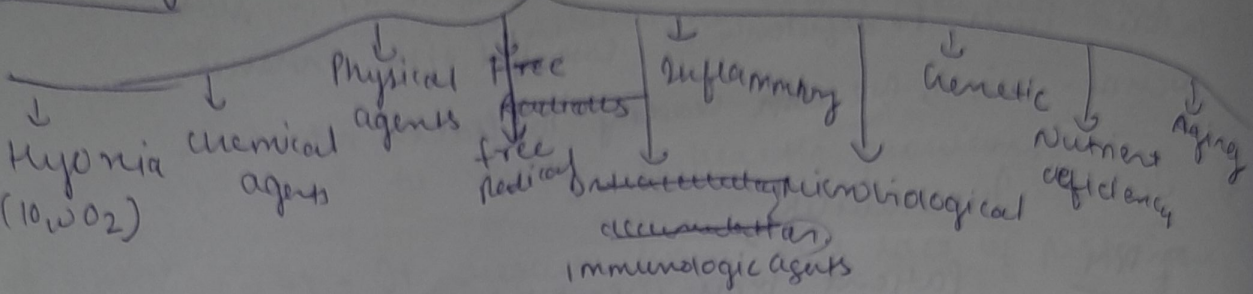
Cell Injury

It is a variety of changes that a cell suffers due to external as well as internal environmental changes.
- It is reversible.

Etiology — **Causes**



1) **Hypoxia & Ischaemia** – cells of different tissues requires O_2 to perform metabolic functn.
- Deficiency of O_2 ie hypoxia in tissues causes failure to perform these activities.
- Hypoxia is the MOST common cause of C.I.
- Due to their reduced supply of blood to cells occurs ie - Ischaemia.
- However, hypoxia can also result from heart diseases, COPD.

2) **Chemical Agents** –

Eg - Polycyclic hydrocarbons such as- Cigarette smoking
- Alcohols, Narcotic drug
- heavy metals such as- Mercury poisoning, lead poisoning
- Strong acids & alkalis
- Insecticides, pesticides, pollutants
- Some drugs can also cause cell injury.

3) Physical Agents - Mechanical trauma (like- Road accidents, burn, wound),
Exposure to radiation,
Thermal trauma (eg- by heat n cold)

4) Microbial agents - Injuries by microbes include- infection caused by bacteria, virus, fungi, protozoans etc.
Eg- Dysentry.

5) Immunologic agents - They protects our body against various injuries n may also turn lethal n cause cell injury eg - Hypersensitivity rxn,
Anaphylactic rxns,
Auto Immune disease.

6) Nutritional deficiency - It occurs due to insufficient amt of nutrients in diets
Eg- Marasmus, kwashiorkor.
Excess nutrients also cause disease- such as- heart problems, Yes cholesterol level

7) Aging or Senescence - Cellular aging leads to impaired ability of cell to undergo replication or repair which leads to cell injury.

Patho

- Any etiologic agents eg- Ischaemia

↓

Hypoxia in cells

↓

↓ O_2 in Mitochondria

↓

↓ Oxidative phosphorylation

↓

↓ ATP

↓ Na^+ Pump

↓

It used of efflux Na^+ & influx K^+ in cells but due to ↓ in Na^+ pump

↓

↑ in Na in cell due to which water & Ca^{+2} is too ↑ed inside cell

↓

Cellular swelling

Anaerobic glycolysis ↑ as O_2 is decreased

↓

↑ lactic acid

↓

acidic pH

↓

Clumping of nuclear chromatin

Detachment of Ribosome

↓

↓ Protein Synthesis

↓

Lipid deposition

↓

Reversible cell injury

2022 GTU

Q1(a) Inflamm is the body's natural response to injury. When tissues are damaged the body's I.S. responds by sending Immune cells to affected areas & fight with pathogens.

Inf. mediaters are the substance produced by cells in response to injury or inf^n, which trigger Inflammatory response.

Types

1) Cell Derived - Histamine,
Serotonine,
Prostaglandin,
Nitric oxide,
Cytokines, leukotrienes
Platelet activating factor

2) Plasma-Protein Derived - complement system,
Coagulation system,
Kinin system,
fibrinogen system.

1) Cell derived mediaters are ~~chemicals~~ mediaters that are released by cells in response to various stimuli.

1) HISTAMINE - Best KI chemical mediater which plays an IMP role in Inflammatory response.
It is released from mast cells & basophils.
- Histamine ↑es the permeability of blood vessels allowing Immune cells to enter the affected tissue.
& leads to Redness & swelling.

