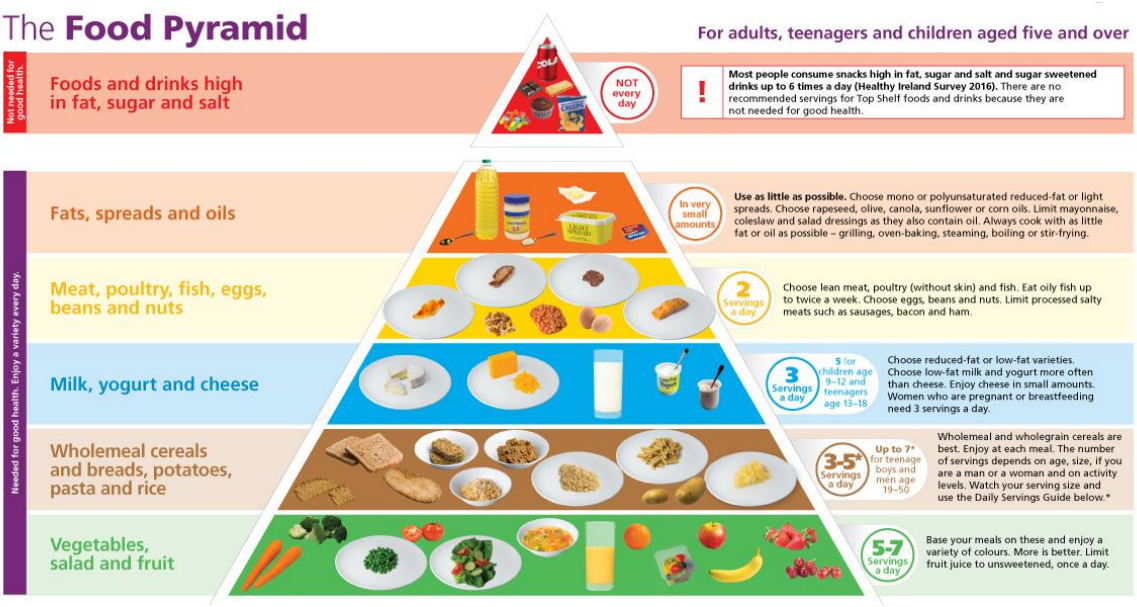


What are 5 components of food plate?

1. **VEGETABLES**
2. **PROTIEN**
3. **GRAINS**
4. **FRUIT**
5. **WATER**

VP-GF

FOOD PYRAMID



- **ECTOPARASITES** Organisms which live **on the surface** of the skin or temporarily invade the superficial tissues of the host (e.g. lice Infestation
- **ENDOPARASITES** Organisms that live **within the body** of the host e.g. All protozoa & helminthic parasites of man…. Infection
- **Parasitism** A relationship in which a **parasite benefits** from the host and the **host gets** nothing but **injury**.
- **Obligate Parasites:** Parasites that cannot exist without a host. (e.g. Toxoplasma gondii).
- **Facultative Parasites:** Organisms that under favourable circumstances can live a Parasitic or Free-living existence. (e.g. Naegleria fowleri, Acanthamoeba spp., B. mandrillaris).
- **Accidental Parasites:** Organisms that attack an unusual host e.g. Echinococcus granulosus in man.
- **Aberrant Parasites:** Organisms that attack a host where they cannot live or develop further (e.g Toxocara canis in man).
- **Opportunistic Parasite** Infects Only Immunocompromised
- **Free-living:** It describes, the non-parasitic stage of existence which can be lived independent of host, e.g. Hookworms have active free-living stages in the soil.

4.Community viva

Define health= WHO defined health as: "a state of complete physical, mental, and social well-being and not the mere absence of disease or infirmity."

