

Pharma

Difference between PPI and PCABs?

PCABs work by binding to the potassium-binding site of the H⁺/K⁺ ATPase enzyme, which blocks the final step of stomach acid production. This results in more rapid and potent acid suppression than proton pump inhibitors (PPIs), which require activation in an acidic environment. PCABs also have several other advantages over PPIs, including:

Acid stability: PCABs can be dosed independently of food consumption.

Faster symptom relief: PCABs provide faster and more durable symptom relief than PPIs.

Less variability in patient response: PCABs have less variability in patient response than PPIs. PCABs are acid stable, facilitate reversible inhibition, and may be dosed independent of mealtimes.

MOA of albendazole?

- It binds to beta tubulin and inhibits microtubule polymerization
- Also block glucose transport into parasite
- Intestinal parasites are immobilised or die slowly

Triple regime for H. Pylori x 14 days (2 weeks)

- Lansoprazole (PPI) 30mg BD
- Clarithromycin 500 mg BD
- Amoxicillin 1g BD

Bismuth quadruple therapy for H Pylori x 14 days (2 weeks)

- PPI at standard dose twice daily
- Plus Bismuth
- Plus tetracycline
- Plus metronidazole
- Recommended in a region where Clarithromycin resistance rate > 15%

Drugs given for ascites

- potassium sparing diuretics
- furesomide
- beta blocker[carvidolol]

Oral drugs of HBV and their MOA

Interferons - inhibit host cell enzymes that inhibit viral RNA translation, ultimately leading to degradation of viral mRNA and tRNA

Lamivudine (cytosine analog) - competitively inhibit HBV RNA-dependent DNA polymerase

Adefovir - it is a nucleotide analog that is phosphorylated by cellular kinases to adefovir diphosphate, which is then incorporated into viral DNA. This leads to chain elongation and prevents replication of HBV

Entecavir (guanosine nucleotide analog) - Following intracellular phosphorylation to triphosphate, it competes with the natural substrate, deoxy guanosine triphosphate, for viral RT

Hepatitis B Drugs

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.

Alternative Antiviral

- Lamivudine: Inhibits reverse transcriptase, preventing the conversion of RNA to DNA.
- Adefovir: Inhibits HBV DNA polymerase, blocking viral replication.

Interferon Therapy

- Pegylated Interferon-alpha: Stimulates the immune system, enhancing antiviral activity and inhibiting HBV replication.

For Acute Hepatitis B

- Supportive care, no antivirals unless severe.

Interferons

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

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.

oral drugs of HCV and their MOA

Ribavirin (competitively inhibits viral rna synthesis)

Sofosbuvir(inhibits viral rna polymerase)

Daclatsavir and ledipasvir(inhibit hcv replication)

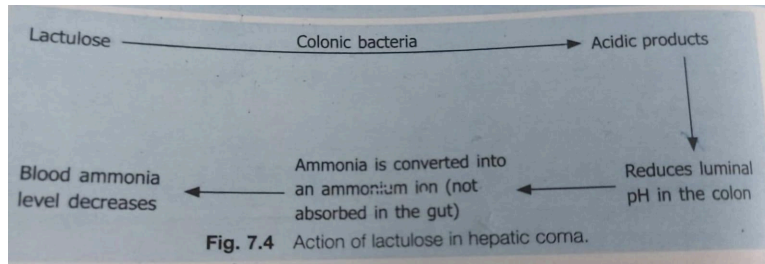
Drugs used for treatment of IBD (Inflammatory bowel disease)

Aminosalicylates	Sulphasalazine Mesalamine Olsalazine Balsalazide
Glucocorticoids	Prednisolone Methyl prednisolone Hydrocortisone Budesonide
Immunomodulators	Azathioprine 6-Mercaptopurine Methotrexate Cyclosporine
Biological response modifiers (TNF alpha inhibitors)	Infliximab Adalimumab
Antibiotics	Metronidazole Ciprofloxacin Clarithromycin
Probiotics	Lactobacillus Bacteroides

Role of lactulose in hepatic Coma

Lactulose is a non absorbable disaccharide sugar that plays a crucial role in management of hepatic encephalopathy, a condition characterized by cognitive disturbances, altered mental status, and potentially life threatening complications, including hepatic Coma.

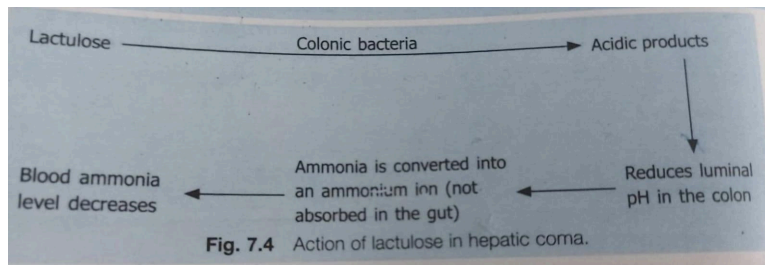
- Lactulose act as a laxative, increasing the bowel movement frequency and softening the stool. This helps to reduce absorption of ammonia and other toxins from gut.
- pH reduction - Lactulose is fermented by colonic bacteria, producing organic acids that lower colonic pH. This acidic environment inhibits the growth of ammonia producing bacteria, reducing ammonia production and absorption.
- Lactulose can trap ammonia in colon, preventing its absorption in bloodstream.