- Histamine here helps to limit blood loss from injured tissue.
- However, excess release of histamine causes chronic inflamm^{n} & tissue damage Eg- asthma & allergies

2) SEROTONIN - also klas 5-HT neurotransmitter.
- Released by mast cells & platelets.
- It causes vasodil^{n} & ↑es blood flow at site of infl^{n}.
- ↑es vascular permeability.
- It activate nerve endings & enhance the perception of pain, which happens in inflamm^{n}.

3) PROSTAGLANDINS - they are produced from lipids.
- It ↑ vascular permeability of blood vessel, allowing immune cells to move to tissues.
- causes vasodilation of smooth muscles & ↑es the blood flow to site of inflamm^{n}.
- when a blood vessel is injured, a prosta. called Thromboxane stimulates the form^{n} of blood clot to heal the damages.

4) CYTOKINES - they acts as chemical messanger ~~in~~ that signal other cells to mount an immune response (inflam^{n}).
- cytokines such as - Interleukin-1 & TNF-α are pro-inflammatory cytokines that promote inflamm^{n}.
- other cytokines such as interleukin-10 & transforming growth factor are Anti-inflammatory cytokines that counteract the effect of pro-inflammatory cytokines.

The balance b/w pro- & anti-inflammatory cytokines is imp for maintaining normal func^n & preventing excessive inflam^n.

3) NITRIC OXIDE - NO is produced by macrophages, neutrophils.
- It is produced in response to pro-inflammatory cytokines
- It causes vasodil^n of blood vessel

6) PAF - It recruit & activate D.C at site of tissue damage. It can also induce reactive O2 species (ROS) & prostaglandins which further cause tissue damage & inf^n.

2) Plasma-Protein derived - These are derived from plasma proteins & are synthesized in liver & released in blood stream.

1) THE COMPLEMENT SYSTEM - It's a complex system of plasma proteins. & causes:-
(i) Recruitment of Immune cells
(ii) Opsonization -
(iii) Activation of Immune cells
(iv) ~~Clearance of dead cells~~ Elim^n of pathogens
(v) Regulation of inflam^n

(2) KININ SYSTEM - These are activated during inflam^n

Kininogen is converted to.

↓ kallikrein or ACE

Bradykinin

↓

It is a vasodilator & ↑ vascular permeability.

- ACEi which is used for HTN can inhibit kinin system.

3) COAGULATION SYSTEM – Also klas clotting system.

During inflam, tissue damage & release of Pro-inflammatory cytokines activates CS

↓

This can result in production of thrombin

Thrombin stimulate release of more pro-inflamtory cytokines leading to inflammation

Thrombin can also activate Kinin system.

4) FIBRINOLYTIC SYSTEM – It contributes by formn of blood clot & recruitment of immune cells at site of injury.

When tissue damage occurs, f.s. activates leading to formn of fibrin clot which prevents blood loss

Q1 (B) Shock with ~~शॉक~~ ~~स्टेजीस~~ & ~~मैकेनीज़्म~~

- Shock is a life-threatening condn which occurs when body is not getting enough blood.

Types

1) Hypovolemic – It is due to loss of amt. of blood or other body fluids.

Causes – Bleeding from cuts or other body injuries, vomiting, diarrhea

Stages – (1) – loss of upto 15% of your body total vol. Blood vessels narrows slightly to maintain B.P. HR is normal.

Stage 2 – loss upto 20% of vol. Your HR rises your body pull blood away from limbs & sends to vital organs like – heart & brain

- you feel anxiety, rapid breathing

stage 3 loss of 30% of blood. your body stop making urine. your limbs are cold,
skin → Pale.
you might feel flustered & confused.

S-4 - loss of 40% of blood. Vital organs begin to fail. damage becomes irreversible. very low BP & no or weak pulse

Mechⁿ

HV. Shock
↓
↓ in O2
↓
Hypoxia
↓
Reversible cell injury
↓
Anaerobic metabolism
↓
↓ in ATP
↓
cell injury
↓
cell death

2) Cardiogenic - occurs when heart cannot pump adequate amt. of blood to organs & Blood may back flow to lungs (pulmonary oedema).

Stages A - A pat. is not experiencing any s/s but is at it's risk. These include pat. who has MI, heart failure.

Stge 2 - Begining of cardiogenic shock.

Stge-3 - Patient who has hyperperfusion.

4 - Shock worsens. deteriorating

5 - patient collapse

Right Atrial Pressure - It's

S's - low BP, weak pulse, rapid breathing.

3) ANAPHYLACTIC SHOCK - It is caused due to allergen. occurs when body encounters with that substance like - food. It causes allergy.

During anaphylactic shock, body releases large amt of histamine - which causes swelling,
shortness of breath,
N, V, Dizziness,
Hypotn.

- It is a medical emergency & requires immediate treat. of adrenaline. & anti-histamines.

