~~Platelet Activating factor~~

PATHOPHYSIOLOGY (PYQ)

Q1) Patho & complication of D.M.

- DM is a group of metabolic disorders characterized by Hyperglycemia & abnormalities in Carbohydrate, protein & fat metabolism.

Etiology :-

1) Type I or Insulindependent - caused due to Immune mediated destruction, our I.S. attacks the B-cells of pancreas resulting in ABSOLUTE Insulin deficiency.
Hyperglycemia occurs when 80-90% of B cells are destroyed.

2) Type-2 or Non-Insulin dependent - It is caused due to :-

i) Obesity, Physical inactivity
ii) family history of diabetes
iii) Insulin Resistance - occurs when our cells of muscles, fats, liver dont respond well to Insulin & doesnt take up glucose.
iv) Relative Insulin deficiency.

S/S -
1) Feeling more thirsty
2) Polyuria ie frequent urination
3) weight loss
4) Presence of Ketone in urine
5) feeling weak & tired.

unhealthy lifestyle, obesity, family history of D.M.

↓

INSULIN RESISTANCE

(liver cells, adipose tissue, muscle cells become less responsive to insulin & less able to use glucose)

↓

So, Initially β-cells of pancreas work more to keep blood glucose level normal

↓

over many years, Insulin Resistance worsen & β-cells "tire out"

↓

↓ in Insulin sec^{n}

↓

Relative Insulin deficiency

↓ This causes

ADIPOSE TISSUE	LIVER	MUSLE
↓	↓	↓
Lipolysis	Glycogenolysis	Proteolysis
↓	↓	
Glycerol + free fatty acid	↑ Glucose	

↓

β-cells deteriorate & finally STOP producing Insulin

↓

D.M.

Treatment options - DM I is treated by Insulin therapy.

1) Metformin - Most prescribed medicine for type II DM
- It works by lowering glucose prodⁿ in liver.

2) Sulphonylureas - helps the body to secrete more insulin. Eg- Glipizide, Glimipride

3) SGLT-2 Inhibitor - Affects the blood filtering mechanism in Kidney & blocks the return of glucose to blood stream.
Eg- Dapagliflozin, canagliflozin

4) Thiozolidinediones - Makes body tissues more sensitive to insulin.
eg - Pioglitazone

5) Glinides - These are faster acting than Sulphonylurea but their effect is shorter.
Eg - Nateglinide, Repaglinide

6) DPP-4 inhibitor - Sitagliptin, Linagliptin

Complication

1) Cardiovascular disease - D.M. can damage blood vessel & ↑ the risk of developing heart disease & stroke. CAD

2) Nerve damage (Neuropathy) - High blood sugar level can damage nerves throughout the body, causing pain in body parts & numbness.

3) Kidney damage (Nephropathy) - D. can damage the kidney ~~heart~~. which can lead to kidney failure.

4) Eye damage (Retinopathy) - It can damage the blood vessels in eye leading to Blurred vision.

5) foot damage - Nerve damage & poor blood circulation can lead to foot damage & in severe cases amputation is the only soln.

6) Dental Problems - People with D. are at higher risk for gum disease, tooth decay.

7) High blood sugar can damages the nerves of Digestive system, leading to delay in gastric emptying, Nausea & Vomiting & bloating.

---

(02) Patho of HIV

AIDS (Acquired Immunodeficiency Syndrome) is caused by HIV (Human Immunodeficiency virus).

- AIDS is a chronic Immune system disease.

Etiology - HIV is caused by a virus. It ~~can spread through sexual contact, used drug inj blood in contact~~

→ It can spread by contact with infected blood.
→ HIV is a STI (Sexually transmitted disease), unprotected sex with infected partner can cause HIV.
→ sharing needles with infected person
→ transmission from mother to child during Pregnancy, childbirth or breastfeeding.

S/S - It depends upon phase of infection:

1) Primary Infection (Acute HIV) - Some people develop a flu-like illness. which may last for a few weeks. - fever,
Headache,
cough,
Diarrhea,
Sore throat, muscle aches

2) ~~Chronic HIV~~ - these symptoms can be so mild that you might not even notice them. However, the amt. of virus in your bloodstream is quite high. As a result, the infection spreads more easily than other stages

3) Chronic HIV - HIV is present inside the body - many ppl. may not have any symptoms during this time.
This stage last for many years if one recives Anti-Retroviral therapy (ART).

4) Symptomatic HIV - virus continues to multiply destroy your immune cells with S/S of -
-fever, wt loss, swollen lymph nodes, pneumonia

5) Progression to AIDS - If left untreated, HIV typically turns into AIDS in around 8-10 yrs.
S/S include - sweats, chills, Fever, Skin rashes, weakness, fatigue

19/2/19

HIV transmission occurs via 3 primary modes.

- Sexual
- Parenteral
- Perinatal

↓

Infⁿ of HIV

1) **Binding** - HIV binds to receptor on the surface of CD4 cells

↓

2) **FUSION** - The HIV envelope & CD4 cells membrane fuse together, which allows HIV to enter inside CD4 cell

3) **Reverse transcriptase** - RT occurs.

HIV RNA

↓

HIV ~~DNA~~ DNA

This allows HIV DNA to enter CD4 nucleus & combine cells genetic material, cell DNA

↓

4) **Integration** - Inside CD4 cell ~~nucleus~~ nucleus, HIV releases integrates (on HIV enzyme).

HIV uses Integrase, to insert its viral DNA to CD4 cell DNA.

↓

5) **Replication** - HIV begins to use the machinery of CD4 cells & starts making long chains of HIV proteins & thus these chains are building blocks of HIV

↓

6) **ASSEMBLY** - New HIV protein & new HIV RNA move to the surface of cell & assemble into immature HIV

↓

7) **Budding** - Immature HIV is pushed out of the cell & protease activates

immature into mature infectious HIV.

