Paper-K (GIT and Hepatobiliary-2)

Table-3: MCQs

Subject	Total MCQs
Pharmacology	16
Pathology	41
Forensic medicine	16
Community	18
medicine	
PRIME	01
Medicine	11
Surgery	12
Pediatrics	03
Family medicine	02
Total	120

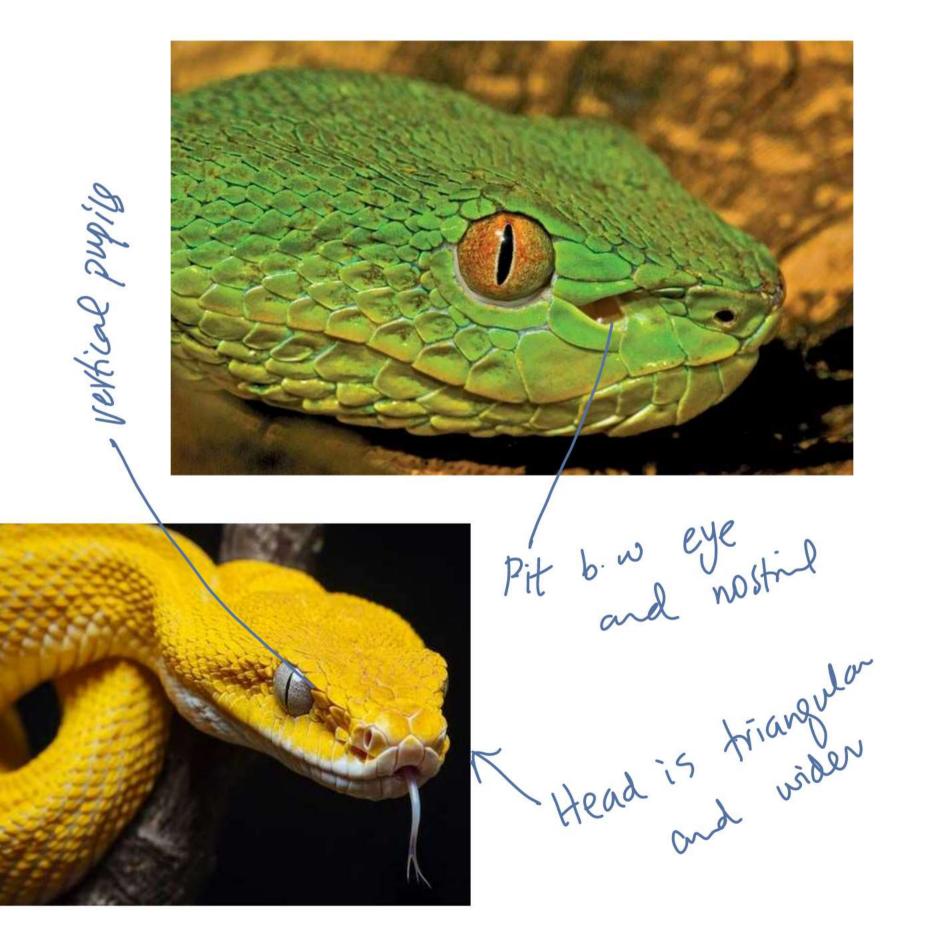
Table-4: OSPE

Subject	Viva stations	OSPE/OSCE stations	Total
Pharmacology	2	2	4
Pathology	2	2	4
Forensic medicine	2	2	4
Community medicine	2	4	6
Medicine (GIT examination)	х	1	1
Surgery (GIT/local examination)	X	1	1
Total	8	12	20

^{*} A minimum of 20 stations will be used in final exams. Total marks will be 120 (6 marks for each station).

Pit Viper

- · Head scales large
- · vasculotoxic



o neurotoxic

Cobra LElapid)

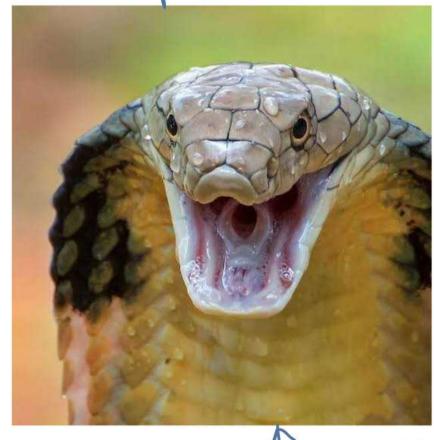
Indian cobra, or Indian spectacled cobra (Naja naja)