4) NEUROGENIC SHOCK - life threatening condn caused by loss of ANS funcn.

It can occur due to trauma, brain injury, Spinal cord injury, nerve damage.

S's - loss of reflex, bowel dysfunc, Bradycardia.

In case of traumatic injury, Spinal stabalization & surgery is necessary.

5) SEPTIC SHOCK - It is severe form of sepsis, occurs when Inflamn in body triggers an inflammatory response leading to organ dysfunc.

S's - Hypotn, decrease blood O_2 level which may lead to organ damage., Inflamn, Altered mental status

In severe cases ventilation is also needed.

(Stages of Shock) (Grey Shock.)

1) Initial Stage — Shock

↓

Body switches from aerobic to anaerobic due to deficiency of O_2

↓

↑ lactic acid level

↓

2) Compensatory stage — Sympathetic N.S. activates

↑ catecholamine release

↑ H.R.

↓

Vasoconstriction occurs

↓

↑ B.P.

↓

3) Progressive stage — Electrolyte imbalance

↳ Resp. acidosis

↳ Metabolic acidosis

↳ Oedema

↳ Pale skin

↳ Altered level of consciousness

↓

4) Refractory stage — Cell damage

↓

Irreversible organ damage

↓

DEATH.

Q1(c) It refers to Inflamm^n of liver.

- It can be due to any viral Inf^n & Alcohol abuse.

4 types:-

1) Hepatitis A - Highly contagious liver infection caused by Hepatitis A virus.

- It can occur due to contaminated food & water

S's - N, V, Abd. pain, jaundice, dark urine, Dark urine.

- Hep A virus doesn't cause long term liver damage

- vaccine is available for hepatitis A.

2) Hepatitis B - Serious liver infection caused by Hepatitis B virus (HBV).

It can be acute (short term) & chronic (long term) & chronic can cause liver damage.

S's - Jaundice, Dark urine, loss of appetite, Abd. pain, N, V, fever.

It is caused due to -

1) Sexual contact
2) Sharing needles
3) Mother to child

Prevention involves - getting vaccinated, safe sex, avoid using sharing needles

Treat involves antiviral med^n.

3) Hepatitis C - caused by HCVirus (HCV).
It leads to liver inf^n & liver damage.
- It is usually spreaded thru contact with infected blood, sharing needles, from mother to child.
S's - Jaundice, poor appetite, Bleeding easily, pedal oedema, wt. loss;
- It can be acute & chronic
Acute H.C.V ~~can~~ doesn't cause much symptoms.
Chronic HCV leads to liver damage, cirrhosis, liver cancer.
- Vaccines are NOT yet available for HCV.
- antiviral chem^n are used.

4) Hepatitis D - also/as delta hepatitis.
caused by HDV.
- It is ONLY found in people who are infected with HBV.
- HDV cannot replicate by its own & requires HBV to replicate
S's are similar to those of HBV. Some people may not even experience S's at all.
HD can be acute & long term.
Chronic leads to liver damage, cirrhosis & liver cancer.
- Prevention in values getting vaccinated against H.B as HBV is a prerequisite for HDV.

5) <u>Hepatitis E</u> - caused by → <u>HEV</u>

- Is usually a <u>self-limiting</u> disease that resolves on its own. However, it can be dangerous.
- It is 1° transmitted thru <u>fecal-oral route</u>
  - also be transmitted via blood contacts.

<u>S/S</u> - N, V, Jaundice, loss of appetite.

- There is no sp. treat. for HEV.

Q2 unknown 2023

<u>Q3(a)</u> CCF is a long term condⁿ which occurs when heart is unable to pump enough blood. the fluids accumulates in various parts of body. like lungs causing shortness of breath

<u>Etiology</u> - <u>CAD</u> is a most common cause of CCF. Here plaque is buildup in arteries. this reduces blood flow to heart causing ♡ attack.

2) <u>HTN</u> - It forces the ♡ to work harder & extra wo[rk] can make heart muscle stiff & weak

3) <u>Heart Valve disease</u> - Valves maintain the blood in ~~atrium &~~ auricles & ventricles.

Valve problem can cause heart failure

4) <u>Myocarditis</u> - Inf^n of heart muscle.

5) ~~b~~ Congenital heart disease

6) Diabetes Mellitus.