It is Diagnosed by Ag-Ab test

Treatment - Currently, there is no cure for HIV. However the complications can be prevented.

1) Everyone diagnosed with HIV shld be started on Anti-Retroviral therapy (ART)

2) NRTIs (Nucleoside Reverse Transcriptase inhibitors) -
They inhibits R.T.

3) Fusion inhibitors - Block entrance of HIV into CD4 cell

4) Integrase i - Inhibit integrase enzyme.

---

03 Patho of obesity.

- Abnormal or excessive fat accumulation leads to obesity.

A BMI over 30 is considered obese.

Etiology -

- usually obesity results from inherited, Physiological & environmental factors, combined with diet, Physical inactivity.

- Obesity occurs when you take more calories than you burn.

- your body stores extra calories as fat.

- It can occur by eating large amt. of fast foods that are high in fat & sugar.

- Drinking too much alcohol.

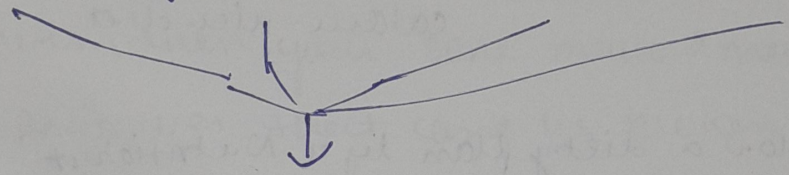
- Drinking too much sugary drinks.

- Physical inactivity,

. It can also be genetically, genes associated with obesity & overweight can pass from gen^{n} to generation.

→ An underactive thyroid gland (Hypothyroidism) can also contribute to weight gain

Excessive calorie intake | Excess intake of fastfood | Physical Inactivity | Excess alcohol consumption



Imbalance btw^{n} Energy expenditure & Energy intake

↓

Adipocytes Hypertrophy

↓

Adipocytes releases – Leptin (Leptin is a hormone that helps to maintain normal body wt.
The level of leptin in blood is Directly related to how much your body has fat).

↓

↑ leptin → ↑ in Body fat

↓

↑ leptin causes – ↑ in Satiety (satisfied feeling of being full after eating)
– overeating
- Excessive hunger

↓

↑ in lipogenesis (fat Synthesis) &
↓ in lipolysis (fat Breakdown)

↓

fat accumulation

↓

OBESITY.

Subdivided into :-

Class I - BMI - 30 - 35

Class 2 - BMI - 35 - 40

Class 3 - BMI - >40 → "Severe" obesity

- Non-pharmaco. treat.

1) → Lifestyle changes - It includes wt. loss, burn calories, less intake of high calorie rich diet

2) → Follow a dietry plan by a Nutritionist

3) - Exercise.

Pharmaco. treat. - ??

Complications of Obesity

1) Type II DM - Obesity can lead to Insulin Resistance.

↑ Blood sugar level

↓

DM

2) HTN - It can lead to high B.P.

3) CVS disease - Obesity can lead to development of atherosclerosis, a condn in which plaque build up in arteries & ↑es the risk of heart attack

4) fatty liver disease - accn of fats in liver causes infln & damage of liver.

5) Depression & other mental health problems

6) Sleep apnea - condn in which breathing is interrupted during sleep.

Q2 (A) Amyloidosis is a disorder characterized by extracellular deposition of an abnormal protein called Amyloid. which builds up in different organs & tissues. (as in question only pathogenesis is asked)

Etiology - It has several causes depending on the types type.

1) AL amyloidosis - (Immunoglobulin light chain Amyloidosis)
- MOST common type. & also called as Primary Amyloidosis.
- It happens when your bone marrow makes abnormal Antibodies that can't be broken down

2) AA amyloidosis - Also called - 2° Amyl. It is caused due to - Inflammatory disease such as - Rheumatoid arthritis or IBD - such as ulcerative colitis or crohns disease.
- The infl^n triggers the prod^n of SAA which accumulate in various organs.

3) Hereditary Am - It is caused by mutations or in Ap. genes that leads to prod^n of abnormal proteins.
- An abnormal protein called TTR (Transthyretin) is usually the cause.

4) Age-related - Mostly occurs in older mens. deposition of TTR protein.

5) Dialysis Related - occur in people who have been on long-term dialysis treatment.

Chronic Infⁿ or IBD

↓

↑ in SAA Protein

↓ Incomplete Proteolysis

AA Proteinaicⁿ

↓

AA amyloidosis

Transthyretin (TTR)

Mutation

↓

Mutant TTR

↓ Aggregation

ATTR Protein accumulation

- Mutation

↓

Bone marrow makes abnormal proteins

↓

Immunoglobulin light chains (Abs)

↓ incomplete Proteolysis

AL-Protein accumulation

↓

AL- amyloidosis

Complications →

1) Kidney problems - Amyloidosis affect kidneys & cause Proteinuria, ie presence of protein urine. & can lead to kidney damage and kidney failure

2) Heart Problems - It affect the heart & cause Cardiomyopathy, a condⁿ in which heart becomes stiff & unable to pump blood. This can lead to C.V. problems.

3) Liver Problems - It affect liver & causes hepatomegaly ie enlargment of liver.

4) GI Problems - It affects GIT & causes symptoms such as - Diarrhea, constipation.

5) Skin Problems - It can cause skin lesions, purpura. Is a condn in which blood vessels leak & cause purple spots on skin

6) Resp. Problems - It affects the lungs & cause breathing diff.

7) It can also cause hormonal imbalance.

Q2(b) Pathogenesis of tuberculosis.

→ TB is a serious disease that mainly affects our lungs. It is caused by a bacterium called → Mycobacterium tuberculosis.