- **Disease** is the existence of some pathology or abnormality of the body, which is capable of detection using, accepted investigation methods.
- **Illness** is the subjective state of a person who feels aware of not being well.
- **Sickness** is a state of social dysfunction: a role that an individual assumes when ill

Components of health education= human biology, nutrition, hygiene

Dysentery; Any episode of diarrhea in which **blood** is present in **loose watery stools**

The most common types of dysentery are:

1. **Amoebic dysentery**. Entamoeba histolytica. Trophozoite Form (dividing form) are responsible for producing pathology, fecal matter, liver abscess, metronidazole, iv fluid,
2. **Bacillary dysentery**. Bacteria Shigella, Ors, iv fluids, antibiotics

Difference between amoebic and bacillary dysentery

Macroscopy

Character	Amoebic dysentery	Bacillary dysentery
Number	6-8 motions per day	> 10 motions per day
Amount	Copious	Small
Odour	Offensive	Odourless
Colour	Dark red	Bright red
Reaction	Acidic	Alkaline
Consistency	Non-adherent	Adherent

SPREAD OF SHIGELLA

Shigella spreads through:

1. **Fecal-oral route** (contaminated hands)
2. **Contaminated food and water**
3. **Person-to-person contact** (especially in crowded places)
4. **Poor hygiene and sanitation**
5. **Flies** (carrying bacteria from feces to food)

Common in areas with poor water quality and sanitation.

Symptoms of dysentery

1. **Frequent watery stools with blood and mucus**
2. **Abdominal cramps and pain**
3. **Tenesmus** (painful straining during defecation)
4. **Fever and chills**
5. **Nausea and vomiting**
6. **Dehydration** (in severe cases)

Principles of Primary Health Care (PHC)

1. **Equity or Equitable Distribution of Health Services**
   Health services should be provided according to the needs of people, regardless of their ability to pay. Equity means fairness in service distribution, not necessarily equality.

2. **Community Participation**
   Involving individuals, families, and communities in promoting their health and welfare is essential. For example, lady health workers in Pakistan are selected and trained locally to ensure that the services are culturally acceptable.

3. **Intersectoral Coordination**
   Health cannot be achieved by the health sector alone; it requires collaboration with other sectors such as housing, food, and sanitation to address broader health determinants.

4. **Appropriate Technology**
   Technology should be accessible, affordable, acceptable, and available. For example, using oral rehydration salts (ORS) for diarrhea at home instead of hospitalization

Difference between equity and equality

- **Equality** focuses on equal treatment.
- **Equity** focuses on **fair treatment**, ensuring everyone gets what they need to reach the same health outcome.

Monitoring vs. Evaluation

Aspect	**Monitoring**	**Evaluation**
Definition	Continuous process of tracking activities and progress.	Periodic assessment of overall impact and effectiveness.
Focus	Day-to-day activities and outputs.	Outcomes, goals, and long-term results.
Objective	Ensure the project is on track.	Determine success, relevance, and lessons learned.
Timing	Ongoing during the project.	Conducted at specific intervals (mid-term, end).
Data Type	Quantitative (how many activities done).	Both quantitative and qualitative (impact and change).
Example	Tracking how many patients received vaccines in a campaign.	Assessing if the vaccination campaign reduced disease incidence.

Quick Summary:

- **Monitoring = Process Tracking**
- **Evaluation = Impact Assessment**

Evaluation is also called analyzing

How policy is formulated?

1. **Agenda setting** (Problem identification)
2. **Policy formulation** – Involves exploring a variation of options or alternative courses of action available for addressing the problem. (appraisal, dialogue, formulation, and consolidation)
3. **Decision-making** – Government decides on an ultimate course of action, whether to perpetuate the policy status quo or alter it. (Decision could be 'positive', 'negative', or 'no-action')
4. **Implementation** – The ultimate decision made earlier will be put into practice
5. **Evaluation** – Assesses the effectiveness of a public policy in terms of its perceived intentions and results. Policy actors attempt to determine whether the course of action is a success or failure by examining its impact and outcomes