Treatment for multi drug resistant enteric fever

azithromycin, carbapenems.

role of lactulose in hepatic encephalopathy



Qualities of ideal antacid

1. it should be insoluble and capable of neutralizing acid
2. non absorbable
3. donot disturb the acid base balance of body
4. Should not liberate CO₂

Omeprazole action

- Omeprazole is a PPI
- Acts by irreversible inhibition of proton pump (H⁺/ K⁺ ATPase) that is responsible for final step in gastric acid secretion from the parietal cell (they covalently bind to the pump)

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.

Treatment for Multidrug-Resistant (MDR) Enteric Fever:

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

For Morning Sickness:

- Ondansetron
- Promethazine
- Doxylamine + Vitamin B₆

For Chemotherapy-Induced Vomiting:

- Ondansetron
- Granisetron
- Dexamethasone

Antidiarrheal agents

- ORS and parenteral rehydration
- Anti motility agents (codeine, loperamide, diphenoxylate)
- Anti secretory agents (racecadotril, Octreotide)
- Probiotics (lactobacillus, bifidobacterium, saccharomyces boulardii)
- Antimicrobial agents (ciprofloxacin, doxycycline, metronidazole)

Classify antiemetics

1. 5-HT₃ Receptor Antagonists (Ondansetron, Granisetron, Dolasetron, Palonosetron, Ramosetron)
2. Prokinetic agents - D₂ receptor antagonist (Metoclopramide, Domperidone, Levosulpiride)
3. Anti histamines/ H₁ blockers (Dimenhydrinate, Diphenhydramine, Cyclizine, Meclizine, Hydroxyzine, Promethazine, Doxylamine)
4. Anti cholinergics (Scopolamine, Dicyclomine)
5. Neuroleptics (Chlorpromazine, Fluphenazine, Prochlorperazine, Haloperidol)
6. Neurokinin (NK₁) Receptor Antagonists - Aprepitant, Fosapretant
7. Cannabinoids (Dronabinol)
8. Adjuvant anti emetics (Glucocorticoids, benzodiazepenes)

Drugs used for peptic ulcer

Drugs that inhibit gastric acid secretion

1. PPIs (omeprazole, esomeprazole, lansoprazole, pantoprazole)
2. H₂ receptor blockers (Cimetidine, ranitidine, famotidine, roxatidine, nizatidine)
3. Anti muscarinic agents (Pirenzepine, Telenzipine)
4. Prostaglandin analogues (Misoprostol)

Ulcer protectives

- Sucralfate
- Colloidal bismuth sub citrate (CBS)

Drugs that neutralize gastric acids (Antacids)

- Non systemic antacids (magnesium hydroxide, magnesium trisilicate, aluminium hydroxide, calcium carbonate)
- Systemic antacids (sodium bicarbonate, sodium citrate)

Anti h pylori drugs

- Amoxicillin
- Tetracycline
- Clarithromycin
- Metronidazole
- Bismuth subsalicylate
- H₂ antagonists (nizatidine, famotidine)
- PPIs

How metoclopramide acts as prokinetic

- Metoclopramide is a D₂ antagonist, serotonin Receptor antagonist
- Central anti emetic effect at the area postrema
- Peripheral antiemetic effect in the GIT (prokinetic effect). Causes increase in gastric contractions, duodenal and jejunal motility and resting tone of lower Esophageal sphincter.
- Together with decreased pylorus sphincter activity allows food to pass more quickly through the stomach and small intestine.

Mechanism of action of omeprazole

Omeprazole is a PPI and decrease gastric acid secretion

Forensic

Mechanism of action of all poisons almost...

Caustic alkali MOA - highly corrosive substances that can cause severe damage to tissues and organs upon ingestion, inhalation and skin contact.

Oxalic acid MOA

- Corrosive action
- Chelation of calcium ions, forming insoluble calcium oxalate crystals that can deposit in tissues and cause damage
- Inhibition of cellular respiration by binding to mitochondrial enzymes leading to decreased ATP production and cellular energy
- Denaturation of proteins

Carbolic acid (phenol) MOA - corrosion and necrosis

Carbolic acid, also known as phenol, acts as a corrosive poison, causing local tissue damage by denaturing proteins and disrupting cell membranes upon contact with skin or mucous membranes, leading to burning

Hydrocyanic acid and cyanides MOA

- Hydrocyanic acid is a general protoplasmic poison. It inhibits cytochrome oxidase system for oxygen utilisation in cells. Death is due to cytotoxic or histotoxic anoxia, although the blood may contain a normal oxygen content
- Cyanides show corrosive effect on mucus membrane

Phosphorus MOA

- Phosphorus is a protoplasmic poison. It affects cellular oxidation. Its effects on cellular metabolism is comparable to ischemia. Under such anoxic condition, the metabolism of cells diminishes considerably. This is known as necrobiosis, which is classically manifested in liver.