& It can spread when a person with illness, coughs, sneezes. This can put droplets in air & can infect other person.

Site of action of Mycobacterium TB - Pulmonary Alveoli as this bacteria needs O_2 for survival, they mainly target lungs.

S/S - (1) 1° TB infection - Most ppl doesn't get any sym. few ppl has flu like s.

(2) Latent TB - There are NO symp. during latent TB.

(iii) Active TB - Cough, cough with blood, Chest pain, fever, chills, wt. loss, Anorexia, Fatigue.

Patho

Entry of Mycobacteria into pulmonary alveoli

↓

Alveolar Macrophage detects the presence of pathogens & Phagocytize the mycobacterium into the cell. thus forming a Phagosome.

The lysosome present in macrophage generally forms phagolysosome & kills the bacteria.

But here, the lysosome doesn't forms Phagolysosome.

↓

So, the Mycobacterium tuberculosis remains inside the Macrophage.

↓

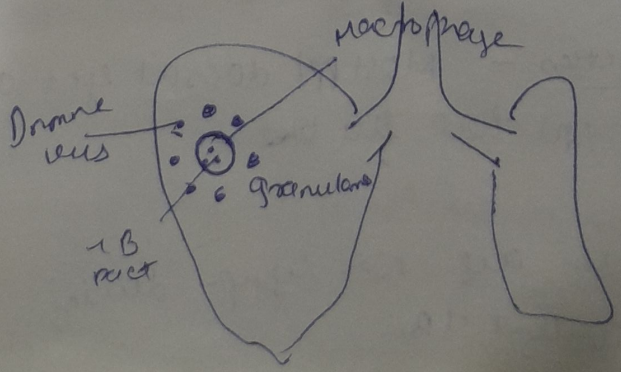
The bacteria starts Replicating inside macrophage

↓

Primary Infection occurs.

↓

3- weeks after i° Inf", the cell mediated immunity activates & surrounds the Immune cells around the site of Inf" & forms a GRANULOMA.



↓

Necrosis of tissues occurs in almost ~~that~~ that time is termed as ~~tumor~~ Gone focus.

↓

If lymph nodes are also involved then its called Gone complex.

↓

then, fibrosis & calcification of Gone complex occurs

↓

Elimination of tuberculosis.

Diagnosis - ~~CXR~~ CXR, Blood test, Sputum test. Here, the sample of your sputum is taken. If you have active TB in your lungs, lab test can detect it,

Skin test (TST test) - Here, a tiny substance called tuberculin is injected just below the skin 2-3 days. The test is +ve -> If there is a ~~bump~~ bump where the fluid is inj.

Treatment - options include Abs
- The most common treatment for active TB is Isoniazid,
- Rifampin, Rifapentine,

Is an exaggerated or inappropriate response occuring in respond to an allergen or antigen.

Types - I, II, III, IV.

I) Type-I or Atopic - Immediate response occurs after an allergen is exposed.

- Peak action time - 15-30 mins
- Mediated by - IgE Abs.
- Etiology - Genetics, Viral Inf^n, pollutants.

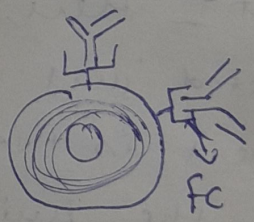
पथो)⇒ 1st Exposure to allergen

↓

allergen triggers the activation of T-helper type2 (Th2) cells.

↓ It stimulate

B-cells to produce IgE Abs.



↓

The IgE binds to fc receptors on Mast cells.

↓

Cell becomes Sensitized.

→ on Repeat Exposure to allergen

↓

The allergen binds to IgE Abs. on the sensitized Mast cells

↓

causing them to Release large amt. of Inflammatory medicines. such as-

↓

Histamine, leukotrienes, & Prostaglandins.

↓

these causes symptoms of allergic reactions, like - Redness, Swelling, Bronchoconstriction.

eg - Systemic Anaphylaxis

1) Adm. of Anti-Serum
2) Adm. of drugs like - Penicillin
3) Insect sting such as sting by bee or wasp.

Local Anaphylaxis

1) Hay fever due to pollen
2) Asthma due to allergy
3) food allergies - common food allergy includes - milk, eggs, peanuts.
4) Contact dermatitis - It's a skin rash caused by contact with an allergen - such as poison. Itching, Rash & Redness are seen.

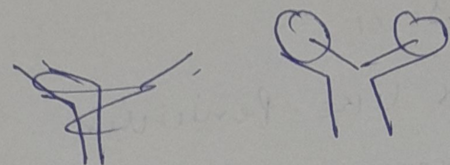
Type-2

Type-II or Cytotoxic - occurs when the Immune system produces Sp. Abs against ~~Ag~~ antigens resulting in tissue damage

- Peak action time - 15-30 mins
- Mediated by - IgG or IgM Abs
- Etiology - HLA-linked, Exposure to foreign substance

<u>पैथो</u>

Exposure to Antigen triggers the Immune System activation
↓
The Abs (usually IgG & IgM) are released & are binds to antigen.
~~On the~~



Ag - Ab - complex

↓

The bound Abs activates the <u>complement system</u>, leading to recruitment of Immune cells & destruction of the target cells.

↓

The I.C. releases Inflammatory mediators & enzyme that causes tissue damage & inflamm^n

↓

Type-II Hypersen^i Rxn.

- In some cases, the I.S. may mistakely recognize self-antigens as foreign leading to Auto Immune reaction & damages the body's tissue.

Eg - 1) <u>Hemolytic disease of new born</u> >
Here, the Mother's इम्यून सीस्टम प्रड्यूसीस एंटीबॉडीस that टारगेट & destroy ~~Abs~~ RBC of fetus leading to anemia.

