26/9

GANGRENE & CALCIFICATION

Infarction
→ Area of ischemic necrosis - occlusion of arterial supply or venous drainage
→ Arterial thrombosis or embolism
→ Local vasospasm, hemorrhage into an atheromatous plaque
→ External vessel compression - tumor.
→ Venous thrombosis - bypass channels rapidly open - outflow - improves arterial inflow.

classification
① colour → red - hemorrhagic
→ white - anemic.
② Infection → bland.
→ septic.

What is Gangrene?
→ Necrosis of tissue associated with superadded putrefaction, most often following coagulative necrosis due to ischemia.

It is the death of tissue caused by necrosis with infection.

causes
Arterial occlusion
Venous obstruction - DVT
Nervous disease
→ diabetic gangrene
→ physical → heat
cold
corrosive
electricity
radiation.

Types:
→ Dry & Wet

Dry
Ischemia - hypoxic injury
↓
Coagulative necrosis
↓
limits the blood supply
↓
favours bacteria.

WET. Infarction
↓
pooling of blood
↓
↑ bacteria.

—

Macroscopic death of tissue with putrefaction

Dry gangrene
→ Blood supply: arterial - gradually disrupted
venous flow - unimpeded
→ Arterial occlusion - chronic or acute
→ colour change:
greenish black. dry - mummified
→ line of separation present

→ Stump is conical.
→ Bord of ~~hyperemia~~ hyperemia
→ Senile, diabetic

Wet Gangrene
Arterial & venous block
cold, pulseless, oedematous, debilitated, swollen
Horrible odour
No line of demarcation
constitutional symptoms present
Spreads fastly
Seen in acute inflammation venous thrombosis, gas gangrene
→ mouth, bowel, lungs, cervix and vulva
eg: Diabetic foot.
Bed sores
Gangrene of bowel due to strangulated hernia

• Fournier's gangrene
↳ vascular infective gangrene of scrotum.

Gas gangrene
→ considerable mass of body tissue dies
→ cause - clostridium perfringens
→ gram +ve anaerobic gas forming bacilli

→ It is also called as
signs
→ swollen organ
→ Edematous
→ Crepitus
→ Blisters with foul smelling discharge
→ Microscopically, bacterial colonies seen
ms fibres show coagulation necrosis with liquefaction
Gram +ve bacilli.

• Special form of wet gangrene caused by which gains entry into tissues through contaminated wounds.
types: fulminant, massive, group.

Contrasting features of dry and wet gangrene - Table.
coagulative necrosis - dry
liquefaction → wet

Key point
etiology, site, mechanism, gross, microscopy.

diagnostic
→ Xray
→ Blood.
→ CT

Managment

→ antibiotic - penicillin
Ampicillin
Piperacillin

Supportive care
→ IV fluid
→ Anticoagulant
High protein diet
Oxygen therapy

Surgical
→ Amputation
→

Pathological calcification

def: Abnormal dep of Ca^{2+} in tissue other than osteoid & enamel.

2 types: → Dystrophic
→ Metastatic

→ Etiology & pathogenesis diff & morphology similar

Gross: chalky white, granular mass

Micro: H&E - deeply basophilic, irregular granular deposits

Site: extra or intra cellular

Special stain: von kossa (black colour)

VIVA
Ca^{2+} deposits basophilic, special stain - black

Dystrophic
in dead & degenerated tissue
→ Ca^{2+} metabolism normal
→ Serum Ca^{2+} normal
→ cause organ dysfunction in valves affected
eg: aortic stenosis

Pathogenesis → formation of normal hydroxyapatite

eg:
→ caseous necrosis in TB
c/c ulcer
→ fat necrosis
→ infarcts
→ Thrombi in veins
→

Old scars, atheroma, Monckeberg sclerosis (uterus)
→ wall of longstanding cysts
→ calcinosis cutis (Ca^{2+} in skin & S/C tissue, cutaneous tumor)
→ in senile degenerative changes (costal & tracheal cartilage)

Metastatic
site:
kidneys → basement membrane of tubules ep

stomach
Blood vessel → internal elastic lamina
Cornea.
Synovium
Etiology
Excessive mobilisation for bone ↑.
Excessive immobilisation
Pathogenesis.
→ cardiac
→ Initiation & propagation
→ local alteration of pH.