Arsenic MOA

- inhibit sulphhydryl enzymes interfering with cellular metabolism
- Locally, it causes irritation of mucus membranes and remotely depression of nervous system

Mercury MOA - inhibit sulphhydryl enzymes interfering with cellular metabolism

Copper MOA

Lead sulphide MOA

- causes spasm of capillaries and arterioles
- The toxic effects result from fixation of lead in certain tissues such as brain and peripheral nervous system

Thallium MOA

Zinc MOA

Lead MOA

- Inhibits heme synthesis, leading to anemia and CNS toxicity.

Scorpion venom MOA

- Blocks potassium and sodium ion channels, causing autonomic storm.

Ricinus communis (castor oil plant) MOA - Active principle is ricin, a toxalbumin which causes agglutination and lysis of RBCs and has antigenic properties

Abrus precatorius MOA - active principle is abrin, a toxalbumin and resembles viperine snake bite

Capsicum MOA - active principles are capsaicin and capsin, which cause irritation and vesication on skin.

Calotropis MOA - 5 active principles (uscharin, calotoxin, calactin, calotropin, gigantol). These act as cardiac glycosides which inhibit Na/K ATPase

Semecarpus anacardium (marking nut) - Active principles are semecarpol and bhitawanol. They cause blisters in mouth and throat, dyspnea and cyanosis

Croton Tiglium (jamalgota) - Active principle is crotin, a toxalbumin, which is an irritant and a vesicant

Arbus precartorius (jequirity bean) - Active principle is Abrin, a toxalbumin. Its action resemble those of viperine snake bite

Potash alum MOA - Alum ions neutralise the charges on plasma proteins causing the blood to coagulate producing astringent effect

Sulphuric acid MOA

- Extraction of water from tissues
- Coagulation of cellular proteins
- Conversion of Hb to hematin

Effects of corrosives

EARLY EFFECTS

- Pain and shock

- Vomiting
- Dyspnea due to respiratory obstruction from laryngeal edema

LATE EFFECTS

- Perforation of stomach
- Pulmonary edema or bronchopneumonia

DELAYED EFFECTS

- Esophageal or pyloric stricture
- Laryngeal stricture
- Pulmonary fibrosis

classify vegetable posions.

(Remember as SERAP-CCCC)

1. Ricinus communis (castor oil plant)
2. Croton tiglium (jamalgota)
3. Abrus precatorius (jaqueirty bean)
4. Colocynth (bitter apple)
5. Ergot
6. Capsicum (chillis)
7. Semecarpus anacardium (marking nut)
8. Calotropis (madar)
9. Plumbago rosea (lal chitra)
10. Plumbago zeylanica (chitra)

Phenolic crystals

5 vegetable poisons with active principle

1. Ricinus communis (castor oil plant) - Ricin
2. Croton Tiglium (jamalgota) - crotin
3. Arbus precartorius (jequirity bean) - Abrin
4. Capsicum - capsaicin or capsin
5. Semecarpus anacardium (Marking nut) - semecarpol, bhilawanol
6. Calotropis - uscharin, calotoxin, calactin, calotropin

Difference between action of mineral acids and vegetable acids!

- Mineral acids have stronger corrosive action than vegetable acids

- Mineral acids cause immediate coagulation of proteins, while vegetable acids cause delayed coagulation
- Mineral acids tend to cause local effects, while vegetable acids can cause systemic effects

Toxic compounds of mercury...mercuric and mercurous chloride....which is more toxic.

- mercuric is more toxic
- (The poison which is absorbable, soluble is more toxic)

Is mercury metal toxic

Mercury is not toxic when ingested but it is toxic when inhaled

smokey stool syndrome

- Dark luminous offensive faeces that occur in acute poisoning with phosphorus(white phosphorus)
- 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.

phosphorus poisoning cause which type of burns?

Slow healing burns

oxalic acid antidote

A suspension of 300gm of chalk in water or milk will neutralise about 20g of oxalic acid

Arsenic poison management

- Stomach should be repeatedly washed out with warm water or milk
- A saline purgative, such as sodium sulphate is left in stomach
- No alkalis should be given by mouth since they increase the solubility of white arsenic
- Antidote: Freshly precipitated hydrated ferric oxide
- If ferric oxide not quickly prepared - calcined magnesia or charcoal

Lead line

- Seen in chronic lead poisoning
- It is stippled bluish-black line due to subepithelial deposition of lead sulphide granules on the gums at the junction with the teeth (not on the teeth)

Mees line

- Seen in arsenic and thallium poisoning

- These are white bands crossing the nails of fingers and toes

Phossy jaw

- Seen in chronic phosphorus poisoning
- The first symptom is toothache, followed by swelling of the jaw, loosening of teeth, necrosis of gums, and then sequestration of bone in mandible.