10 cm 4 inches

e cannot bite through dothing and inject complete poison

Roundpils

Fangs present antenorly but covered with a fold of mucus



King ropera





· Glistening black ocreamy white belly

Common krait

Alternate black and yellow bands



Bandled Krait



nostrils

Flanks have
yellowish white
une

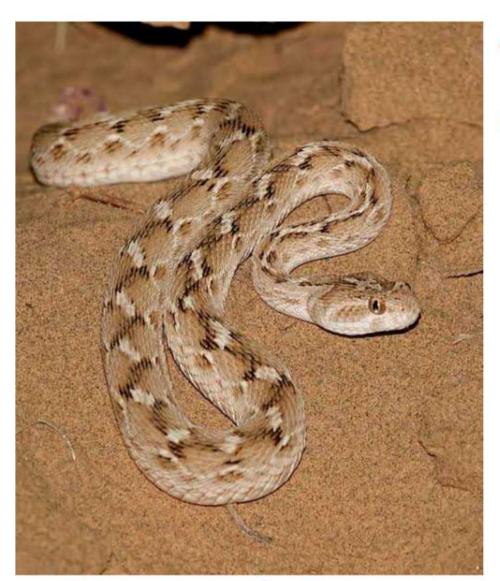
Common Green pit viper

· Three rows of black chamond shaped spots Terrific hissing

sound when about to bite

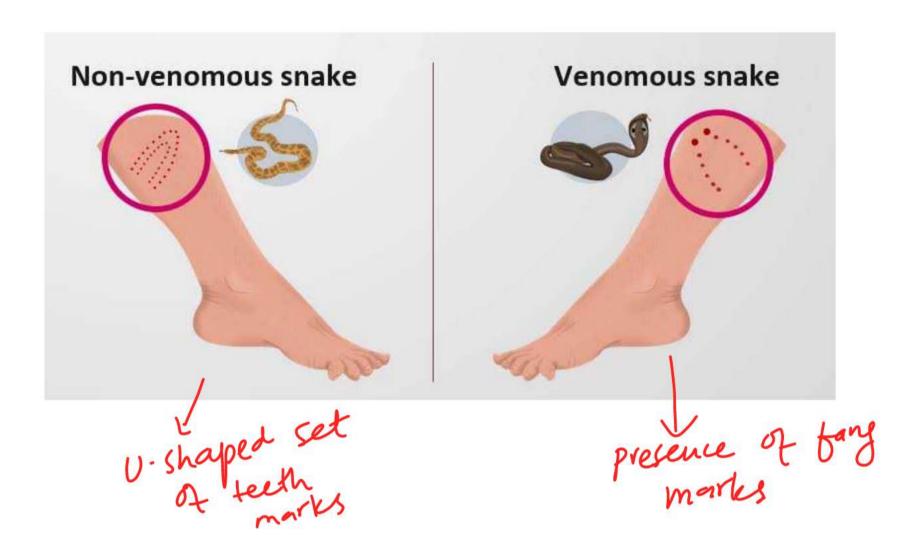


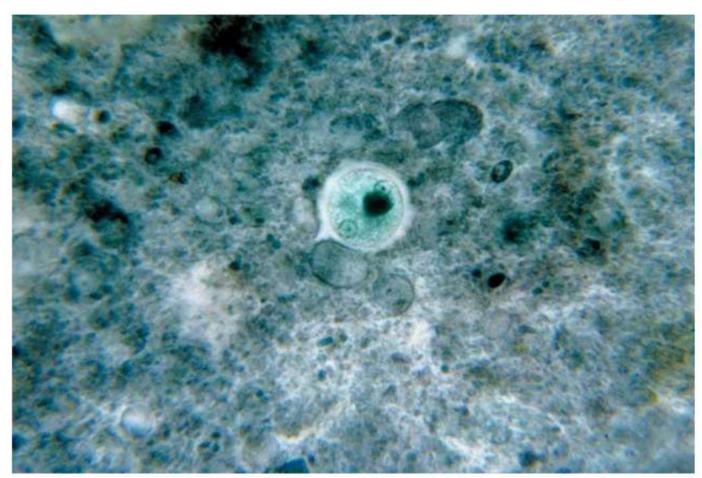
Russel's viper



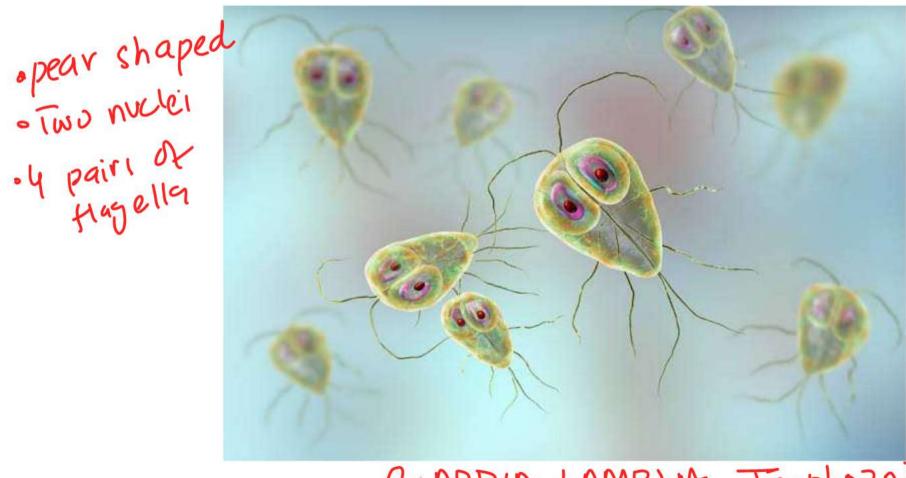
Saw Scaled yper

- · body scales serrated like
- · peculiar hissing sound when it moves



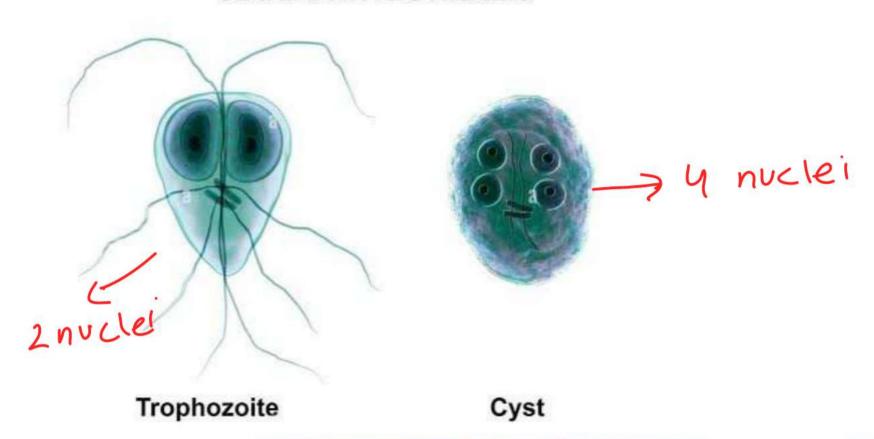


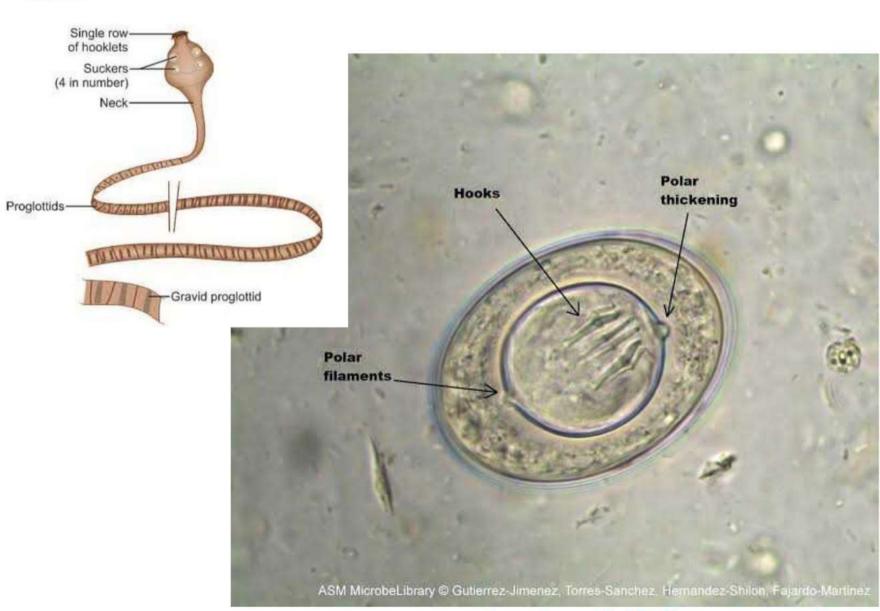
ENTAMOEBA HISTOLYTICA



GLARDIA LAMBLA Trophozoite

GIARDIA INTESTINALIS





Hymenolepsis nana (Dwarf tapeworm)