Content of policy

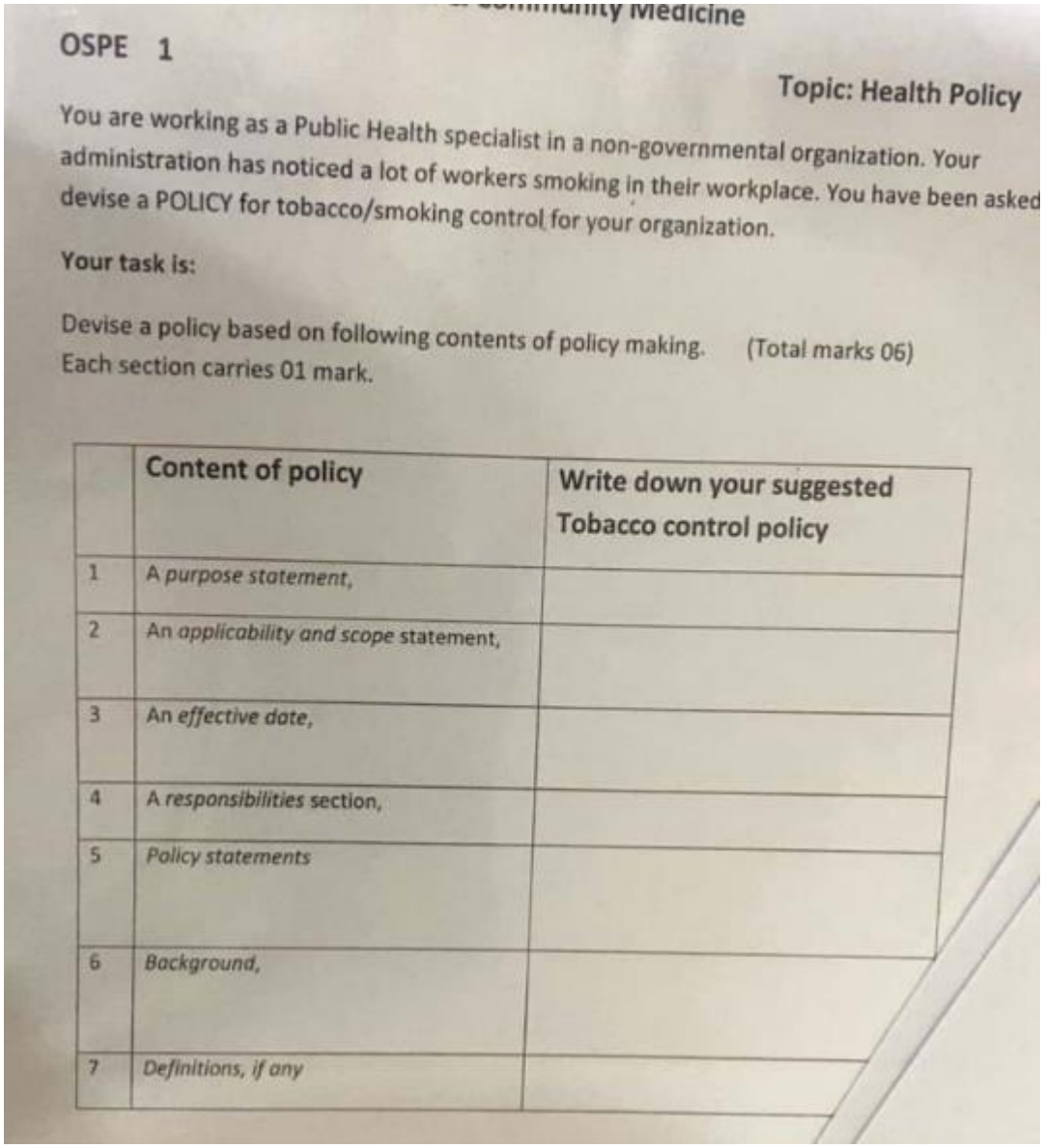
- **A purpose statement**, why the organization is issuing the policy, and what its desired effect or outcome of the policy should be
- **An applicability and scope statement**, describing who the policy affects and which actions are impacted by the policy. The applicability and scope may expressly exclude certain people, organizations, or actions from the policy requirements. Applicability and scope is used to focus the policy on only the desired targets, and avoid unintended consequences where possible
- **An effective date**, indicates when the policy comes into force
- **A responsibilities section**, indicating which parties and organizations are responsible for carrying out individual policy statements. Many policies may require the establishment of some ongoing function or action
- **Policy statements**
- **Background**
- **Definitions**