2) Auto Immune Hemolytic Anemia - In this cond, the body produce Abs Abs attacks their own RBC, leading to Anemia

3) Goodpasture's syndrome - Abs attacks lungs & kidney of their own body.

4) Graves Disease - The I.S. produces Abs that stimulate thyroid gland leading to Graves disease

5) Myasthenia Gravis - I.S. blocks the transmission of nerve impulses to muscles. Leading to muscle weakness & fatigue

6) ~~Eg~~ Blood transfusion rxn

Type III - HS Rxn . Also klas Immune-complex mediated H.S.

occur when there is an excessive formation & deposition of Ag-Ab complex in various tissues. leading to tissue damage

- The I.S. produce Abs in response to Ag. which bind to Ag- to form Immune complex. These complexes deposited into various tissues & triggers inflammatory response.

Eg- Rheumatoid Arthritis

- Type IV - also klas Delayed type

- It is Ab - Independent.
- It takes several hrs to days to develop
- Mediated by T-cells
- Eg - Graft rejection, Granulomatus Inflammation
- The Rxn takes - 24-72 hrs. to develop

Q3 (a) Inf. mediators.

Inflm^n is the Wdays natural response to injury to infection.

Inflammatory mediators are substances produced by cells in response to tissue injury which then triggers inflammatory response

TYPES

1) Cell Derived Mediators
   - Vasoactive amines - Serotonin, Histamine
   - Platelet activating factor
   - Cytokines, Chemotrienes.
   - Arachidonic Acid metabolites (Eicosanoids)

2) Plasma - Protein Derived
   - Kinin System
   - Clotting System
   - fibrinolytic System
   - complement System

→ Cell Derived Mediators

These are प्रोड्यूसड बाय सेल्स इन रीस्पॉन्स टू इन्फ्लामेशन.

1) Cytokines

Types of Allograft rejection

It is the process by which the I.S. of recipient ~~of an organ~~ recognizes the transplanted tissue as foreign subst. & releases Immune response against it. leading to destn of transplant organ/tissue

टायप्स → 3 main types.

1) Hyperacute Rejection - Onset - Immediate. after transplantation.

- It is caused by pre-existing Abs in the recipient's blood that react with transplanted tissue.
 It is Rare. It can be prevented by careful screening of the donor and recipients prior to transplantation.

2) Acute Rejection - occurs within few months to weeks after transplantation.

- Most common type.
- caused by Immune Response.
- I.R. is against the transplanted tissue.
- It can be treated by immunosuppressive drugs.

c) Chronic Rejection - This type of rejection occurs usually years after transplantation.

- It is characterised by a gradual deterioration of the transplanted tissue.
- It is irreversible.
- It is difficult to treat & may require retransplantation.

s's of C.R include - ↓ed func^{n} of transplanted organ.

- ↓ urine output in case of kidney transplant
- Shortness of breath in case of lung transplant
- fatigue, weakness, chest pain in case of heart transplant.

→ Acute Rejection continue →

s's of AR - fever, swelling, pain, ↓ in func^{n} of transplanted organ.

- The diagnosis of AR is typically confirmed through biopsy of the transplanted organ

Treat - involves - Immunosuppressive drugs.

which help to suppress the I.S. & prevent it from attacking the transplant organ - these includes.

Eg - Corticosteroids, Monoclonal Ab.

OSCE - Pathology of IBD

IBD is a group of intestinal disorder that involves chronic inflammation of digestive tract.

Types

1) Ulcerative colitis - Inflamm^n & ulcers in large intestine (colon) & Rectum.

2) Crohn's Disease - It affects any part of GIT.

S/S - Diarrhea, stools with bleeding, Abd. pain, fatigue, wt loss, Anemia

Diagnosis - Colonoscopy - to view your colon, during this process, a small sample of tissue (Biopsy) may be taken.

2) Upper Endoscopy

3) x-ray of Abd. area.

4) Blood test

Etiology → (1) Genetic - 1 in 4 have family hx of IBD.

(2) Environmental pollution.

(3) Diet, smoking, alcohol

(4) Autoimmune - In IBD, our I.S. mistakes food as foreign substance & attacks it.

Patho

Q4 Cab Tumor is an abnormal growth of body tissue. which can be cancerous or Non-cancerous.

class

1) Benign - There are non-cancerous. & do not spread to other parts of body.

2) Malignant - There are cancerous tumors & can invade nearby tissues & organs & can spread to other parts of body. They are life-threatening.

→ Malignant t. are further classified into on the basis of type of cells they originate from:-

3) carcinomas - There tumors arise from cells that make up the skin, glands or other internal organs.

4) Sarcomas - Tumors originate from cells in connective tissues such as - Bone, cartilage

5) Leukemias - There are cancers of blood-forming cells. & involves bone marrow.

6) Lymphomas - There tumors arise from cells of Immune system mainly lymph nodes. & Lymphatic tissues.

Biology of Tumors

Tumors are abnormal growth of cells that can arise in any part of body.

This abnormal growth can be caused by mutations or changes in DNA of cells.

The biology of tumor is complex & varies depending on the type of tumor & its stage of development.

It can generally divided into 4 phases.

1) Abnormal cell growth - Tumor cells grow & divide more rapidly than normal cells & form a mass.