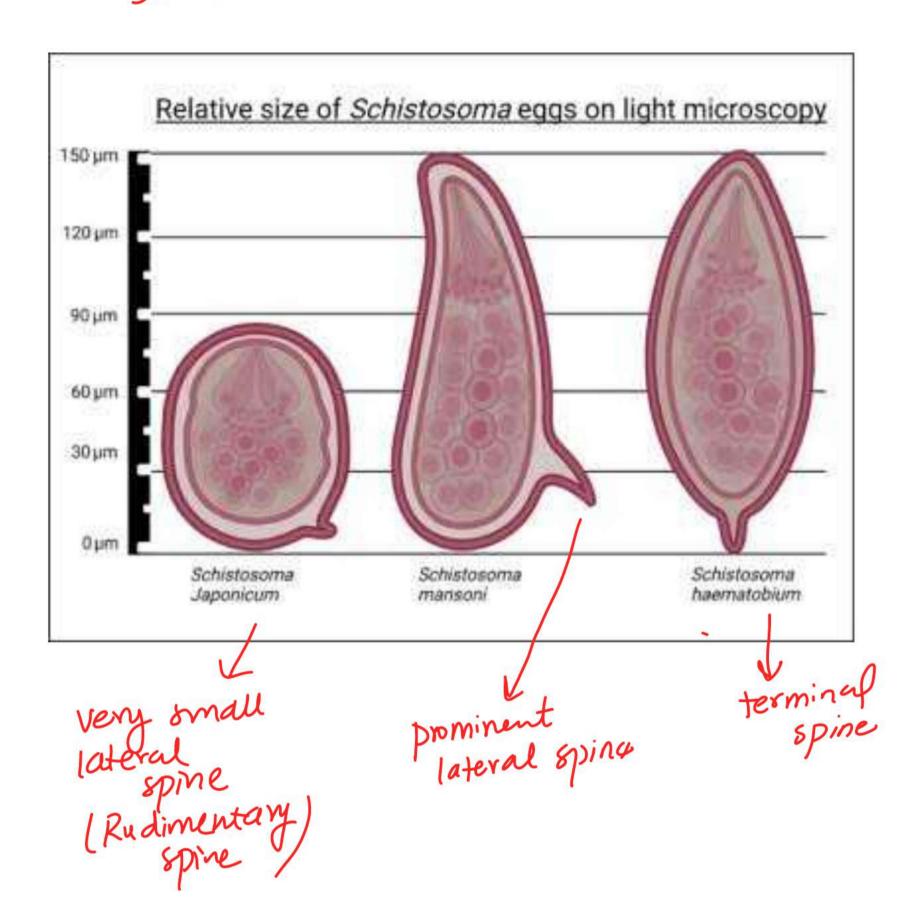


e lid like operwoom (operculum) · longest tapeworm

Diphyllobothrium Latum (fish Tapeworm)



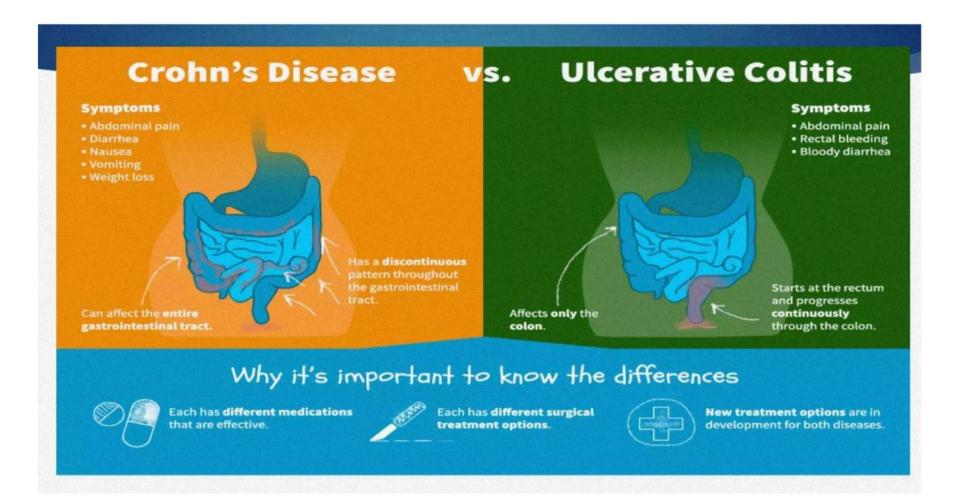
Schistosoma (Blood Fluke)



Ascaris Lumbricoides

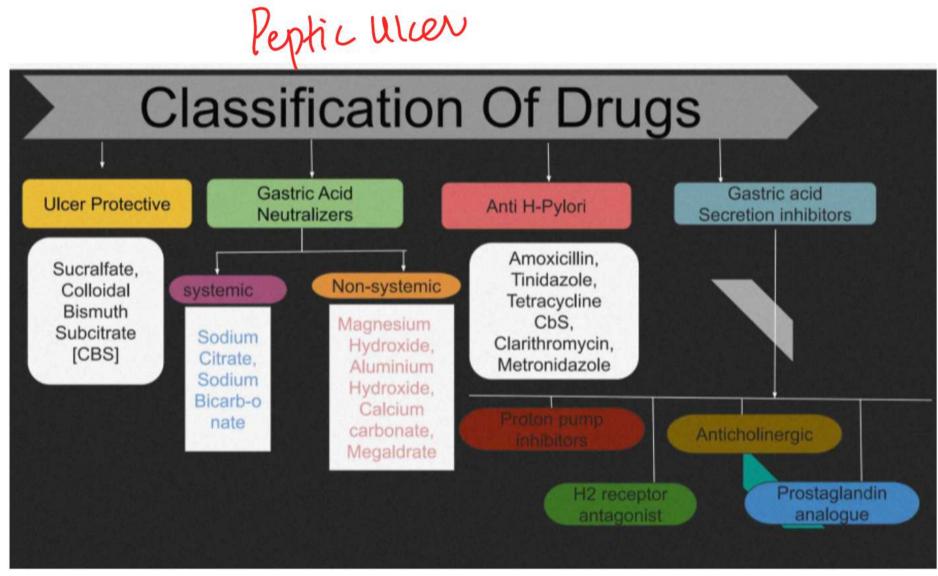


* egg is oval with an irregular surface



Differences

Ulcerative colitis	Crohn's disease
Continuous without skip areas	Segmental with skip areas (skip the corn) (C not for CContinuous and colon)
Commonly rectum, sigmoid colon	Commonly terminal ileum and/or ascending colon
superficial, confined to mucosal layers : Superficial inflammation	involves the entire thickness of the aff ected segment of bowel wall : Transmural inflammation
Fibrosis rare	Fibrosis common
Malignant change may occur if > 10 yrs	Malignancy rare



PAIN Location

- * Pancreatitis Epigastric pain
- * Biliary Colic Waxing and waning Right upper Quadrant
- * Acute cholecystitis -> Right upper quadrant pain, often radiating to right Scapula
- * Chronic Cholecystias -> Vague Right upper quadrant pain. especially after eating

Surgery Obstructive jaundice history Investigations Liver enzymes Pruritus: (It is thought that bile salts that deposit into the skin are responsible for the pruritus (itching)) Urine clr Stool clr **ERCP** Potassium-competitive acid blockers (P-CABs) pharma Vegetable poisons Hcn external postmortem Oxalic acid Lead

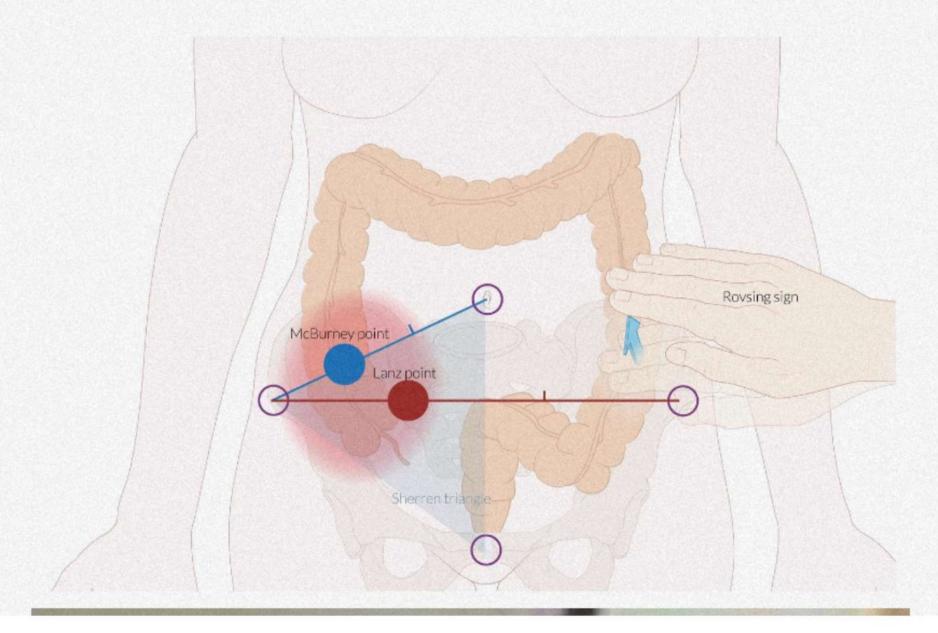
H pylori triple regimen prescription Enteric fever prescription Amoebicides classification