7) Obesity,

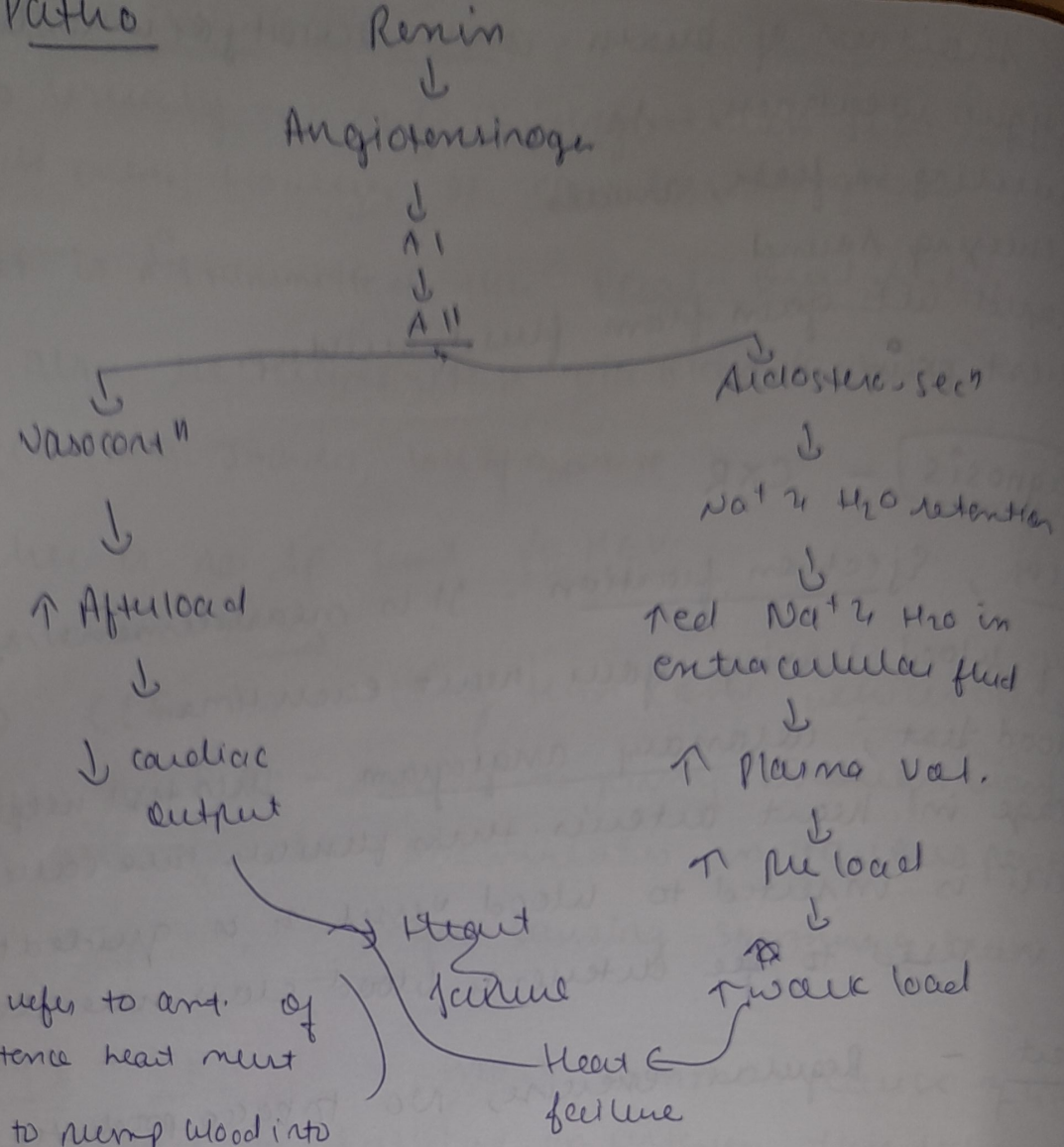
S/S – Shortness of breath while activity or when lying down.
- fatigue, weakness, ↓↓
- Swelling in feets, ankles
- wheezing sound
- Rapid wt. gain from fluid buildup.
- Chest pain

- Diagnosis – CXR
- ECG, Ejection fraction – It is measurement of the % of blood leaving your heart each time.
- Blood test; Coronary angiogram – This test helps spot blockage in heart arteries. thin flexible tube called catheter is inserted to blood vessel & is guided to M. It is used to see arteries & blood clots more clearly.

Treat – Regular exercise, NO tobacco products.

→ ACEi –, ARB, B-Blocker, Aldosterone antagonist, SGLT2i, diuretics, cholest

Patho



Inflammation

- a dynamic process & body defense rxn to eliminate the spread of infec" & removal of necrosed cell.

Types

1) Acute - Is of short duration. lasting for few mins to few days. Quick rxn.

2) Chronic - Is of longer dur". lasting from days to years. persist for a longer period

ACUTE is divided into 2 stages — vascular
— cellular

1) Vascular - Alteration in arteries, veins & capillaries is the earliest response to tissue injury.

↓

this causes → Haemodynamic Changes

↓

irrespective of the type of injury, Immediate vascular response is - Transient vasoconstn of arterioles to control blood loss.

