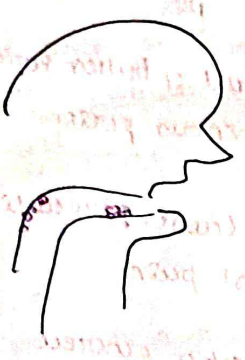
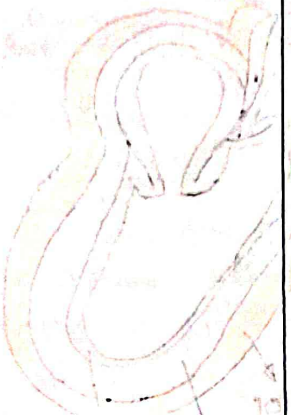
Rheumatic Fever & Rheumatic heart disease





If a person is affected by group A β hemolytic streptococcus - causing streptococcal tonsillitis / pharyngitis ...

↓

macrophages present (APC) & Ab are formed.

↓

due to molecular mimicry in (2%).

the Ab cross react with our own antigens.

↓

in 98% .... Ab are highly specific, so they only act on streptococcus.

↓

but in 2-3% individuals having genetic predisposition.

↓

Ab cross react with Ag (glycoproteins of our own) mainly.

heart, CNS, joints, skin, subcutaneous tissue.

- Ab take 2-6 weeks to develop.
- so post group A β hemolytic streptococcal infection - 2-6 weeks later.
- there will be cross reaction & inflammation.

Acute Rheumatic fever.

→ multisystem affected - immune mediated (Ab), nonsuppurative (no bacteria) Inflammatory Condition.

- CNS - Basal ganglia affected.

↓

Sydenham's chorea. (F>M)

- rare, but diagnostic

Chorea - involuntary purposeless jerky movements.

- Joints → arthritis → painful, tender, swollen.
  - arthralgia → only pain. (tenderX, swollenX)
- migratory poly arthritis.

↓

dif joints are affected one after other.

↓

magic drug - Aspirin.

- these are completely reversible after. fever is over.

But not in case of ♡

- after fever, joints & chorea will be fine.
- no - long term complications in joints,

↓

Skin.

- rapidly developing lesions.. that get healed in centre first
  ↓
  erythema marginatum. (painless).
- Common - trunk
  less - hands & feet
- No - Long term effect!
- heals completely.

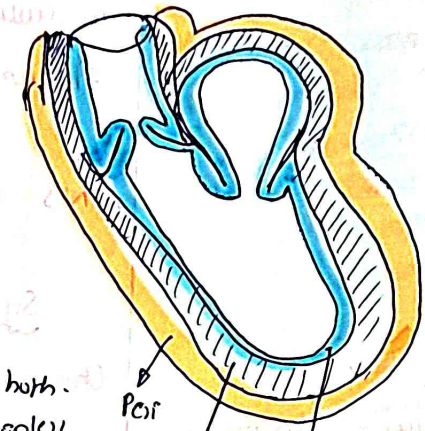
Subcutaneous tissue.

- subcutaneous nodules.
  → nodules of connective tissue.
- extensor surfaces of limb/ bony prominence.] site.

No long term complications

Cardiac problems - Heart.

Bad :(



both. pericar.

- when all tissues are involved
  → pancarditis.
  R. endocarditis + myocarditis + pericarditis.
  { others - 1/2 occur }

Rheumatic pericarditis.

- due to attacks...
- ↑ endothelial gaps.
- leakage of large molecular proteins like fibrinogen from blood to pericardium
- fibrinogen polymerise.
  ↓
  fibrin strands (thread) eosinophilic
  ↓
  fibrinous pericarditis.

} it is a feature of any immune mediated pericarditis

→ fibrin deposits in pericardial sac.
↓
Bread & butter pericarditis/ fibrinous pericarditis.

- It causes pericardial chest pain.
- which is central chest pain, sharp, well localized AND! relieved on leaning forward.
  ↓
  should know how to differentiate from ischemic pain.
  ↓
  dull, poorly localized, radiating, not relieved on leaning forward.
- pericardial sub. on auscultation
  ↓
  scratchy sound due to pericardial layer rubbing together.
- No Long term effects as fibrin will be gone afterwards, and heart completely.

myocardium.

- Classical Immune mediated lesions that are found in myocardium. are called

↓

Aschoff bodies

↓

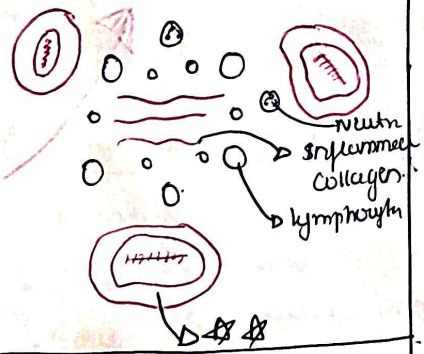
Very small, pinhead lesion due to Immune mediated granuloma.

↓

especially around perivascular connective tissue.

{around blood vessel
Connective tissue of pericardium}

Aschoff bodies - granuloma.



activated plump macrophages with prominent nucleoli & ribbon shaped caterpillar nuclei are called

Anitschkow cells / Caterpillar cells.

- pathognomic.

Aschoff body - granuloma.

- Anitschkoff cell.
- ↓ fuse → Aschoff giant cell.

myocarditis.

- most serious complication during acute phase.
- myocardium become loose & flabby

↓

poor contraction

↓

right & left ventricular failure. - cardiac failure.

- No long term complications
- But very serious / most serious complication during acute phase.

- If a child dies during acute rheumatic fever. the most common cause is Rheumatic myocarditis

Rheumatic Endocarditis.

- 2 types of lesions

↓ during fever

↓ in between fever.

- during ARF →.

Immune system mainly attacks → valvular Connective tissue.

↓

Rheumatic valvulitis.

- the valves are already inflamed ...
- one of the patient has tachycardia due to fever ... even asymptomatic.
- due to inflammation & constant hitting... erosions will form @ Line of closure of valves.

↓

fibrin, platelet deposit

↓

Rheumatic vegetations.

↓

multiple small, vegetations along line of closure - sterile, firmly fixed.

↓

don't embolise.

↓

unlike IE.

after fever ...

they start to heal .. & platelet produce - PDGF.

- lots of fibroblast are there & they lay down Collagen.

↓

Vegetations become scars

↓

Edges fuses.

↓

Cusps develop adhesions.

- After fever is over, vegetation heal by fibrosis & adhesion

↓

distortion of valves.

↓

Long term Complications

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acute rheumatic heart disease { what's sus during aR fever }

ie, pericarditis, myocarditis.

Chronic RHD → valve problm.

what's sus btw fever episodes.

ie, endocarditis ✓

never peri & myo as they heal completely after fever.

☆

Monckerberg

not dangerous at all.

media

Calcification

medium sized arteries — especially radial, ulnar etc.

{ ie, muscular arteries

↓

there is no threat actually, due to calcification we can easily palpate the radial vessel and all.

- Like even after occluding Brachial artery, we can feel the radial artery. due to calcification of vessel wall.

- It is not a dangerous condition as it never encroaches on intima & there is no lumen occlusion.

Classical example
Elastic arteries →
aortic
Carotid
Iliac artery.

atherosclerosis

very very dangerous.

Intima.

Fibro-fatty plaque.

- large sized arteries. aortic, popliteal etc.

↓

{ Elastic arteries }