QUESTIONS (REMEMBER "PQRST")	THINK ABOUT
Provoke	
Does eating worsen the pain?	Pancreatitis, gastric ulcer, mesenteric ischemia
Does eating alleviate the pain?	Duodenal ulcer, gastroesophageal reflux disease
Quality or associated symptoms	
Is the pain associated with nausea and vomiting?	Pancreatitis, bowel obstruction, biliary colic
Is the pain "tearing"?	Aortic dissection
Is the pain "crampy"?	Distention of a hollow tube (ie, bowel, bile duct, or ureter)
Is the pain associated with emesis of undigested food?	Esophageal obstruction
Is the pain associated with emesis of undigested food with acidic, digestive juices from the stomach but no bile?	Gastroparesis or gastric outlet obstruction
Is the emesis bloody?	Gastroesophageal reflux disease, esophageal or gastric varices, PUD, gastric cancer, aortoenteric fistula
Radiation	
Does the pain radiate to the back?	Pancreatitis, duodenal ulcer, gastric ulcer, aortic dissection
Does the pain radiate to the right shoulder?	Biliary colic, cholecystitis
Does the pain radiate to the left shoulder?	Splenomegaly or splenic infarction
Does the pain radiate to the left arm or neck?	Myocardial ischemia
Severity	
Did the pain in your right lower abdomen suddenly improve from an 8 or 9 to a 2 or 3? (on a scale of 0 to 10)	Perforated appendix
Did the pain hurt the most at its onset?	Aortic dissection
Timing/treatment	
Is the pain continuous with intermittent waves of worsening pain?	Biliary colic, renal colic, small bowel obstruction
Are there multiple waves of pain that increase in intensity, then stop abruptly for short periods of time?	Small bowel obstruction
Did you recently take antibiotics?	Colitis due to Clostridium difficile
Does the pain occur once monthly around 2 weeks after the beginning of your menses, occasionally associated with vaginal spotting?	Mittelschmerz

· Clinical signs of appendicitis

- McBurney point tenderness (RLQ tenderness)
 - Tenderness at the junction of the lateral third and medial two-thirds of a line drawn from the right anterior superior iliac spine to the umbilicus
 - This point corresponds to the location of the base of the appendix.
- · RLQ guarding and/or rigidity
- o Rebound tenderness (Blumberg sign), especially in the RLQ
- Rovsing sign: RLQ pain elicited on deep palpation of the LLQ [8]
- Psoas sign: can be performed in two different ways
 - Can be elicited on flexing the right hip with stretched leg against resistance
 - RLQ pain may be elicited on passive extension of the right hip when the patient is positioned on their left side.
- o Obturator sign: RLQ pain on passive internal rotation of the right hip with the hip and knee flexed

The location of the pain may be variable as the appendix's location varies, especially in pregnant women. [9]



A 50 years old male presented with melena, abdominal pain and altered bowel habits for the last two months. His Hb is 8gms/dl. A colonoscopy was done which showed an infiltrative lesion in the rectum. Biopsy report shows irregular small glands lined by atypical cells.

Examine the photomicrograph and answer the questions.

What is the most probable diagnosis?

- (1)
- 2) Give two microscopic points of identification of this lesion?
- (2) (3)

3) Enlist three risk factors for this lesion?





A RIGHT-SIDED GROWTH



B. LEFT-SIDED GROWTH

Diagnosis - Colevertal Adenocarcinoma

2 microscopic points 4 Identifications.

, thyperchiomatic Hulli (pleo morphisms)

> Mucin pool

> invasion of Musularis propria

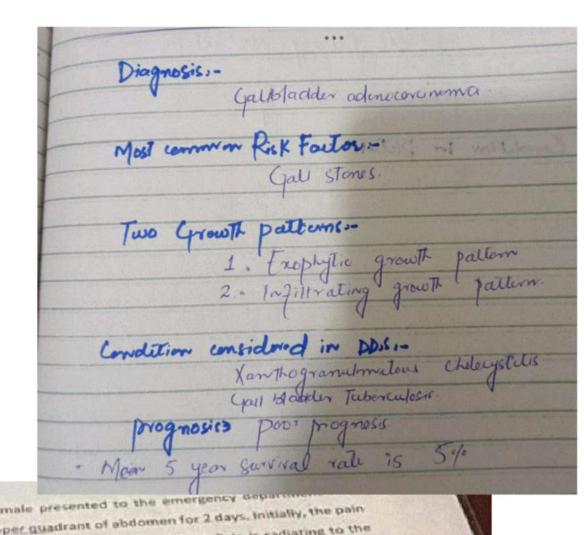
Risk Factors :-

-> low fiber diet

-> high intake of corbohydrates of
-> obesity

-> IBD

-> alcohol intake of Smoking.



This photograph is from a 54-year-old female presented to the emer 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.

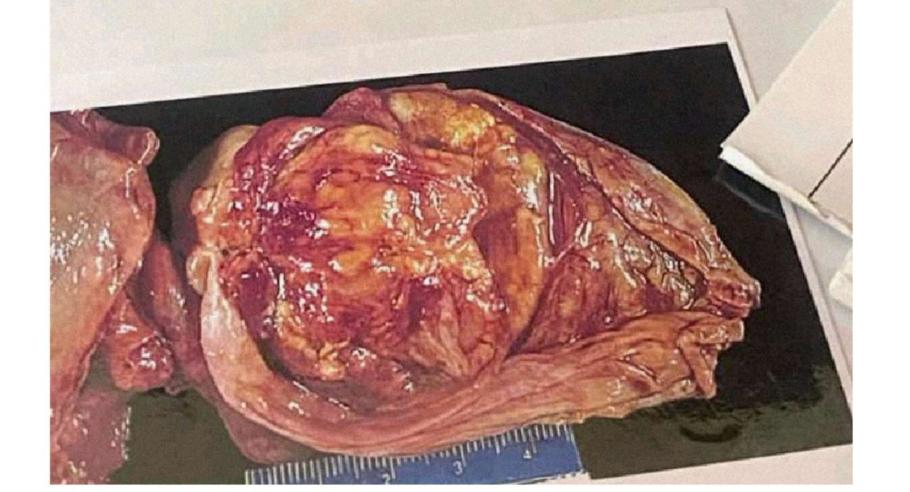
1. What is your diagnosis?

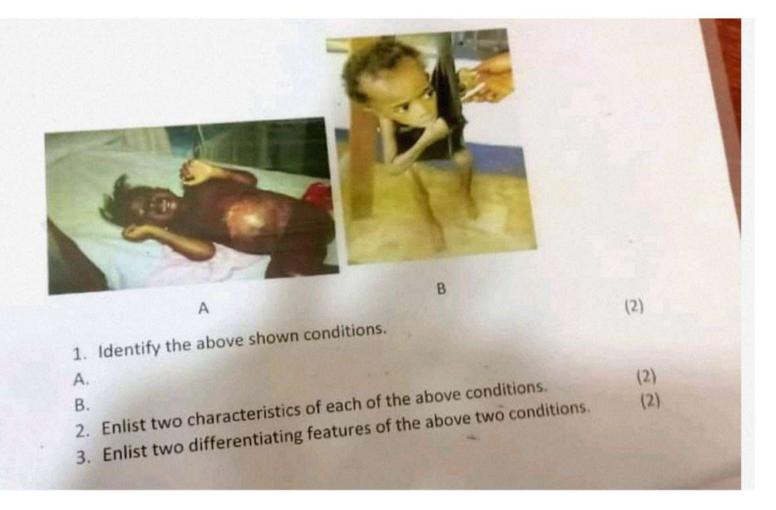
2. What is the most common risk factor for this condition?