↓

Persistant vasodiln occurs to ↑ the blood flow in nearby arteries & in microvascular bed.

↓

this causes Redness & warmth.

↓

Progressive vasodiln elevates local hydrostatic pressure (As blood moves along the capillary fluid moves out to intracellular space).

↓

transduction of fluid into extracellular space & causes swelling.

↓

leucocytic migration along the vascular Endothelium (Lining of cells) & then leucocyte moves to extracellular space.

↓

Accn of oedema fluid

↓

swelling.

2) cellular — Emigration of leucocytes

— Phagocytosis

The escape of leucocyte from lumen of microvasculature to interstitial tissue is the MAIN inflammatory response.

↓

WBC migrate to surrounding tissue & towards microorganism

↓

Phagocytosis occurs

↳ engulfment

↳ death & digestion of microorganism

↳ Discharge of waste material

↓

Redness & Inflamm^n

2) CHRONIC - is also klas systemic Infl^n

eg - chronic arthritis.

~~Stimulus~~ Injury or Infection.

↓

Acute Phase Response

↓

Recruitment of Immune cells at the site of Injury

↓

This cells release Inf. mediators such as

cytokines, chemokines.

↓

Anti - Inflammatory cytokines

IL6, IL1, TNF

↓

Liver cells release

↓

CRP ← fibrinogen

↓

Release of mediators triggers activation of

T cells & B cells

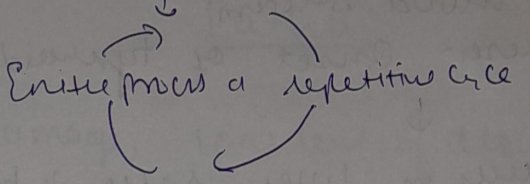
↓

Prolonged exposure to mediators ↓ I. cells infiltration can lead to tissue damage

↓

Differentiation & Proliferation
B cell
Macrophage
Leucocytes

↓



↓

Chronic Infl^n

06(a) ~~That~~ Typhoid is an infection caused by Salmonella typhi.

They are usually spread through contaminated food & water.

C.M - The symptoms likely to start slowly, occurs after 1-3 weeks after exposure to bacteria.

- fever, chills, headache, weakness, Body pain, Abd. pain, diarrhea or constipation.

~~Pat~~ - 1-3 weeks after infection, S. typhi spends in intestinal wall & then it enters blood stream.

Ingest contaminated food or water

↓ S. typhi

Invade small intestine & after 1-3 weeks enter blood streams → causes diarrhea

↓

then after it is carried by ~~the~~ WBC to liver, spleen & Bone marrow

Ingest (on) food or water

↓ S. typhi

Enters small intestine & spread there for 1 / 3 weeks

causing - Diarrhea

↓

Enters blood stream

causes - Onset of typhoid fever

↓

Carried by WBC cells in liver, spleen & bonemarrow

↓

Thereafter Bacteria invade Gall bladder & lymphatic tissue of bowel, there it multiplies in large no.

↓

Then pass to intestinal tract & it can be identified by stool test.

Etiology & causes of oedema

06(B)

→ Oedema is a medical cond^n characterised by accumulation of excess fluid within tissues of the body leading to swelling.

causes / Etiology - (1) CCF, when heart chambers fails to pump adequate blood, as a result blood back up in legs, lungs, ankles & feets leading to Oedema.

2) Liver damage - Cirrhosis can causes fluid build up in stomach area (ascites).

3) Kidney damage - Damage of nephrons & blood vessels of kidney can result in nephrotic syndrome which can lead to Oedema.

4) lymphatic system damage can cause lymphoedema. It also causes swelling of body tissues.

Our L.S. is a network of tissue, organs & vessels that work together to move fluid back to circulatory system

5) Protein-lack in our diet causes oedema such as low level of albumin

Patho

CCF, liver damage, low level of albumin in plasma

↓

Reduced plasma oncotic osmotic pressure

~~(oncotic P → protein pull water into & osmosis tries to equals pull (amt. of water in blood & interstitial fluid)~~

(oncotic P → is a pressure induced by plasma protein that pulls water molecules back to circulatory system & osmosis equals amt. of water in blood & interstitial fluid).

↓

Shift of water & electrolytes to interstitial space from intravascular space

↓

↓ in arterial blood volume

↳ ↑ RAAS system

↳ ↑ sympathetic nervous system

↓

Renal retention of Na^+ & water

↓

↑ed plasma vol.

↓

transduation of fluid

↓

Oedema

Q7(a) - P.D. is a chronic, progressive neurological disease that is associated with loss of dopaminergic neurons & causes - slowed movements & tremors.