2) Angiogenesis - Tumors need a blood supply to grow, so they can stimulate the growth of new blood vessel to supply the tumor with nutrients

3) Invasion & Metastasis - Some tumors invade nearby tissues & organs & also spread to other parts of body through the blood stream & forming new tumors in other organs

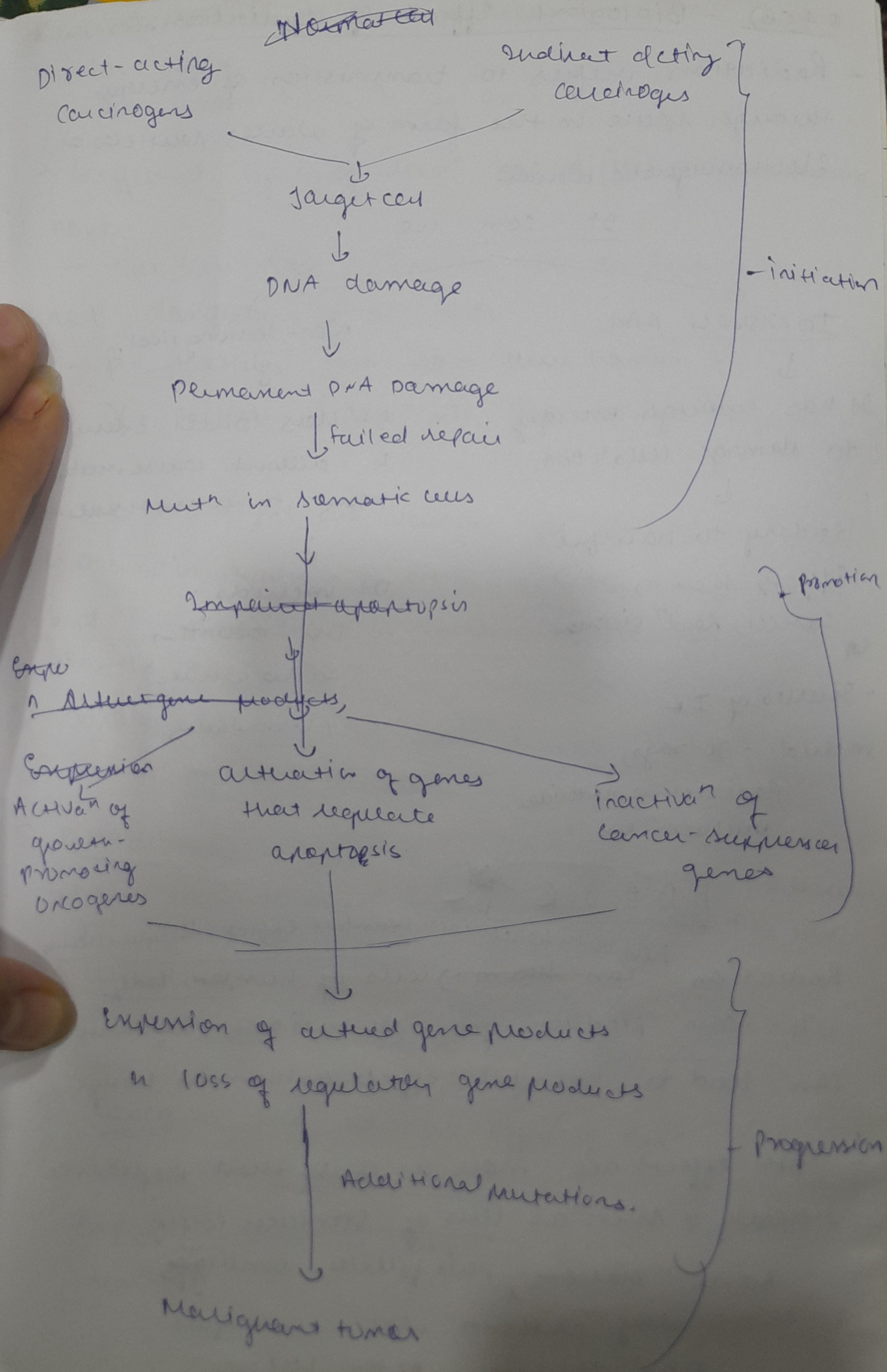
4) Immune System Evasion → Tumors can also invade the body's Immune System.

The biology of tumors is also influenced by microenvironment in which they grow.

- Tumor interact nearby cells & can alter the surrounding tissues. eg - tumor cells secrete growth factors or other signaling molecules that stimulate the growth of vessels & suppress I.S.

- Understanding Bio. of tumor is imp for developing effective treatment.

- Many cancer therapies target specific aspects of tumor biology such - Blocking angiogenesis & Boosting I.S


Normal cell
Direct-acting carcinogens
Indirect acting carcinogens
Target cell
DNA damage
Permanent DNA damage
failed repair
Mutn in somatic cells
Impaired apoptosis
Expn
↑ Altered gene products,
Expression
Activation of growth-promoting oncogenes
activation of genes that regulate apoptosis
inactivation of cancer-suppressor genes
Expression of altered gene products
↓ loss of regulatory gene products
Additional Mutations.
Malignant tumor
initiation
promotion
progression

Biological effects of radiation:

- Radiation refers to transmission of energy through space in the form of waves, particles or Electromagnetic waves.

It can be

IONISING RAD.

↓

It has enough energy to damage cells & DNA.

↓

leading to harmful effects such as cancer, Rad^n sickness.

Eg

- Sources of IR include - x-rays, Radioactive materials, Nuclear bombs

Non-Ionising Rad.

↓

It has lower energy & doesn't cause much harm to human tissues.

↓

It includes -
- UV radiation
- Radio waves
- Microwaves

B.E.O.R -

Rad^n has both beneficial & harmful effect they can harm cells of human body.

& can produce gene mutations which can lead to harmful effects on human body.

- Its effects are more on cells that reproduce rapidly - such as cells of stomach lining, bone marrow, hair follicles, embryos

& that's why patients who undergoes Rad^n therapies often feel :-

nausea & sick to their stomach,
lose hair
have bone aches & so on..