Forensic non-observed...marking nut, it's active principle, fatal dose and medicolegal importance

- Marking nut also called *Semecarpus anacardium*
- Active principle: semecarpol and bhillawanol
- Fatal Dose: 5 - 10 gm
- Medicolegal importance: (a) Used as abortifacient (abortion stick) (b) used by malingerers to produce artificial bruise or irritant conjunctivitis

Classify corrosives

A corrosive poison is simply a highly active irritant and not only produce inflammation but also actual ulceration of tissues. This group consist of strong acids and strong alkalis.

- Mineral acids - sulfuric acid, nitric acid, hydrochloric acid
- Organic acids - oxalic acid, carbolic acid, acetic acid, salicylic acid
- Vegetable acid - hydrocyanic acid
- Concentrated alkalis - caustic soda, caustic potash, and carbonates of ammonium, sodium and potassium

Irritant Poisons

Irritant poisons produce symptoms of pain in the abdomen, vomiting and purging. The post-mortem appearances are usually evident to the naked eye, and show redness and ulceration of gastrointestinal tract. This group consist of inorganic, organic and mechanical substances. Corrosives in dilute solutions act as irritants.

- Inorganic - consist of non metallic and metallic poisons. The non metallic poisons include Phosphorus, chlorine, bromine, and iodine. The metallic poisons include arsenic, antimony, mercury, lead, copper, thallium, zinc, manganese, barium and radioactive substances
- Organic - consist of vegetable and animal poisons. Vegetable poisons include castor seeds, croton seeds, abrus precatorius, colocynth, ergot, capsicum, semecarpus anacardium (marking nut), calotropis (madar) and plumbago. The animal poisons include cantharides, snakes, scorpions, spiders and poisonous insects.

- Mechanical - include coarsely powdered glass, chopped hair, dried sponge, and diamond dust

Mechanism of action of mineral acids

- Extraction of water from tissues
- Coagulation of cellular proteins
- Conversion of Hb to haematin

Treatment of hydrocyanic acid and antidote

- Converting Hb to methemoglobin, as cyanmethemoglobin is non toxic
- Antidote: IV administration of methylene blue
- Mercury cyanide poisoning - injection of BAL (dimercaprol)

Uses of phosphorus

- Yellow phosphorus is an ingredient of fireworks and certain rodent and insect poisons.

Phosphorus mechanism of action

Phosphorus is a protoplasmic poison. It affects cellular oxidation. Its effects on cellular metabolism is comparable to ischemia. Under such anoxic conditions, the metabolism of cells diminish considerably. This is known as necrobiosis, which is classically manifested in liver. Deposition of glycogen in liver is inhibited while deposition of fat is increased.

Types of burns caused by phosphorus

In acute poisoning there is burning pain in throat, esophagus and stomach with intense thirst, frequent gaseous eructations, nausea, vomiting, and diarrhea.

Burns in carbolic acid (phenol) poisoning

- White, bleached, hardened

Chronic arsenic poisoning...stages...

4 stages

1. Nutritional and GIT disturbances
 - Gradual ematiation - extreme thinness and weakness (earliest signs)
 - Loss of appetite
 - Nausea and intermittent attacks of vomiting and diarrhea
2. Catarrhal changes
 - Symptoms of common cold
 - Mucus membranes are inflamed resulting in conjunctivitis, runny nose, coughing, hoarseness of voice, and branchial catarrh

3. Skin rashes
 - nettle rash
 - raindrop type pigmentation - patchy brown pigmentation of skin
 - hyperkeratosis of palms and soles
 - Mee's lines - white bands crossing nails of fingers and toes
4. Nervous disturbances
 - Arsenical neuritis resemble chronic alcoholism
 - Tingling, numbness of hands and feet, tenderness of muscles, sometimes with paresis

Abrus precatorius...seeds types

Oval red odourless tasteless seeds with black spot on one pole, size of a pea

Abrus precatorius...medicolegal importance

- Poison cattle by means of sui
- Malingers use powder seeds to produce conjunctivitis
- Arrow poison

Castor oil...active compound...

- Active compound: Ricin
- It is a toxalbumin and cause agglutination and lysis of red cells and has antigenic properties

Xanthoproteic reaction

- Seen in nitric acid poisoning
- Yellow discoloration of skin

What is contraindicated in mineral acids poisoning treatment

- Stomach wash and emetics
- If acid taken - alkaline carbonates are contraindicated

Patho

Ascites diagnosis

Ascites is the accumulation of excess fluid in the peritoneal cavity. It becomes clinically detectable when at least 500 ml has accumulated.