C.M - 1) Tremor - rhythmic shaking. can occur at rest & usually begins from limbs.

2) Bradykinesia (slowed mvmts.) - overtime, P.D may slow your movement, making simple task difficult.

3) Muscle stiffness - It can be painful.

4) Impaired posture & balance.

5) Speech changes - you may speak softly, quickly, hesitate before talking.

6) Writing changes - small handwritings & hard to write.

7) Sleep disturbance, Ataxia

Etiology - (1) Genes - Genetic changes can cause P.D

2) Presence of Lewy bodies - Clumps of specific substance within brain is a marker of P.D & this is called Lewy bodies & it is known that Lewy bodies hold an imp clue of cause of P.D.

3) α-Synuclein found in Lewy bodies - It is a protein present in Lewy bodies.

& it is present in clump form & can't be break down.

4) When neurons damage the dopamine levels drop & causes P.D.

5) Medication like antipsychotic drugs also causes P.D.

Pathogenesis

Genetic factors — Age — Environmental factors

↓

Mutation & deposition of α-synuclein, & Lewy bodies are formed

↓

Mitochondrial dysfunction, generation of ROS

↓

Oxidative stress causes DNA damage & Alterations in dopamine level

↓

Induction of Apoptosis & onset of abnormal movements

↓

Symptoms of P.D

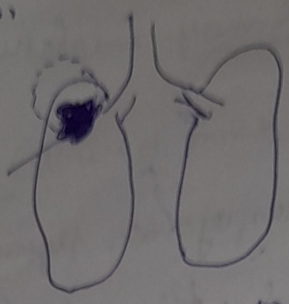
↓

Tremor,
Bradykinesia,
Rigidity,
Ataxia,
Speech & writing changes.

Q7(b) There are 3 main ways by which cancer can spread:-

1) ~~Direct~~ 2) lymphatic 3) Hematogenous 4) Spread across body cavities / natural lining

1) Direct spread refers to growing of tumor locally into adjacent structures.



Eg - lung cancer

↓

which spreads by invasion into chest wall, airways, Brachial plexus

-> Breast cancer may metastasize to bones, liver, lu

1) Lymphatic spread - all carcinomas spread through L.S.

Spread via 2 forms:-

i) Lymphatic permeation - cancer cells invade walls of lymphatics & form a continues growth in lymphatic channels.

ii) Lymphatic emboli - Malignant cell detach & forms

↓

Tumor emboli

↓

carried to lymph node

↓

enter subcapsular sinus & starts growing

Eg- Breast cancer - The tumor can spread from breast to axillary lymph nodes.

2) Haematogenous Spread - blood- borne metastasis

- Spread thru blood stream.
- Common route of sarcoma.

Some carcinomas ~~like~~ also spread via this route. Esp. - kidney, breast, kidney, prostate, ovary.

- The sites at which this spread commonly occurs are liver, lung, brain, kidney, bones (By seed-soil theory)

1 → Systemic vein → Venae cavae ↓

drain blood to limbs, head, & neck.

• So, cancer at these sites often metastasize to lungs.

2 → Portal vein → spleen, pancreas ↓ liver

Cancer at these sites often metastasise to liver.

3 → Arterial spread - It less likely as they are thick walled. However, they can occur when tumor cell pass through pulmonary arteries as they have thin walls.

Eg - Cancer of lungs may metastasize by pulmonary arterial route to bones, kidneys etc.

3) Spread Along Body cavities & Natural passage -

1 → transcoelomic fluid
2 → Spread along epithelium
3 → Spread via cerebrospinal fluid
4 → Implantation

1) Transcoelemic - cancer invades to coelemic cavity
The fragment of tumor breaks & is carried to coelemic fluid where it spreads.
Eg - carcinoma of stomach., ovary.

2) Via epithelium - It is unusual for a malignant tumor to spread epithelium lined surface as they are quite resistant to tumor but exceptionally they spread through -
Eg - Endometrium of fallopian tube to ovaries
- Bronchus to alveoli,
- ~~Urinary tract~~ to ureters to urinary tract.

3) Via CSF - The fragments of tumor spread via CSF into CNS.

4) Implantation - Rarely occur.

factor influencing cancer -

1) Genetic factors - Mutations can be the risk of cancer.
2) Age - Risk of cancer ↑ with ↑ in age due to cell damage which accumulates
3) Env. Factors - such as Exposure to -
- carcinogens - eg - tobacco smoke
- Radiations
- Pollutants
4) Use of Tobacco & Alcohol ↑ the risk of cancer
5) Obesity.

Q4 Leprosy also known as Hansen's disease. It is an infectious disease caused by bacteria Mycobacterium leprae. It primarily affects skin, nerves, etc.

It is characterised by formn of skin lesions, loss of sensn, & nerve damage.