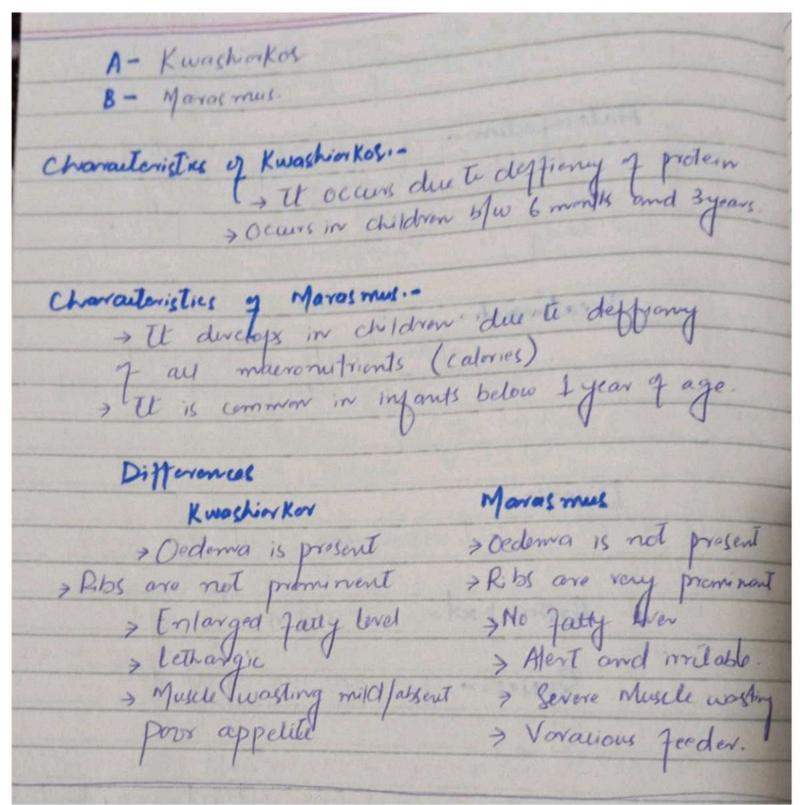
3. Name two growth patterns of this condition on gross and microscopic examination

4. What condition can be considered in differential diagnosis of this lesion?

5. What is prognosis of this condition?







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. Ireatment given and mother was warned about possible re infection.

1	Write the confirmatory test for diagnosis of pinworms infestation?	[1]
2		[2]
1	Describe differences between reinfection and autoinfection?	[2]
		[1]

+ Scotch Tape Test
> Suo sample examination under microsco
Make Children
wash wands with Coan
o i g chea
Donot depecate in open, always use tools

Recovered, and then again become injuled once, Recovered, and then again become injuled due to readination of the same organism.

Autoinfections— injulian by a pathogenic eyont already present in the body or injection fransferred from one part of the body to another.

Treatments—

> Albertable, Mebendasele

> pyromtel parnoal.

opic. nealth 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 to 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,	Ensure a smoke free healthy workplace
2	An applicability and scope statement,	Applies to all employees, visitors, and premises
3	An effective date,	Effective Date: 18.02.2025
4	A responsibilities section,	· Managemut -> Enforcement of policy · Employees -> Compliances
5	Policy statements	buildings and within 20 feet of entrances and exits
6	Background,	Tobacco use is a leading cause of preventable diseases and premature death. Reducing second hand smoke is essential to safeguard the health of all employees
7	Definitions, if any	Smoking -> The act of inhaling, exhaling or carrying any lighted tobacco product, including cigarettes, cigars, or pipes



(1 mark)

(1 mark)

(1 mark)

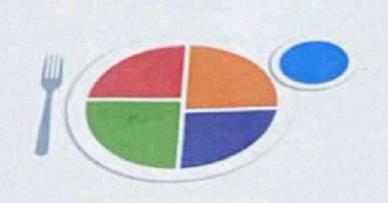
1. Identify the disease.

2. Which arthropod is responsible for causing this disease?

Name species of arthropod that cause this disease.

Enlist three preventive, measures to reduce the risk of this disease at the primary level.
 (3 marks)

Discose -Leshmeniasis (Culenous) Arthropod: Sandfly Speies .- phlebotomus papatasi + Avoid outdown artivities after dusk -> Use inseil repellent > Sleeping under Budnets > Secure window and dur screens > Use dolles which fully which fully



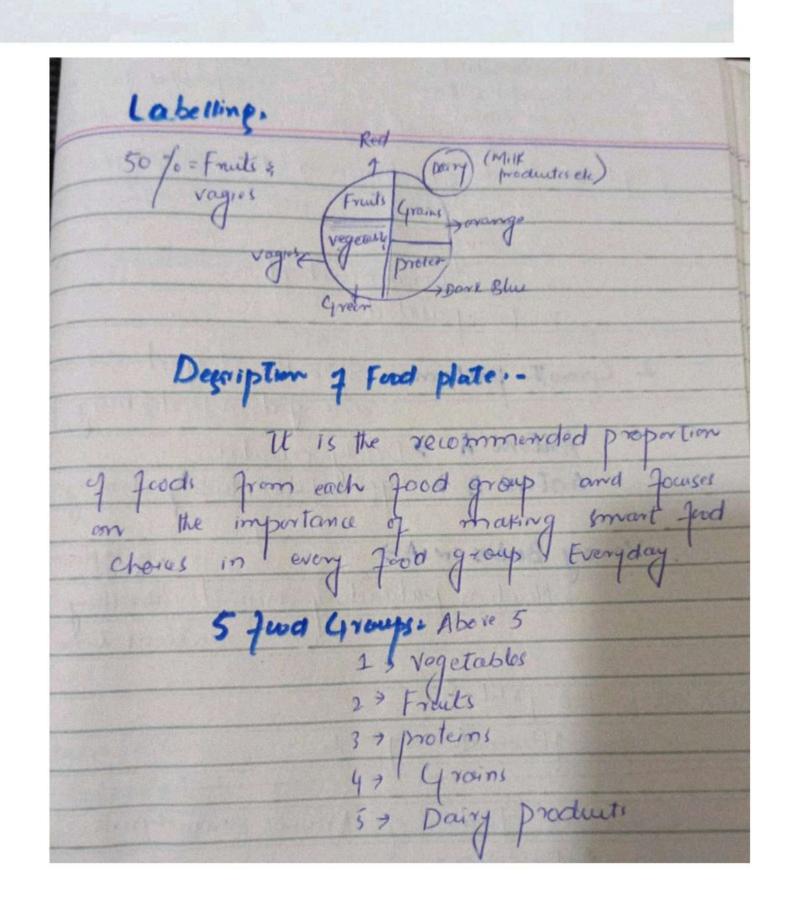


Observe the above image and answer the fo

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]



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 MOA

- * inhibit sulphydryl enzymes interfering with cellular metabolism
- * Locally, it cause irritation of mucus membranes and remotely depression of nervous system

Mechanism of Action of zinc phosphide and aluminium phosphide

- 1. *Release of phosphine gas*: When zinc phosphide comes into contact with moisture or acid, it releases phosphine gas (PH3).
- 2. *Inhibition of cytochrome c oxidase*: Phosphine gas inhibits the enzyme cytochrome c oxidase, which is essential for cellular respiration.
- 3. *Disruption of mitochondrial function*: Inhibition of cytochrome c oxidase disrupts mitochondrial function, leading to a decrease in ATP production.
- 4. *Cellular damage and death*: The decrease in ATP production causes cellular damage and death, particularly in tissues with high energy demands, such as the brain, heart, and liver.