3 clinical features of ascities

- Abdominal distension
- Fluid thrill
- Dullness to percussion - when abdomen is percussed, a dull sound is heard, indicating the presence of fluid
- Shifting of dullness - When the patient changes position, the dullness shifts, indicating that the fluid is free to move within peritoneal cavity

Difference between mild moderate and intense ascities or what are they

Mild Ascites (Grade 1)

- Detectable only on Ultrasonography

Moderate Ascites (Grade 2)

- Can be detected on physical examination
- Fluid volume is usually more than 500 ml

Severe Ascites (Grade 3)

- Causes abdominal distension accompanied by flattening of umbilicus or umbilical hernia.
- Dyspnea indicates that the peritoneal fluid is large, 5 - 15 L

Ascities can occurs because of?

Ascites may be Portal HTN Related or non portal HTN related.

Portal HTN Related Ascites

- Cirrhosis
- Acute hepatitis
- Liver malignancy
- Right sided HF
- Budd Chiari syndrome
- Splenic vein Thrombosis
- Schistosomiasis

Non Portal HTN Related Ascites

- Peritonitis (e.g. tuberculosis)
- Peritoneal carcinomatosis
- Pancreatitis
- Vasculitis
- Hypoalbuminemia
- Meig syndrome
- Hypothyroidism

Difference between Mallory and Boerhaave syndrome?

Mallory syndrome is longitudinal laceration of mucosa of GE junction. It is caused by severe vomiting usually due to alcoholism or bulimia. It presents with painful hematemesis.

Boerhaave syndrome is rupture of esophagus leading to air in mediastinum and subcutaneous emphysema.

Mallory syndrome has risk of developing Boerhaave syndrome.

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

- Reflux esophagitis (GERD, bulimia Nervosa)
- Drug induced esophagitis (NSAIDs, acetaminophen, antibiotics)
- Infectious esophagitis (HSV, CMV, Candida albicans)
- Eosinophilic esophagitis
- Autoimmune esophagitis
- Radiation esophagitis

Role of H pylori in development of gastric cancer...(repeated inflammations...intestinal metaplasia...dysplasia..)

H pylori leads to chronic inflammation in the stomach which can cause damage to gastric mucosa and increase the risk of cancer

Most common type of gastric cancer... adenocarcinoma (intestinal type)

Liver cirrhosis changes

Liver cirrhosis is end stage liver damage characterised by disruption of normal hepatic parenchyma by bands of fibrosis and regenerative nodules of hepatocytes

Clinical Features of cirrhosis

Portal HTN leads to

- Ascites
- Congestive splenomegaly
- Esophageal varices, hemorrhoids, caput medusae

- Hepatorenal syndrome

Decreased detoxification results in

- Mental status changes, asterixis, and eventual coma
- Gynecomastia, spider angiomas, and palmar erythema due to hyperestrogenism
- Jaundice

Decreased protein synthesis leads to

- Hypoalbuminemia with edema
- Coagulopathy due to decreased synthesis of clotting factors

Alcoholic liver disease

Damage to hepatic parenchyma due to consumption of alcohol

It presents in 3 forms

1. Hepatic steatosis (Fatty liver)
2. Alcoholic hepatitis
3. Alcoholic cirrhosis

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:

- Steatosis: Fat accumulation in hepatocytes
- Alcoholic Hepatitis: Inflammation, Mallory bodies, neutrophilic infiltration
- Cirrhosis: Fibrosis, regenerative nodules, liver dysfunction
- Complications: Cirrhosis, hepatocellular carcinoma, portal hypertension.

Complications

- Cirrhosis
- hepatocellular carcinoma
- portal hypertension.

Types of gall stones

- Cholesterol stones (yellow) - radiolucent
- Bilirubin gallstones (pigmented) - radio opaque

4 morphological features of chronic cholecystitis

1. Variable thickening of gallbladder wall
2. Rokitansky Aschoff sinuses - herniation of gallbladder mucosa into muscular wall
3. Mild cases - mono nuclear infiltrate in mucosa and subserosal tissue
4. Severe cases - marked sub epithelial and subserosal fibrosis
5. Porcelain gallbladder is late complication (Porcelain gallbladder is shrunken, hard gallbladder due to chronic inflammation, fibrosis, and dystrophic calcification)

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

Cholesterol stones risk factors

- Age
- Estrogen (oral contraceptives)
- Clofibrate
- Crohn Disease
- Cirrhosis

Bilirubin stones risk factors

- Extravascular hemolysis
- Biliary tract infection (E. Coli, ascaris lumbricoides)
- GIT Diseases: Crohn disease, ileal bypass, cystic fibrosis with pancreatic insufficiency

Most common extra intestinal manifestation of IBD

- Rash
- Uveitis
- Arthritis
- Crohn disease - oxalate stones in kidney

Crohn's disease...characteristic morphological features

Intestinal manifestations of Crohn's disease

- Skip lesions
- Skin nodules
- Ileum (m/c affected)
- Knife like fissures
- Microscopy: non caseating granuloma
- Aphthous ulcers
- Creeping fat
- TNF alpha elevated

Autoimmune gastritis... characteristic morphological features.