Causes - It is caused by Mycobacterium leprae.
- Close contact with the untreated individual may cause leprosy.

Mode of Spread - By coming in close & prolonged contact with the untreated individual.

→ It is a contagious disease.
- The exact method of transmission is still under investigation.
But it is to be believed that it also occur through respiratory droplets expelled by infected person.
- However, leprosy is not transmitted thru casual contact with infected person.

Patho

Bacteria discharged from nose
↓
Inhaled by other person
↓
Taken up by alveolar macrophages
↓
Transmitted through blood
↓
Spreads to nerves & skin
↓
Multiplies in Schwann's cells

Strong cell-Mediated Immunity response

↓

- No skin lesions
- No nerve lesions.

Weak cell response

- Multibacilli leprosy, Additionaly to nerves skin

↓

Dysfunction & destation

↓

leprosy.

Q4(B) Is a sore that forms in the lining of the stomach or duodenum.

Peptic ulcers include -

1) Gastric ulcer - that occurs inside the stomach
2) Duodenal ulcer - that occurs ~~inside~~ on the upper portion of S.I. (duodenum).

s/s - Burning stomach pain,
feeling of bloating,
Heart burn,
Nausea,
Intolerance of fatty food.

Etiology - Helicobacter pylori bacteria which live in mucous layer of stomach & S.I.. It can cause ulcer.

→ use of certain pain relieving meds. such as NSAID. Eg- Naproxen.

NSAID inhibits COX-1 which leads to ↓n in secretion of prostaglandins (eg. inhibit gastric secn)

- Medication such as steroids, anticoagulant, can cause ulcers
- smoking & alcohol can also ↑es the risk of ulcers.

Diagnosis - Esophagogastroduodenoscopy.

- a form of endoscopy where location & severity of ulcer can be seen.
- If P.U. perforate, air leaks into GIT & on x-ray can be seen.

Pathophysiology -

Alcohol, smoking age

NSAID

H. pylori

↓

↑es acid secn

&

↓

chronic infn of H. Pylori in stomach

H. Pylori colonization in duodenum

↓ Bicarbonate secn,

↓ Prostaglandin ↓

Inflammation of duodenum

Ulcer Mucosal damage & ulcer prodn.

Treat - Abs - Amoxicillin,
PPi - Pantoprazole
3) H_2 blockers - Ranitidine
4) Antacids - Rantac
Amoxicillin + metronidazole
Abs + Pantop + PPi

Nutritional disease

Also klas - nutritional deficiencies & or malnutrition.
- occurs when body does not receive adequate amt. of essential nutrients

↓

like - fats, carbohydrates, protein, vit. & minerals.

1) Protein-Energy Malnutrition - occurs when there is inadequate consumption of protein & energy which can cause loss of body ~~mass~~ muscle resulting in PEM.
It is seen in developing country & can manifest as - marasmus (severe calorie & protein deficiency) & macronutrient deficiency

& kwashiorkor - results from lack of protein in diet
It leads to poor growth of child.

2) Obesity - dietry imbalance, overnutrition intake & physical inactivity may lead of obesity.
(More from 2023 Ques Paper).

3) Iron deficiency Anemia - Fe is essential for prodn of RBC & its deficieny leads of anemia.
& skin turns pale.

4) Starvation - It is a state of severe deprivation of nutrients.
It can cause due to - deliberate fasting - religious fasting

- Poor financial condⁿ.
- ↳ Secondary undernourished due to chronic disease such as- cancer.

↳ during starvation period many metabolic changes occurs.

→ During fasting, insulin independent tissues such as Brain takes up glucose. but insulin dependent such as muscle cell do not take up glucose.

. there is also breakdown of proteins to A.A. ↳ triglycerides to glycerol + fatty acid.

UTI

It is an infⁿ of the urinary tract

- any infⁿ in U.T → kidneys, bladder ↳ urethra.

Womens are at greater risk of developing a UTI than a men.

Etiology- the common cause of UTI is ↑ in sexual activity.

- the 1° bacteria that cause UTI is Escherichia coli which normally resides in digestive tract but can spread to U.T.

→ use of catheter, which is tube inserted into bladder. It can directly introduce bacteria to U.T.

→ when urine stays in bladder for a longer period of time, it provides an environment for bacteria to multiply.

S/S - Burning micturation,
Strong smell of urine,
pelvic pain, ~~dcps~~ often urination but only small amt of urine passes

Risk factor - (1) Gender women are at higher risk of developing UTI than men. bcz women have a shorter urethra which allows bacteria to reach ~~ut~~ bladder more easily. Also harmonal chgs during pregnancy can ↑ risk of UTI

2) Sexual activity - Sexual intercourse can introduce bacteria into urethra & ↑ risk of UTI.