Managemet of phosphorus poisoning

- . Gastric lavage with KMnOy
 - · Bowel is evacuated by brisk purgative
- · Antidote -> Dilute solution of Cusby



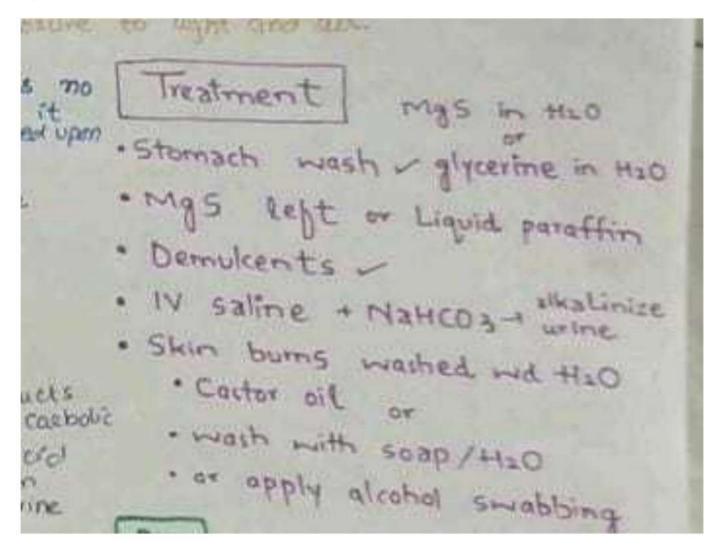
(a) Carbolic Acid/Phenol
Poison Class > Corrosive poison

(b) o Evodes The surface with which it comes in contact due to the corresive effect of Extract water from tissues

· Coagulate cellular profeire

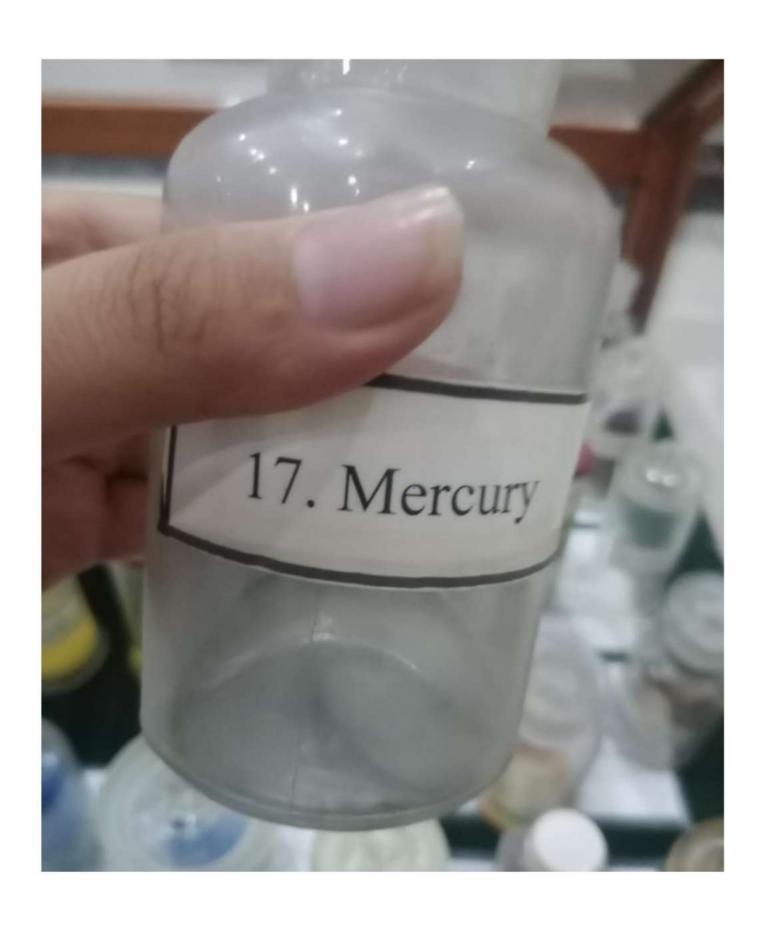
(c) Gastric Lavage using plan water to which some MgSO4 is added which some MgSO4 is added or milk . Demuleur or with so egg winte or milk may be helpful so diven bicartownate of saline with so diven bicartownate of saline with so diven be administered.