> Different types of rad" has different penetrating power.
- Our skin stops α-particle. So, α-particles are not dangerous to our body.
- β-particles can pass thru hands &
γ-particles penetrate the thick layer of

☆

1 → the UV light are necessary for life as they are involved in synthesis of vit. D & in photosynthesis of plant but excessive exposure can cause skin damage & ↑es the risk of cancer.

2) Ionising Radiation can cause damage to living cells, mut" of DNA & cell death.

3) DNA damage - Rad" cause damage to DNA molecules which leads to mut" & other changes & ↑es the risk of cancer.

4) Cell death - High dose of rad" can cause cell death, tissue damage & other problems

5) Cancer - The harmful effect of Rad".
- Exposure to rad" can cause ↑ in risk of cancer. Esp when there is excessive exposure to rad".

6) Radⁿ sickness - It is caused by high dose of Radⁿ. s/s include - N, V, diarrhea, dizziness.

7) Reproductive effect - Radⁿ can damage our reproductive cells ie Testes & ovaries ~~leading~~ which can lead to infertility or genetic damage in offspring

8) Acute Radⁿ syndrome - High doses of Radⁿ can cause ARS - common s/s - fever, N, V, skin burns.

9) long term health Effect — Exposure to radⁿ over a long period can ↑ the risk of chronic health probs such as - heart disease, stroke, cataracts.

(Q4(c)) Patho of cirrhosis

Cirrhosis is a late stage liver disease in which healthy liver is replaced with scar tissue & liver is damaged. ~~reversible~~ Permanent

Etiology - - liver tissues are damaged by long term alcohol abuse

- It can be due to hepatitis (B, C & D). Hep. is an inflammⁿ of liver.

non-alcoholic
→ ↑ fatty liver disease, can also cause cirrhosis, here fat is accumulated in liver.

- Auto immune liver disease - here the liver tissues are damaged by our own Immune cells
- cystic fibrosis.
  Destruction of bile ducts

S/s → Jaundice (yellowing of skin & eyes),
N, Abd. Pain, fatigue, loss of appetite,
Wt. loss, ~~fluid abnormalities~~.
fluid accumulation in Abd. ie Ascites.
- In women - absence of regular periods ie Menopause
- for men - Breast enlargement ie Gynecomastia.

Patho

Alcohol consuption	fatty liver disease	~~Hereditary~~ Wilson's disease	Hepatitis

↓

Red^{n} in oxidation of fatty acids & ↑ed in synthesis of fatty acids

↓

fat accumulation in liver cells. ↓ is called → liver Steatosis / fatty liver.

↓

This leads to an inflammatory response & causes → liver cell death & activation of cells called - Hepatic Stellate cells (HSCs).

↓

They produce matrix, collagen, leading to fibrosis

↓

As fibrosis progresses, the liver becomes stiff & loses its ability to func^{n} properly.

↓

Obstruction of blood flow & leading to

Portal Hypertension (Portal vein is a vein which carries blood from intestine to liver)

↓

Portal HTN can lead to bleeding; Hepatic Encephalopathy can also cause cirrhosis.

↓

Scar formation

↓

cirrhosis

OR(A) Risk factor & ~~Def~~ of pneumonia

Pneumonia is an infection that inflames the alveoli of lungs. The alveoli may fill with fluid or pus.

Rf –

1) Age - Infants & elderly people are at higher risk of developing pneumonia due to weak, Immune Syst.

2) Smoking – Smoking can damage lungs & weaken the I.S ~~making it easier for etc~~

3) Influenza – It is a flu, a resp. illness. & it ↑es the risk of pneumonia

4) Hospitilized patient – HP are at higher risk of developing pneumonia due to exposure to bacteria in hospital environment

5) Chronic Medical cond^{n} – such as lung disease, diabetes, liver disease can ↑ the risk of pneumonia

6) lung disease like COPD, asthma, cystic fibrosis ↑es the risk of pneumonia.

7) weak I.S.

Patho

Gastric Content Aspiration

(→ A cond^n in which the contents of stomach acid, food, & other debri like microbes are inhaled into resp. tract)

↓

Entry of bacteria into lungs

↓

① COLONIZATION - The bacteria colonize the upper resp. tract & it can be inhaled into lungs.

This bacteria (S. pneumoniae) produces certain virulence factors that help in colo. & invasion.

- This Bacteria is surrounded by a polysaccharide capsule that makes it resistant to phagocytosis.

↓

S. pneumoniae also produces toxins that triggers Inf^n

↓

② INFLAMMATION - Once, S Pneumoniae reaches LRT it can cause Pneumonia

NOW, S. pneumoniae reaches lower resp. tract → They trigger inflammatory response.

& cytokines & chemokines are released. & they recruit I. cells at the site of Inf^n.

& they try to phagocytose & destroy the bacteria

↓

leading to form^n of pus.

↓

③ TISSUE Damage - The Inf^n response causes damage of lung tissue, leading to oedema of alveolar mmp. impaired gas exchange & causes Resp. failure.

The bacteria also enters blood stream causing Bacteremia. S. pneumonia can also cross BBB leading to meningitis.

↓

The inflammatory mediators are also released into Blood stream causing - fever, chills & difficulty in breathing.

Etiology - The most common cause of bacterial Pneumonia is Streptococcus pneumoniae. other bacteria can also cause Pneumonia like - Haemophilus influenza

→ Viral Pneumonia can be caused by - diff. virus like - Influenza virus, parainfluenza virus.

→ fungal Pn. is caused by exposure to environmental fungi such as Aspergillus.

→ It can also caused by aspiration of stomach contents, inhalation of toxic subs. or chemicals.

Types → (1) Community acquired Pn. (CAP) - Most common & occur outside of hosp. or other healthcare facilities. Childrens are most affected. It is mostly caused by S. pneumoniae. S/S - cough, chestpain, diff in Breath, N, V.