3) use of urinary catheter
4) urine retention for a long period of time
5) Improper hygiene
6) Diabetes can also ↑ risk of UTI

Diagnosis - By analysing urine sample like there is a protein presence in your urine
- H/O taken by dr. ~~of~~ patient & problem he/she's facing.

Mt - Mainly by Abs - cephalosporins.

Patho –

Sexual intercourse — E. coli — Exposure of improper urine catheter. hygiene

↓

1) Bacteria enters the urinary tract

↓

2) Ascension of Bacteria – Bacteria ascend the urethra & reach the bladder. In women Bacteria reach the bladder more easily.

↓

3) Adherence & colonization – Bacteria attach to bladder using fimbriae or pili.

↓

4) Multiplication – Bacteria multiply rapidly.. & produced local infection.

↓

5) Inflammatory Response – Presence of Bacteria triggers I.R. in UT. The Immune response occurs which can result in pain, burning sensation & micturition.

↓

6) Spread of Inf – Bacteria can infect ureters (ureteritis) & kidneys (pyelonephritis)

↓

UTI, also the bacteria may enter the blood stream.

Acute Renal failure

Also k/s – Acute kidney injury. occurs when any inf^n & injury damages the kidneys.

Occurs when kidneys are unable to filter the waste products & fluids from the blood.

– It is a temporary cond^n. with proper tret. It can be reversed with no permanent damage to kidneys.

Etiology – BP med^n, Blood or fluid loss, liver failure. severe dehydration, shock,

- Renal artery stenosis (narrowing of the arteries that supply blood to kidneys),

kidney inf^n, kidney stones

S/S – Anemia - The RBC are responsible for producing erythropoietin (EPO), a hormone that stimulates RBC prod^n.

Kidney disease causes shrinkage of kidney, RBC prod^n is reduced leading to anemia

- fluid retention which causes swelling in your ankles, feet
- ↓ urine output, Nausea, stomach upset
- lower back pain – patient suffering from kidney probs may have pain where kidneys are located

patho - ARf comprises of 3 main mech^n.

Pre-renal causes → Glomerular disease

Intrinsic renal causes → Disease of glomerulus or tubule → Release of Renal vasoconstrictor

Post renal cause → glomerulo - nephritis

Tmt - Tmt. to balance the amt. of fluid in your body.

Diuretics mech^n to expel extra fluids.

- mech^n to control K+ level
- Dialysis to remove toxins - one may need temporary hemodialysis - to remove toxins & excess fluids from your body.

Chronic Renal failure

- Also KIS - Chronic kidney failure is a slowly progressive loss of renal failure. It leads to severe illness & requires renal replacement therapy (dialysis) & is ckd - End Stage Renal disease.

Etiology - Most common cause is Diabetic nephropathy, HTN, Glomerulonephritis, kidney disease, kidney inf^n also ckd - Pyelonephritis.

Diagnosis - kidney in CRF are usually small.

Patho

Diabetic nephropathy — kidney disease — glomerulonephritis

↓

Renal Infl^{n} & fibrosis

↓

Vascular changes in kidneys

↓

Imbalance btw^{n} ROS & antioxidant

↓

Presence of protein in urine

↓

activ^{n} of RAAS

↓

CRf.

ARf

Onset - over days - weeks

- Is Reversible
- Doesn't cause Renal failure
- Temporarily Dialysis is done
- Kidney size is normal
- ~~Is~~ Serum Creatinine level is delayed.

Is done for short period for temporarily.

CRf

over weeks to months

Irreversible

cause Renal failure

Dialysis is repeatedly.

Reduced kidney size

↑ CRP.

- chronic long period.

IBD

Term used to describe disorder that involve - chronic inflam of Digestive tract.

Types -

1) Ulcerative colitis - Inf^n & ulcers in the lining of colon & Rectum.

Etiology - Most common symptom is Abd. pain & Bloody diarrhea. Patient may also experience - Anemia, wt. loss, loss of appetite, loss of body fluids, fatigue

Etiology - The exact cause is unkl.

- one possible cause is Immune system malfunctn. when I.S. attacks the cells of digestive tract.

→ ~~Here~~ Heredity.

Depression

Is a mental disorder characterized by persistant sadness & lack of interest in activities

- Sadness of mood
- ↑ed appetite & wt. gain
- Disturbed or ↑ sleep
- lost of pleasure in activities
- ↓ in ability of think/ concentrate

Mania

- Condn in which you've a period of abnormally elevated, extreme changes in your mood, energy level.
- Energetic, Excitment mood
- loss of appetite & ↓ wt. gain
- little desire to sleep.
- quick speaking on wide ranging topics

↑ talking, thoughts