Management of phenol poisoning





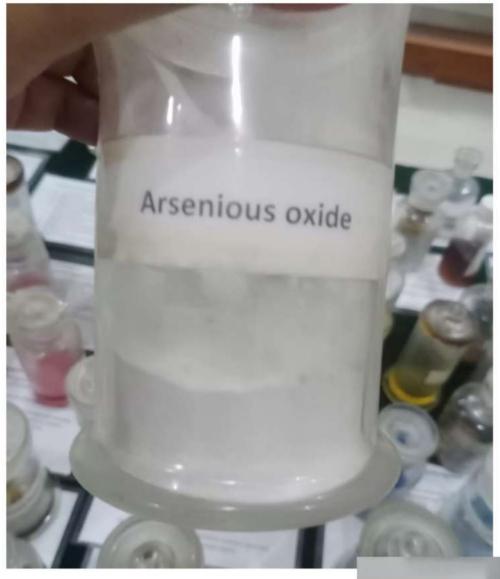


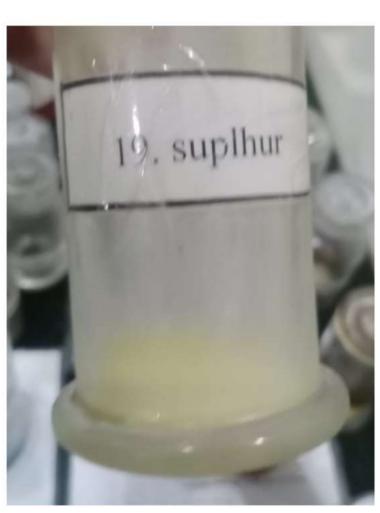






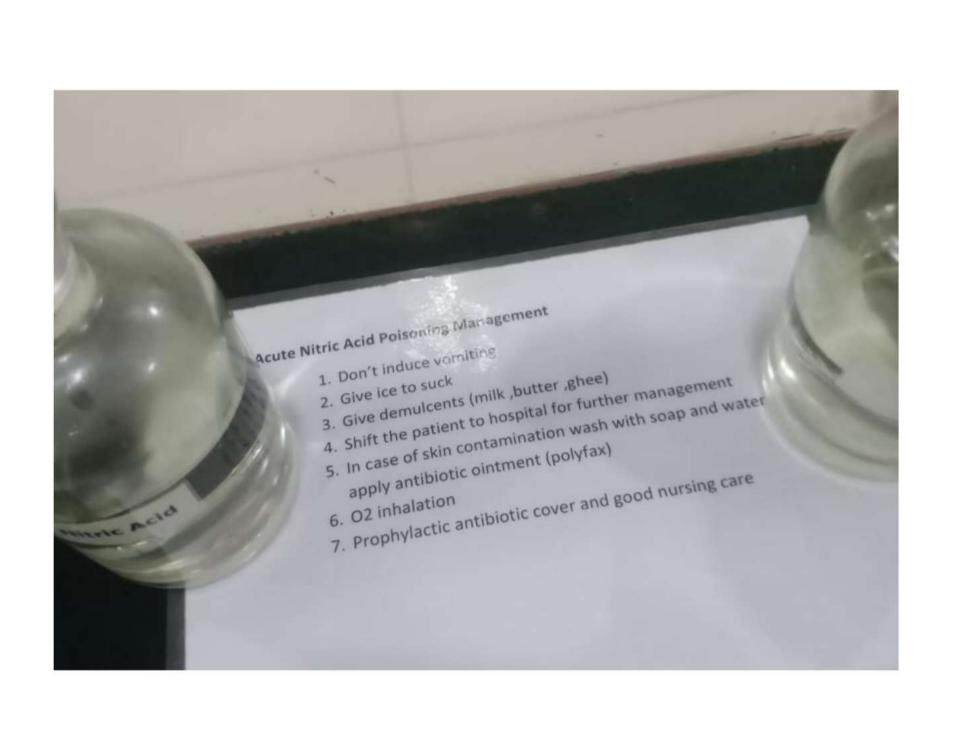




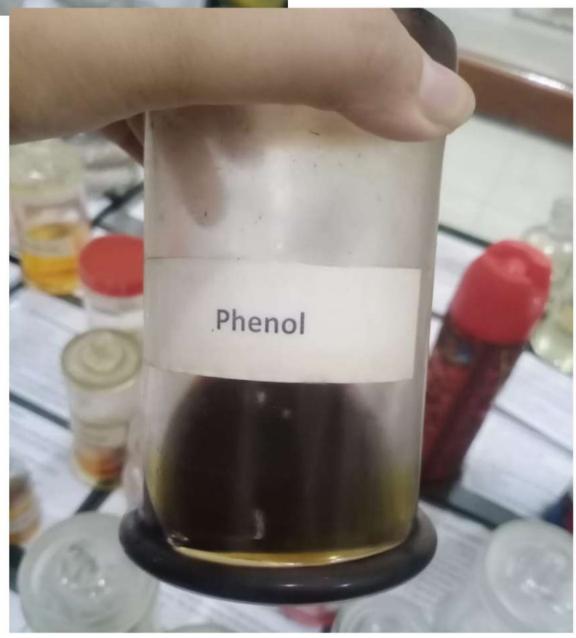






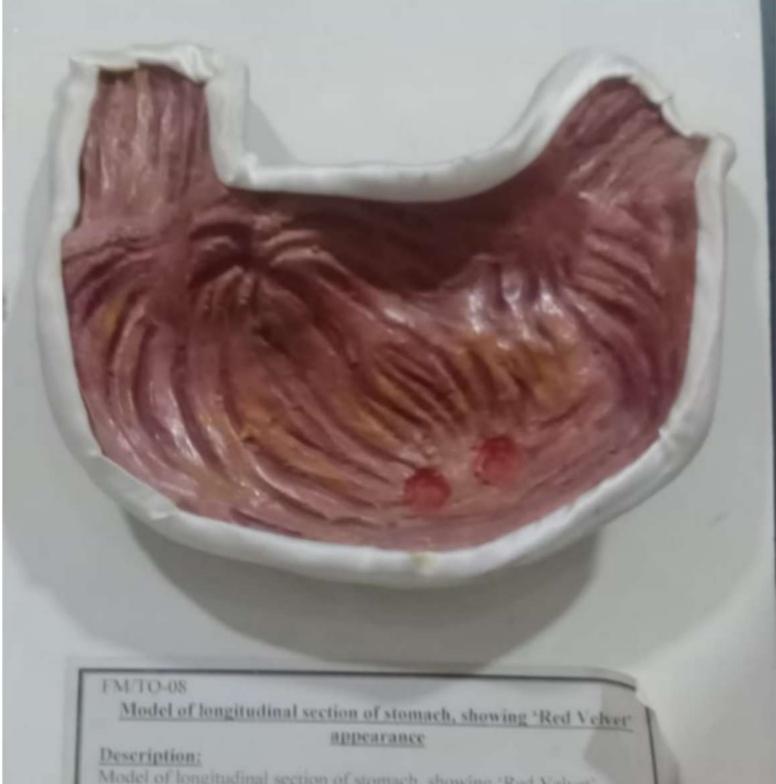












Model of longitudinal section of stomach, showing 'Red Velvet' Appearance seen in cases of ACUTE ARSENIC POISONING.

M.L.Importance:

Arsenic was used as homicidal poison since centuries but poisoning is very rare now a days. Accidental poisoning occurs due to its injudicious use as love philter or aphrodisiac.



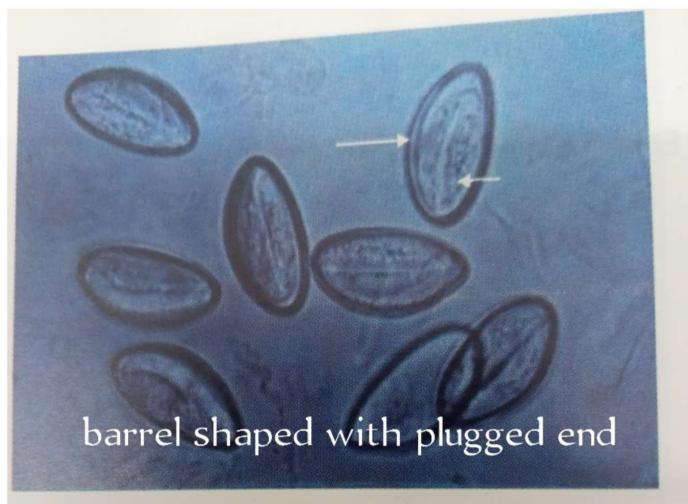


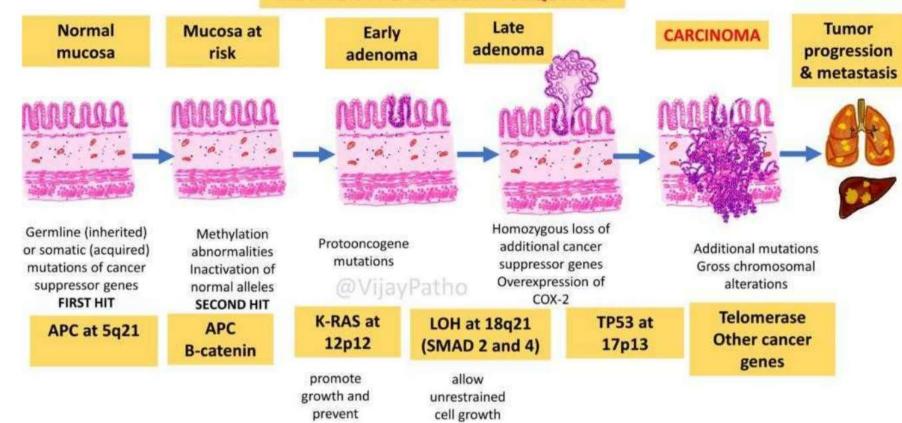
FIGURE 56–4 Enterobius vermicularis—eggs. Long arrow points to an egg of the pinworm, E. vermicularis recovered on "Scotch tape." Short arrow points to the embryo inside the egg. (Reproduced with permis-



egg of Ascaris. Note the typical "scalloped" edge of the Ascaris egg.