2) Hosp acquired Pneumonia (HAP) - Occurs within hosp. when person is admitted & hospitalized.

S/S - cough with mucus, Fever, Shaking chills, shallow breathing, shortness of breath, chest pain, low energy, wheezing

Treat - Abs - Azithromycin (adults) & Amoxicillin (reduced) (high medication, Rest, Fever reduced, Inhaler

Q5(b) - Etiology & pathogenesis of COPD.

Chronic Obstructive Pulmonary Disease is a lung disease involving constriction of airways & difficulty in breathing. Chronic Bronchitis & Emphysema are the most common cond^n that cause COPD.

Etiology → COPD is mainly caused by Smoking.

→ It can also occur by Environmental factors- such as long term exposure to smoke, air pollution, harmful chemicals such as- in const^n site, mining.

→ Genetics disorder - low level of α-1 antitrypsin. It is a protein that helps to protect the lungs.

→* Emphysema - A cond^n in which the air sacs ie alveoli of lungs gets damaged & loses their elasticity.

→* Chronic Bronchitis - A cond^n in which airways become inflamed & produce excess mucus, leading to coughing & difficulty in breathing.

S/S -
- Shortness of breath
- wheezing
- chest tightness
- Chronic cough
- Feet swelling
- Frequent Resp. Infections.
- cough with mucus.

Pathogenesis

Smoking → ↓ level of α-1 antitrypsin ← Environmental factors

causes

CHRONIC BRONCHITIS

↓

Due to these irritants

↓

Hypertrophy & hyperplasia of goblet cells occurs.
(GC, synthesize & secretes mucus).

↓

Inflammation occurs & more mucus is produced to compensate the irritants.
(Mucus helps to trap smaller particles like - smoke & expel them out by cough).

↓

Cilia which secretes mucus becomes shorter & less efficient

↓

The mucus forms plug in alveoli

↓

Air trapping

EMPHYSEMA

↓

Alveolar macrophages undergo phagocytosis of these irritants

↓

Cytokines are released

↓

which activates Neutrophils

↓

Releases Elastase
(An enzyme that breaks elastin, which recoil the lungs)

↓

Breaks elastin and & components of alveolar wall.

↓

Damage of alveoli

↓

COPD

Q5 (c)

BENIGN	MALIGNANT
- An abnormal growth of cells that doesnt invade surrounding tissue.	- An " " that invades & destroys nearby tissues
- Doesnt not show Metastasis	- shows metastasis i.e spreads to nearby tissues.
- Slowly growing mass	- Rapidly growing mass
- Capsulated	Non - Capsulated
- These are well differentiated	- lack of differentiation
- Seldom recurs after surgery.	- Often recurs after surgery.
- Tumor cells stay attached to the cell mass & do not break away to start new growth	- Tumor cells can break & move to other areas.
- cells are not cancerous	- cells are cancerous.
- usually small in size	- larger in size.
- Resembles to tissue of origin.	- poor resemblance to tissue of origin.
- Secondary changes are less often	- Secondary changes are more often
- Can be treated with surgeries	- can be treated with therapies like - Chemotherapy, Radⁿ therapy etc..

Q6 (B) मलेरिया

Malaria is caused by Plasmodium parasite transmitted by the bite of infected mosquitoes.

C.f - People who have malaria usually feel sick with high fever & shaking chills.

→ fever, Abd. pain, cough,
chills, Dizziness, Rapid breathing.
N, V, muscle pain,
Headache

- Due to breaking of RBC, Malaria may cause Anemia.

Life cycle -

Malaria is caused by Plasmodium, a tiny protozoan.

Diff species of Plasmodium like -

P. vivax,
P. falciparum & P. ~~vivo~~ malaria

causes diff type of malaria.

① When this mosquitoes Bites human, Sporozoites are injected with bite.

② Sporozites reach humans liver & infect hepatocytes

③ liver cells Ruptures as Parasite reproduces asexually & thus bursting the cells & parasites (Sporozoites) releases into blood.

Human host

④ Parasite burst RBC causing - fever, chills & the released parasite infect new RBC

⑤ Parasite sexually develop in RBC

gametocyte

♂ ♀

⑥ female mosquito when bite This is humans, takes up gametocytes

mosquito host

⑦ fertilisation & development of them takes place in mosquitoes' gut.

⑧ Mature infective sporozites migrates to mosq's salivary gland

Q6(c) MI

- MI commonly klas heart attack occurs when blood flow to the heart is blocked & causes damage or death of heart muscle.

S's - Chest pain, discomfort,
Shortness of breath, dizziness
the pain may migrate towards arm, jaw, neck, shoulder or back.

Etiology -

1) CAD - Plaques build up to coronary artery causes CAD. & they restrict the flow of blood to heart.

2) Blood clot - the plaques that develop in Atherosclerosis can rupture, causing a blood clot. This clot might block an artery leading to ♡ attack.

3) High cholesterol level - since Plaque is made up of ch. there are ↑ chance of build up of Plaque.

4) High blood sugar level & High blood pressure can ↑ the risk of ♡ attack → 😳

Patho

Atherosclerosis → obstruction of blood flow

Arterial spasm → sudden transient obstruction

Plaque + Thrombus → occlusion

↓

ISCHEMIA

(A condn in which blood flow is reduced to any part of the body)

↓

HYPOXIA

(absence of enough O_2 in the tissues)

↓

~~Reduced~~ O_2 demand & Reduced blood supply

↓

~~Thrombolysis~~

Permanent Thrombus

↓

Necrosis

↓

M.I

Diagnosis - 1) ECG - ST elevation shows heart attack.

2) CXR - helps to see condn of ♡ & 🫁

3) Ultrasound of moving heart. - used to see how blood moves to ♡ & Valves

4) Blood Test

5) Angiogram - A thin tube (catheter) is inserted into artery & guided to heart. & usually done to see arteries.