Excessive mobilisation
more common
→ Hyperparathyroidism.
① primary (PTH adenoma)
② secondary parathyroid (C/c renal fail)
→ Destructive bone lesion (m. myeloma, metastasis, Paget's disease)
→ Prolonged immobilisation of patients.

Excessive absorption
→ Hypervitaminosis D.
→ Milk alkali syndrome.
→ Hypercalcemia of infancy

(Prepare Table.)

def, ca++ mid, ca++ level (serum) sensitivity, causes, pathogenesis, common sites

Prepare note
→ Types of calcification
→ Difference b/w
→ Pathogenesis
→ Morphology
→ Microscopic features.
→ Special stains, what does the each stain)
→ Infection – types.
→ Cancer – definition
→ Causes
→ Pathogenesis
→ Types
→ Differentials

- Types of Calcification
  1. Dystrophic.
  2. Metastatic.

Abnormal deposition of calcium salts in tissues other than osseous & enamel.

- Differences between

	Dystrophic	Metastatic.
Definition	Deposition of Ca^{2+} in dying or dead tissues.	Dep. of Ca^{2+} in apparently normal tissues.
Ca^{2+} Metabolism.	normal.	Excessive Deranged.
Serum Ca^{2+} level.	normal.	Increased.
Reversibility.	Generally irreversible.	Reversible upon correction of metabolic disorder.
Causes.	Necrosis (caseous, liquefactive, fat), infarcts, rheumatic, hematoma, dead parasites, old scars, atheromas, Monckeberg's sclerosis, certain tumours, cysts, calculus, tuberculosis.	Hyperparathyroidism (due to adenoma, hyperplasia, CRF), bony destructive lesions (eg: myeloma, metastatic carcinoma), prolonged immobilization, hypervitaminosis D, milk-alkali syndrome, Hypercalcemia of pregnancy.
pathogenesis	↑ binding of phosphate with necrotic and degenerative tissue, which in turn binds to calcium, forming calcium phosphate precipitate.	↑ precipitate of calcium phosphate due to hypercalcemia at certain sites such as lungs, stomach, blood vessels & cornea.
common sites		kidneys, lungs, stomach, blood vessel, cornea, synovium.

- Pathogenesis.

Dystrophic calcification:

Since serum Ca^{2+} levels are within normal limits, denatured proteins in necrotic or degenerated tissue bind PO_4 - which react with Ca^{2+} to form precipitate of $Ca_3(PO_4)_2$.

- formation of normal hydroxy apatite in bone in 2 phases

i) Initiation - precipitate of $Ca_3(PO_4)_2$ accumulate intracellularly in mitochondria, or extracellularly in membrane-bound vesicles.

ii) Propagation - mineral deposited in initiation phase are propagated to form mineral crystals

Morphology

Gross : chalky white, granular mass.

Micro : deeply basophilic, irregular granular clumps.

Stains

Von kossa — black.
alizarin reds → red.

Types of Infarction

Based on colour,
→ hemorrhagic and anemic
Based on infection,
→ Aseptic and septic.

Gangrene.

Massive necrosis of tissue associated with superadded putrefaction) most often following coagulative necrosis due to ischemia.

Causes : arterial occlusion (eg. atherosclerosis), venous obstruction (DVT), nervous disease, diabetic gangrene, due to physical factors such as heat (burns), cold (frostbite), corrosive, electricity, radiation.

Types → Dry Gangrene.
→ Wet Gangrene.
→ Gas gangrene.

Differences:

	Dry	Wet
Site	Limbs.	Bowels
Eg.	Gangrene due to atherosclerotic narrowing of blood vessels of lower limb.	Volvulus intussusception.
Cause of ischemia.	Arterial obstruction.	Venous obstruction.
Rate of obstruction	slow	Abrupt
Appearance	Shrunken, dry and black.	Swollen, soft and moist.
Line of demarcation	clear cut	Not clear cut.
Spread	slow	rapid.
Prognosis	Fair.	Poor due to severe septicemia.
Putrefaction	limited due to very little blood supply	Marked due to stuffing of organ with blood.
Bacteria	fail to survive	numerous present.