TYPES OF POLICIES

1. **Distributive Policies** extend goods and services to members of an organization, as well as distributing the costs of the goods/services amongst the members of the organization. Examples include government policies that impact spending for welfare, public education, highways, and public safety
2. **Regulatory Policies**, or mandates, limit the discretion of individuals and agencies, or otherwise compel certain types of behavior. These policies are generally thought to be best applied when good behavior can be easily defined and bad behavior can be easily regulated and punished through fines or sanctions. Example: fairly successful public regulatory policy is that of a highway speed limit
3. **Constituent policies**, create executive power entities, or deal with laws
4. **Redistributive polices**, They are dynamic, they are not just static lists of goals or laws. Policy blueprints have to be implemented, often with unexpected results. Social policies are what happens 'on the ground' when they are implemented, as well as what happens at the decision making or legislative stage

Type of health policies

1. **Regulatory health policies** help standardize and control certain groups of people
2. **Allocative health policies** provide one group of people with money or power by taking it from somewhere else



Community Medicine

OSPE 1

Topic: Health Policy

You are working as a Public Health specialist in a non-governmental organization. Your administration has noticed a lot of workers smoking in their workplace. You have been asked devise a POLICY for tobacco/smoking control for your organization.

Your task is:

Devise a policy based on following contents of policy making. (Total marks 06)
Each section carries 01 mark.

	Content of policy	Write down your suggested Tobacco control policy
1	A *purpose statement,*	
2	An *applicability and scope* statement,	
3	An *effective date,*	
4	A *responsibilities* section,	
5	*Policy statements*	
6	*Background,*	
7	*Definitions, if any*	

BHU – A medical facility situated in the smallest administrative unit of govt.(UC) with an average catchment population of 10-25,000.

- **RHC** – an upgraded PHC facility located at the sub-district administrative unit at the junction of 4 or 5 UCs; the population served ranges from 20,000-500,000.
- **FLCFs** (1st level care facilities)comprise BHUs & RHCs, MCHCs, TB centers & Civil dispensaries.
- **MCHC** of the department of Health.
- **FWC** – family planning facility of the Ministry of Population Welfare.
- **LHW**: village-based female community health worker.
- **THQ**: secondary level hospital.

DHQ: secondary level hospital.

Q. SERVICES PROVIDED BY BHU?

- **BHU= Immunization, antenatal care, family planning, minor treatments.**
- **RHU= Emergency care, deliveries, lab tests, outpatient services, inpatient services.**

Services in Tertiary Care Hospitals

1. **Specialized Medical Care** – Cardiology, Neurology, Oncology, etc.
2. **Advanced Surgeries** – Cardiac, Neurosurgery, Organ Transplants
3. **Diagnostics** – MRI, CT, Genetic Testing, Lab Services
4. **Intensive Care** – ICU, NICU, CCU
5. **Rehabilitation** – Physiotherapy, Counseling
6. **Teaching & Research** – Medical training and clinical research

District Health Management (DHM)

District Health Management oversees healthcare services at the district level to ensure effective delivery of primary and secondary care.

Key Roles

1. **Planning and Policy Implementation** – Develop health strategies and implement national health policies.
2. **Resource Allocation** – Manage budget, staff, and medical supplies.
3. **Service Delivery** – Supervise BHUs, RHUs, and district hospitals.
4. **Monitoring and Evaluation** – Track health indicators and improve service quality.
5. **Health Promotion** – Conduct awareness campaigns on public health issues.
6. **Disease Control** – Implement vaccination programs and control outbreaks.