Treatment

1) Anti-platelet drug - Eg - Aspirin, clopidogrel.
- It reduces blood clotting.

2) Clot busters (Thrombolytic) - to bust the clot.
Eg - streptokinase

3) Blood thinner - Eg - heparin

4) Nitroglycerin - A vasodilator

5) Statin - To lower Ch. levels.
Eg - Atorvastatin

Surgical procedures

1) Coronary angioplasty & stenting

2) Coronary artery Bypass

Q7 (A)

1) Hypertrophy - Term used to describe ↑es in size or volume of an organ, tissue or cell.
- Eg - Muscle hypertrophy which occurs when muscles fibres are subjected to repeated mechanical tension such as - wt. lifting (gym).

2) Hyperplasia - Term used to describe an ↑es in no. of cell or tissues.
- It can occur as a result of ↑ed cell division often in response to any growth factor
There are diff. types.

1) Physiological H.P - Due to any physiological condn. Eg - during Pregnancy uterus undergoes

2) Hormonal HP - Due to hormonal stimulation
Eg - Enlargment of Breast tissue during puberty or pregnancy.

3) Pathological HP - This occurs due to abnormal cell division.
This type of HP can be precursor to cancer

3) Granulomatous Inflammation - Is an aggregation of macrophages that forms in response to chronic inflammation, autoimmune infection.
Eg - o The cause which lead to form of GI. is tuberculosis.
- on one hand, granuloma help to prevent the spread of infection & on other hand they cause tissue damage

4) Metastasis - It is ability of cancer cells to spread from one part of the body to another.
Benign tumor doesn't show metastasis. while, malignant shows metastasis

5) - ?

6) Apoptosis - It is the programmed cell death.
- It occurs as a normal & controlled part of organisms growth.

07(B) types of Dysentery

- It is an intestinal infection that causes diarrhea containing blood.

There are 2 main types of Dys. - Bacillary & Amoebic.

1) Bacillary Dysentery - It is a gastrointestinal disease.

- A bacterial inf^n becomes severe causing inflam^n in the intestine.

Bacterial infection that lead to Bacillary dysentry.

Etiology - Shigella bacteria causes B.D.

- The bacterial inf^n are very contagious.

The bacteria are passed from person to person when fecal matter from an infected person gets into another persons mouth (hein ????)

- It might be due to poor hygiene.

- Most common bacteria lead to B D coli-
- shigella, which leads to Shigellosis.
- salmonella, which leads to Salmonellosis
- Escherichia coli which leads to E. coli inf^n.

S/S - Diarrhea, N,V, ~~vomiting~~, High fever, with blood.

painful Abd. cramps

2) Amoebic Dysentry - Is an infn caused by parasite that your body shed through stool.
When the parasite gets into intestine, it can cause

S'S - Cramp, diarrhea, N, V, upset stomach, wt. loss., loose stoolsss...

Etiology- Parasite that infects our intestine is Entamoeba histolytica., It enters your digestive system when you eat or drink Smtg that is contaminated with parasite.

BD	AD
- It is a bacterial disease caused by bacteria - Shigella.	It is caused by parasite - Entamoeba histolytica.
- Small amt of stool	- The amt of stool is relatively large
- Blood colored stool	- Dark colored stools
- Treated with Abs -	Tr with Antiprotozoal drugs
- ~~6-8~~ 10 motions / day	6-8 motions / day.
- frequent dehydration	Little dehydration.
- Acute onset of action	- Gradual onset of action.
- Odourless stool	- Odour is offensive (~~stool~~)
High grade fever	- Little fever.

Q7(c) Protein-calorie malnutrition.

PCM is a serious nutritional deficiency that occurs when a person's diet lacks sufficient protein & calories.

PCM is commonly observed in developing countries, where poverty, poor sanitation contribute poor nutritional status.

- It can lead to variety of health problems.

1) Kwashiorkor - It results from the lack of protein in diet. Commonly observed in young childs ~~arun~~ where diets are often deficient in protein.

S's → swelling of Abd, Skin lesions, distinte Adec Liver

- It can occur due to poverty, limited amt of food, ~~big~~ intake of food with high carbohydrate & low protein

~~Treat~~ - involves restoring the balance of protein & other nutrients in the diet thru.

~~Prevention~~ nutrient-rich diet - such as - nuts, legumes, eggs, meat, fish.

2) Marasmus - Severe form of malnutrition

~~Characterized by - Stunted growth~~, muscle wasting

Specifically - protein-energy undernutrition It results in overall lack of calories.

- Marasmus is deficiency of all macronutrients - carbohydrates, fats & protein

S's - Severely underwt, visibly depleted, stunted size, Starvation

3) Anemia - PCM can lead to deficiency in iron, which is essential for prodn of RBC. This results in anemia which is characterized by reduced no. of RBC & lack of O2 in body.

4) Vit. & mineral deficiency - A diet deficiency in protein & calorie leads to deficiency in essential vits & minerals. Such as - Vit A, B12 ~~etc~~, leading to health problems.

5) S/s of PCM - Irritability,
Patient looks weak & inefficient,
Diarrhea, wt. loss, hair fall,
Liver, kidney & heart failure,
The skin gets pale.

Treat - Oral feeding, Treat in poverty,
Starvation can be prevented by providing a balanced diet, Avoid lactulose.

Kwashiorkor	Marasmus
- develop in children where diets are deficient of protein	" " proteins & macronutrients
- Oedema is present	- oedema is absent
- Enlarged fatty liver	NO FL
- Muscle wasting absent	Muscle wasting present
- Needs protein in adequate amt.	needs protein, fats & carbohydrates,
- Occur in children 6 months - 3 yrs	btw. 1 yr of age