Chronic autoimmune gastritis is due to autoimmune destruction of gastric parietal cells, which are located in stomach body and fundus.

Clinical Features

- Atrophy of mucosa with intestinal metaplasia
- Achlorhydria with increased gastrin levels and antral G cell hyperplasia
- Megaloblastic (pernicious) anemia due to lack of intrinsic factor
- Increased risk of gastric adenocarcinoma

Colorectal Carcinoma Gene Mutations (Stepwise Progression)

Stepwise Genetic Model (Vogelstein model):

- APC Mutation (Adenoma Formation) – Early event
- KRAS Mutation (Adenoma Growth)
- p53 Mutation (Malignant Transformation)
- DCC Mutation (Invasion and Metastasis)

19.Patho non-observed

Hepatitis-B markers etc..pic shared

16.Patho non-observed..

Ulcerative colitis

Most common extra intestinal manifestations

Why regular colonoscopy is

Medicine

Obstructive jaundice history examination clinical features causes mechanism

History

- When did u first notice you were yellow?
- Have u been unwell recently
- Have u noticed any other associated symptoms
- What's changed with your bowels
- Have you had any urinary symptoms
- Abdominal pain - SOCRATES
- Past medical and surgical history
- Drug history
- Social history (alcohol, smoking)
- Family history

Examination

- Abdominal examination (look for jaundice, lymphadenopathy, hepatomegaly, splenomegaly, Murphy's sign)
- Murphy's sign is elicited by asking the patient to breathe out and then gently place the hand below the costal margin on the right side at the mid clavicular line (the approximate location of gallbladder). The patient is then instructed to breathe in
- If the patient presents with jaundice and they have a non tender, palpable gallbladder on examination, the cause is unlikely to be due to gallstones. This is bcz gallstones form over a prolonged period, which results in a shrunken fibrotic gallbladder which doesn't distend easily. As a result, jaundice and a palpable gallbladder should raise suspicion of malignant obstruction of biliary tree (e.g pancreatic cancer)

Etiology of biliary tract obstruction

- Gallstones
- Tumors (pancreatic, gallbladder, bile duct)
- Stricture
- Parasites (liver flukes)

clinical Features

- Jaundice and icterus
- Pruritis due to increased plasma levels of bile acids
- Abdominal pain, fever and chills
- Dark urine due to bilirubinuria
- Pale clay colored stools
- Lab studies show elevated conjugated bilirubin, elevated ALP, and elevated 5' nucleotidase

ascites history examination clinical features cause management

Surgery

Which appendectomy is preferred in children(laparoscopic is preferred over open one)

Approach for Appendectomy

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

Ulcerative colitis picture identification

extraintestinal symptoms +most common

senario.....

RIF pain and tenderness

name the different D/Ds

what is meant by

subacute and acute

intestinal obstruction???

Difference between true and false bruise?

Table 44.1: Artificial bruise and true bruise—differentiating features

	<i>Artificial bruise</i>	<i>True bruise</i>
1. Cause	Chemical	Trauma
2. Situation	Accessible parts of the body	Anywhere
3. Appearance	Blister formation	No blister
4. Colour changes	Nil	Characteristic
5. Ecchymosis	Nil	Present
6. Contents	Acrid serum	Extravasated blood
7. Itching	Present	Not so
8. Fingers	May show marks due to scratching	Not so
9. Analysis	Chemical found in the blister fluid	Not so

Community medicine

Define health

A state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity

Symptoms of dysentery

Bloody diarrhea Abdominal pain , fever , nausea vomiting, fatigue

Primary health care

It is the essential care based on practical, scientifically sound and socially acceptable method and technology made universally acceptable to individuals and families in the community through their full participation and at a cost they and their country can afford to maintain in the spirit of self reliance and self determination

Principles of primary health care

- Community participation
- Inter sectoral collaboration
- Integration of health care programs
- Equity
- Self reliance/ 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 providing equal treatment and access, while equity focuses on addressing health disparities and promoting fairness and justice.
- Equality aims for uniform distribution of resources, while equity aims for proportionate allocation based on need

Equity

Equity means division of resources according to the needs of community.

Horizontal equity - receiving equal care for equal conditions

Vertical equity - this part of equity points out the fact that different conditions must be treated differently which is an acceptable fact in medicine bcz the treatment of poor eyesight isn't the same as the needed treatment for infertility.