(Reproduced with permission from Public Health Image Library, Centers for Diseas Control and Prevention.)

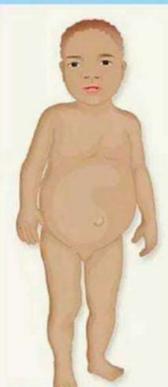
ADENOMA-CARCINOMA SEQUENCE



apoptosis

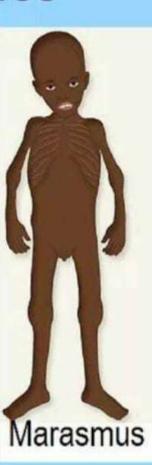
KWASHIORKOR VS MARASMUS

- In preschool children (1-5 years of age)
- Due to low protein intake
- Mild growth retardation
- Mild reduction in body weight
- Protruding abdomen and subcutaneous fat reserved
- · Ribs not very prominent
- Poor appetite
- Enlarged fatty liver
- · Oedema present
- Moonfacies
- · Sparse hair
- · Flaky paint-like skin
- Lethargic
- Requires adequate
 am unif protein



Kwashiorkor

- In weakened infants(<1 year old)
- Due to low calorie intake
- · Severe growth retardation
- Severe reduction in body weight
- Shrunken abdomen and subcutaneous fat not preserved
- Prominent ribs
- · Voracious feeder
- · No fatty liver
- · Oedema not present
- · An old man like face
- · No hair changes noted
- · Dry and wrinkled skin
- Alert but irritable
- Requires adequate amount of protein, fat and carbohydrate



Liver Function Tests (LFTs)

Mnemonic: "BAPA" (Bilirubin, Aminotransferases, Proteins, ALP/GGT)

Test	Normal Range	Increased In	Decreased In
Total Bilirubin	0.2 - 1.2 mg/dL	Hemolysis, Liver disease	Liver failure
ALT (Alanine Aminotransferase)	7 - 56 U/L	Hepatitis, Liver injury	Severe liver failure
AST (Aspartate Aminotransferase)	10 - 40 U/L	Hepatitis, Alcoholic liver disease	Severe liver failure
ALP (Alkaline Phosphatase)	44 - 147 U/L	Biliary obstruction, Bone disease	Malnutrition
GGT (Gamma-Glutamyl Transferase)	9 - 48 U/L	Alcoholic liver disease	Not commonly decreased
Albumin	3.5 - 5 g/dL	Dehydration	Liver disease, Nephrotic syndrome

3/22

Test	Normal Range	Increased In	Decreased In
Prothrombin Time (PT/INR)	11 - 13 sec	Liver failure, Vitamin K deficiency	Not commonly decreased

4TH PROFESSIONAL MBBS

BLOCK K

NON-INTERACTIVE

TOTAL MARKS: 06

A- What is Dane & Decoy particles. (1 Mark)

B- Write down at least 6 markers or antigens and antibodies for Hepatitis- B viral infection along with its importance? (4 Marks)

C- Predominantly which type of genotype does exist for hepatitis – C? (1 Mark)?

A. Dane particles are the complete, infectious form of the Hepatitis B virus (HBV), containing viral DNA, core, and surface antigens.

Decoy particles are non-infectious, smaller spheres and filaments composed of HBV surface antigen (HBsAg) without viral DNA.

B. Six markers for Hepatitis B and their importance:

HBsAg - Indicates active HBV infection.

Anti-HBs - Signifies immunity (past infection or vaccination).

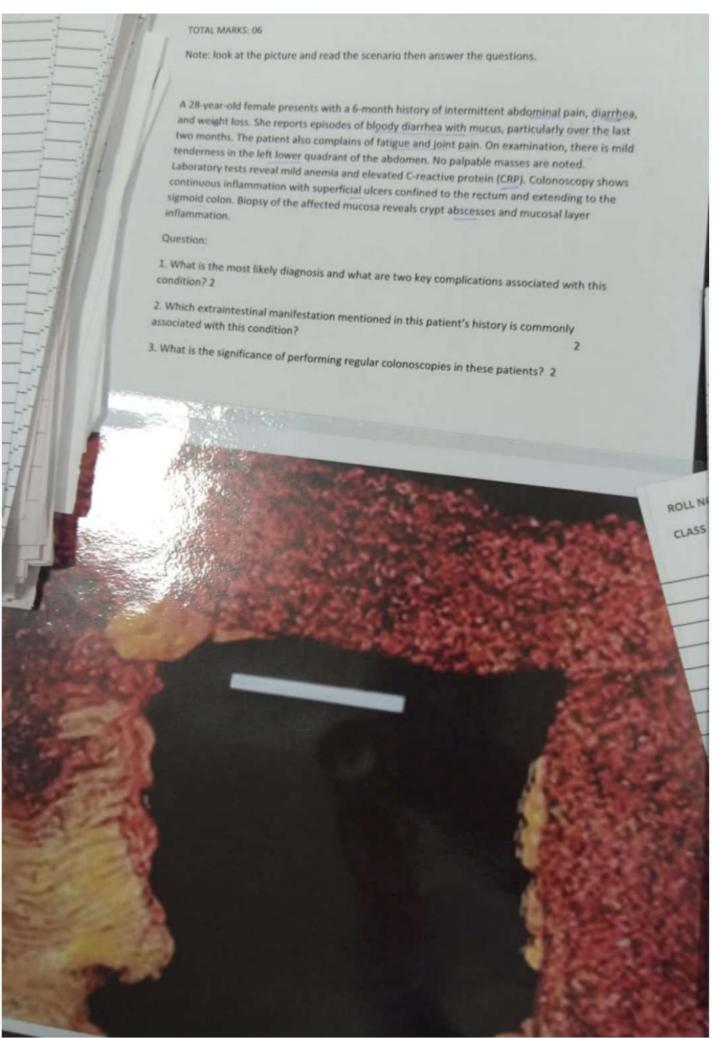
HBeAg - Suggests high viral replication and infectivity.

Anti-HBe - Indicates low infectivity and reduced replication.

HBcAg – found in liver cells, not in serum; indicates active replication.

Anti-HBc (IgM/IgG) - IgM indicates recent infection; IgG shows past infection.

C. The predominant genotype of Hepatitis C worldwide is Genotype 1, though other genotypes (2-6) also exist with regional variations.



Vicerative Colins

(a) · Dx > vicerative Colitis

. Complications

- Toxic Megacolon - Colorectal Cancer

(2) Rash Uveitir Arthrotis Primary Schenberry Cholangitis

(3). Dysplasia and Cancer Detection

- ASSESSIND mucosal healing - Early detection of complications

- Monitoring disease activity

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.
- o Adefovir: Inhibits <u>HBV DNA polymerase</u>, blocking viral replication.

3. Interferon Therapy

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

4. For Acute Hepatitis B

o Supportive care, no antivirals unless severe.

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.

Copper

- MOA: Inhibits cellular enzymes, causing gastrointestinal and hepatic toxicity.
- Fatal Dose: 10 g copper sulfate.
- Special Points: Greenish-blue vomiting, jaundice, and hemolysis.

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

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.

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.