Health Management Information System: An information system specially designed to assist in the management and planning of health programs, as opposed to delivery of care

Components of HIS

1. Resource
2. Indicator
3. Data sources
4. Data management
5. Information products
6. Dissemination and uses

Following is the criteria for high **data quality**

1. Timeliness:
2. Periodicity:
3. Consistency:
4. Representativeness (Accuracy):
5. Disaggregation:
6. Confidentiality:

Uses of Health Information

1. Measure health status
2. Determine the quality of health care
3. Quantify disease burden
4. Evidence based panning and policy making
5. Optimum allocation of resources
6. Local and international comparison of health

7. Management of various services and programs
8. Evaluation of health services
9. Scientific research

Categories of Indicators used:

Availability of health care: -Ratio b/w population in various administrative units& health facilities& health manpower	**Social & economic indicators related to health:** -Rate of population increase Adult literacy rate
Accessibility of health care: -% of population within 5 km or one hour walk of nearest health center	**Indicators for provision of PHC:** -% of women with antenatal visits
Acceptability of quality of health care: -Proportion of imunizable disease communicable cases for which patient hx reveals past imunization for disease contracted	**Health status indicators:** -% of children under 5 who are malnourished Infant mortality rate: Child mortality rate Maternal mortality rate

WHO food safety definition

According to **WHO**, **Food Safety** is defined as:
"Food safety is the assurance that food will not cause harm to the consumer when it is prepared and/or eaten according to its intended use."

It focuses on preventing foodborne illnesses by controlling risks at every stage of the food production and distribution process.

Health systems- combination of resources, organization, financing, and management that culminates in the delivery of health services to the population.
GNP (Gross National Product) vs. GDP (Gross Domestic Product)

Aspect	**GNP**	**GDP**
Definition	Total value of goods and services produced by a country's residents **including income from abroad**.	Total value of goods and services produced **within a country's borders**.
Focus	National ownership (citizens' production globally).	Geographic location (domestic production).
Formula	GDP + Net income from abroad	Domestic production only
Example	Pakistan's GNP includes profits of Pakistani companies operating in the UAE.	Pakistan's GDP includes production from foreign companies within Pakistan.

Quick Tip:

- **GDP** = What's produced **inside** the country.
- **GNP** = What's produced **by citizens**, no matter where they are.

PER CAPITA INCOME = NATIONAL INCOME/POPULATION SIZE

Poverty line: is the minimum level of income deemed adequate in a particular country. US$ 1.90 per day

Standard of living=The generally accepted measure of the standard of living is GDP per capita

- Cost-Minimization: Cost of implementing two systems or programs or treatment regimens are compared
- Cost-Benefit: Outcomes are measured in monetary terms (limitations are that everything cannot be measured in monetary terms) so QALY is used
- Cost Effective: Outcomes assumed in non-monetary units. Two health programs with same objectives are compared and find out which program achieves the objectives at least cost
- Cost Utility: Outcome is measured in utility. Quantity adjusted health outcome (QALY) is one such measure

It assumes that a year of life lived in perfect health is worth **1 QALY (1 Year of Life × 1 Utility = 1 QALY)**

Health plan, policy etc

TYPES OF HEALTH PLANS:

1. Longitudinal
2. Horizontal (Curative Hospital based care)
3. Integrated (integrated primary health care plan)
4. 5 year plans (12^{th} health plan of Pakistan)
5. ADP (Annual Development Plan)
6. SAP (Social Action Program)
7. Short term
8. Long term (National Rural Support Program)

Primary health care: "It is the essential health care made universally accessible to the individual and communities with their full participation at price communities can afford"

Up till now 12 (5 year) plans has been devised in health sector.

6 PRINCIPLES

1. Equity
2. Community Participation
3. Intersectoral Coordination
4. Appropriate Technology
5. Accessibility
6. Affordability