Monitoring Vs evaluation!!! Evaluation is also called analyzing

- Monitoring aims to support quality improvement and decision making, while evaluation aims to assess the effectiveness and impact of healthcare programs or services.
- Monitoring typically focuses on specific processes or performance indicators, while evaluation takes a broader view, examining the overall effectiveness and impact of healthcare programs or services

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

BHU....services provided

- BHU (Basic Health Unit) is located at a Union Council and serves a catchment population of 5000 to 10,000
- Services provided at BHU are promotive, preventive, curative and referral. Outreach/ community based services are part of package provided by BHU.
- Education concerning prevailing health problems and the methods of preventing and controlling them
- Maternal and child health care, including family planning
- Immunisation against major infectious diseases
- Appropriate treatment of common disease and injuries
- Provision of essential drugs

Services provided by tertiary care hospital

- In tertiary level of care, specialised consultative care is provided usually on referral from primary and secondary medical care.
- Facilities for advanced medical investigation and treatment is present
- Services provided include cancer management, neurosurgery, cardiac surgery, advanced lab investigations, advanced ICU care and a host of complex medical and surgical interventions.
- Advanced diagnostic support services and specialised intensive care which cannot be provided by primary and secondary health centers are available at the tertiary health centers.

District health management

District health system has 3 elements

1. Community
2. Health care delivery system (service inputs, service distribution, service outputs, management and organisation, support system)
3. Environment (social, cultural, political, economic etc)

WHO food safety definition

The assurance that food will not cause harm to the consumer when it is prepared or eaten according to its intended use.

Health plan, policy etc

Health planning is a process culminating in decisions regarding the future provisions of health facilities and services to meet health needs of the community

Health policy refers to decisions, plans and actions to achieve specific health goals within a society, in the form of explicit and implicit written documents, mentioning the vision for the future and target areas for short and medium term, outlines priority areas, expected roles of different groups and information to the public.

Marasmus and kawashiokor.

Write strategies to promote awareness in rural areas

- Collaboration with local organizations
- Train community health workers to provide health education and outreach services
- Utilize local media such as newspapers, radio stations, and television channels, to promote health awareness
- Engage with local leaders and decision makers to promote health awareness and support

-common channel?

-which theory or model of health education is used?

-two teaching methods

-Housefly

Biological name - Musca domestica

4 diseases caused by it

- Typhoid fever - House fly can carry bacteria salmonella typhi which cause typhoid fever
- Dysentery - House fly can transmit bacteria shigella
- Cholera - House fly can carry bacteria vibrio cholera
- Conjunctivitis

House fly can spread these diseases through their vomit, feces and contaminated body parts, which can come into contact with food, water, and human mucus membranes.

Mechanism of disease transmission by it

1. Contamination of food and water

- Landing on contaminated surfaces
- Picking up pathogens
- Depositing pathogens on food and water

2. Regurgitation and defecation

- Regurgitation - House flies regurgitate their stomach contents, which can contain pathogens, onto food and surfaces
- Defecation - House flies defecate on food and surfaces, depositing pathogens

Measures to prevent the spread

- Proper waste disposal
- Maintain cleanliness
- Eliminate potential breeding sites such as decaying matter, animal waste, and standing water
- Cover food and drinks
- Store food properly
- Wash hands regularly
- Use insecticides
- Educate the public

leishmania case

Causative Agent: Leishmania species (protozoan parasite).

Arthropod responsible for transmission:

- It is spread by the bite of certain types of sand fly.
- Biological Name: Phlebotomus argentipes (most common vector for visceral leishmaniasis).

- Mode of Transmission: Sandflies inject promastigotes into human skin during a blood meal.

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.

Prevention

- Sleeping under nets treated with insecticide
- Avoid sleeping on grounds as sand fly flies at low flights
- Spraying insecticides to kill sand flies and use repellants

What is food plate?

A food plate, also known as MyPlate, is a visual representation of the recommended amounts and types of foods to eat for a healthy diet:

What it shows

The plate is divided into five food groups: fruits, vegetables, grains, protein, and dairy. The colors of the sections indicate how much of each food group to eat.

Who created it

The United States Department of Agriculture (USDA) created MyPlate in 2011 to replace the Food Guide Pyramid.

What is safe food?

safe food means food that does not contain any poisonous, deleterious, or disease-causing substance or microorganisms that may render such food injurious to human health.

What are 5 components of food plate

Fruits & vegetables, grains, proteins dairy, fats and oils

- Half of the plate should be vegetables and fruits
- About one quarter of the plate should be grains
- One quarter protein
- Drink fat free or low fat (1%) milk and water instead of soda, sports drinks, and other sugary drinks

How policy is formulated

- Agenda setting - problem identification
- Policy formulation
- Decision making
- Implementation
- Evaluation

Content of policy

- A purpose statement
- An applicability and scope statement
- An effective date
- A responsibilities section
- Policy statements
- Background
- Definitions

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

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

4)kwashiorkor marasmus identify

_features

_difference

17.Medicine

30 year old male..

Mild scleral icterus..

Especially after fasting

Abdomen soft, non-tender with no hepato or splenomegaly

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..

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...laproscopic or incision?

If 60 years old female presented with right iliac fossapain....malignancy...Ca Cecum!!!!!!

What is meant by subacute and acute intestinal obstruction

gillbert syndrome senario

common features.....jaundice'yellow discoloration of eyes and urine

everything else normal of liver tests etc

only unconjugated bilirubin is high

enzyme involved??¿?

Gilbert syndrome

- Mutation in the promoter region of UGT1A1 gene → mild reduction of UDP-glucuronosyltransferase activity → ↓ conjugation of bilirubin → ↑ indirect bilirubin
- Most common inherited hyperbilirubinemia
- Impaired hepatic bilirubin uptake
- Inheritance: autosomal recessive or autosomal dominant
- Asymptomatic or unspecific symptoms such as fatigue and loss of appetite
- Transient, usually mild jaundice (varying from mild scleral jaundice to general jaundice)
- Slightly ↑ indirect bilirubin but < 3 mg/dL (higher levels are possible during episodes of increased bilirubin breakdown)
- Normal liver function
- No evidence of hemolysis
- Detection of mutation using PCR
- Treatment: not required (benign condition)

Spleen examination in final proff 2024

Extra surgery questions

Psoas abscess it's causes

- a rare condition characterized by a localized collection of pus in the iliopsoas muscle compartment.
- It is categorized into primary psoas abscess (caused by hematogenous or lymphatic spread of a pathogen) and secondary psoas abscess (resulting from contiguous spread from an adjacent infectious focus).
- The most common causes of secondary psoas abscess are infections of gastrointestinal or musculoskeletal origin.
- A small abscess (< 3.5 cm) in a nonseptic patient can be managed with antibiotic therapy alone. Larger or multiloculated abscesses should be drained under image guidance or with surgery. In a psoas abscess of any kind, the underlying cause should be evaluated and treated.

McBurney's point

- McBurney's point is a specific location on the abdomen that is used to diagnose appendicitis
- Located at the junction of middle and outer thirds of an imaginary line drawn from the anterior superior iliac spine to the umbilicus
- In case of appendicitis, it is often tender to touch
- It is also used as a guide for surgical incision in an appendectomy

There was an X Ray abdomen image in final proff 2024

Aik community ka station tha about health implementation of treating diabetes

Strategies to promote awareness

Which health model

Points for history taking

APPENDICITIS

- Patient often initially develop constant, non specific periumbilical or diffuse abdominal discomfort. Over a matter of hours, the pain localises to RLQ and is associated with anorexia, nausea and vomiting.

Cholangitis

- Charcot's triad (fever, RUQ abdominal pain, and jaundice) occur in 50 to 70% patients

Cholecystitis

- 75% patients have nausea and vomiting
- Persistent and severe RUQ and epigastric pain may occur.
- It can radiate to right shoulder or back
- Acute Cholecystitis is usually due to gallstones

Crohn's disease

- Abdominal pain, fever, weight loss, diarrhea with or without bleeding

Ulcerative colitis

- Bloody diarrhea is principal symptom
- Defecation may relieve lower abdominal cramps

Pancreatitis

- Abdominal pain
- Pain typically starts in epigastrium and radiates to back.
- Common causes include gallstones and alcohol
- Nausea and vomiting

Pain in GIT and their characteristics

1. Epigastric Pain

- *Location:* Upper middle abdomen, just below the ribcage
- *Causes:* Gastritis, peptic ulcer disease, gastroesophageal reflux disease (GERD), pancreatitis
- *Characteristics:* Dull, aching, or burning sensation

2. *Heartburn*

- *Location:* Chest and throat

- *Causes:* Gastroesophageal reflux disease (GERD), hiatal hernia, obesity
- *Characteristics:* Burning sensation, sour or bitter taste in the mouth

3. ***Dyspeptic Pain***

- *Location:* Upper abdomen
- *Causes:* Gastritis, peptic ulcer disease, gastroesophageal reflux disease (GERD), functional dyspepsia
- *Characteristics:* Feeling of fullness, bloating, or discomfort

4. ***Colicky Pain***

- *Location:* Abdomen
- *Causes:* Gallstones, kidney stones, intestinal obstruction, inflammatory bowel disease
- *Characteristics:* Severe, crampy pain that comes and goes in waves

5. ***Referred Pain***

- *Location:* Varies, but often in the back or shoulder
- *Causes:* Gallbladder disease, pancreatitis, peptic ulcer disease
- *Characteristics:* Pain perceived at a location other than the site of the underlying problem

6. ***Visceral Pain***

- *Location:* Internal organs, such as the stomach, small intestine, or liver
- *Causes:* Inflammation, distension, or ischemia of the affected organ
- *Characteristics:* Deep, aching pain

7. ***Somatic Pain***

- *Location:* Musculoskeletal system, such as the abdominal wall muscles
- *Causes:* Inflammation, injury, or strain
- *Characteristics:* Sharp, stabbing pain

8. *Inflammatory Pain*

- *Location:* Gastric mucosa
- *Causes:* Gastritis, peptic ulcer disease
- *Characteristics:* Pain caused by inflammation of the gastric mucosa

9. *Functional Pain*

- *Location:* Varies
- *Causes:* Irritable bowel syndrome (IBS), functional dyspepsia
- *Characteristics:* Pain that occurs in the absence of any obvious structural or inflammatory cause