VASCULITIS:

It is a general term for vessel wall inflammation with protean manifestations depending on the vascular bed affected

Two Pathogenic mechanism of vasculitis are
- Immune Mediated
- direct invasion of Pathogens (from immune complex)

NON INFECTIOUS VASCULITIS:

main immunologic infiltrates are
- immune complex deposits (SLE)
- Anti neutrophil cytoplasmic Ab (ANCA)
- Auto Reactive T cell
- Antibody thelial cell Antibodies.

ANCAS: → Relate Radial injure tissue

Anti neutrophil cytoplasmic Antibodies are heterogenous groups of Auto antibodies direct against is the cytoplasmic granules of neutrophils & lysosome of monocytes.

Two types

→ Anti - Proteinase 3 (PR3-ANCA) (CANCA)
→ Associated with granulomatosis with polyAngiitis.

→ Anti - myeloperoxidase (MPO-ANCA) - PANCA
→ Associated with polyangiitis of eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

RANKA
DATE / /
PAGE

CLASSIFICATION:

LARGE VESSEL VASCULITIS: (Granulomatous disease)
Eg: giant cell Arteritis, Takayasu Arteritis

MEDIUM VESSEL VASCULITIS:
→ Immune complex Mediated
- polyarteritis nodosa
→ Anti endothelial cell Antibodies
- kawasaki disease.

SMALL VESSEL VASCULITIS:
→ small/no amount of immune complex (ANCA)
- vasculitis without Asthma or granulomas (Microscopic polyangiitis)
- Granulomas, no Asthma (granulomatosis with polyangiitis)
- Eosinophilia, Asthma & granuloma (Cheng-Strauss syndrome)

→ Immune complex Mediated
- SLE
- IgA (Henoch-scholer purpura
- cryoglobulin (cryoglobe vasculit)
- Other (Goodpasture diseases.

GIANT CELL ARTERITS:

It is chronic, classically granulomatous inflammation of large to small sized Arteries that principally affect aorta & its head (old elderly people)
→ Biopsy of temporal Artery

NON-HODGKIN'S LYMPHOMA

Pathogenesis: Tcell Mediated immune Response
unkown Antigen ; TH1 of TH17 involved

CLINICAL FEATURES
→ after the Age of 50
→ Fever, Anemia, fatigue, headache
→ Evolved Artery is tender, thickened or nodular
DIAGNOSIS - biopsy of microscopic examination of the Artery.
MORPHOLOGY
→ Nodular thickening of the intima
→ Reduce the size of the lumen and produce ischemic in distal Region
MICRO:- → Lymphocyte, Macrophage Along with giant cell (TB, LGS)
- Intimal thickening, Medial thinning, scarring of Adventitial fibrosis.

POLYARTERTIS NODOSA: (PAN)
It is systemic necrotizing vasculitis of small & Medium sized muscular Arteries. affect Renal & visceral Arteries

Usual involved: affect wall of the kidney (glomeruli are spared), heart, liver, GI tract
- doesn't involve pulmonary Arteries.
Associated with Hep B infection.
↳ HBsAg - Abs Ab (complexes)

MORPHOLOGY:
Segmental transmural necrotizing inflammation of small to Medium sized Arteries. Along with aneurysmal Aneurysm.
→ Transmural mixed inflammation of the wall, wall consist of polymorphonuclear neutrophil & mononuclear cells.

→ Fibrinoid necrosis of luminal thrombus
→ Aneurysm
→ old lesions: Fibrous thicking of vessel wall

CF → Young Adults
→ cause ischemia of infarction
→ Renal involvement manifests hypertension

Granulomatosis with Polyangiitis:

It is a ANCA positive necrotizing vasculitis
↓
Acute in URT or LRT

→ Affect small to Medium Vessels.
→ Focal necrotizing often segmental glomerulonephritis.

Pathogenesis: T cell Mediated hypersensitivity to exogenous Ag.
PCR3-ANCA play Role in Pathogenesis.

Kawasaki Disease: Acute febrile, usually self-limited illness of infancy & childhood Associated with large to Medium sized vessel.
→ DT Hypersensitivity.

CHURG STRAUSS SYNDROME: (Allergic granulomatous Angiitis)
Associated with Asthma, Allergic Rhinitis, lung infiltrates
Peripheral eosinophilia, Extravascular necrotizing

⇒ Thromboangiitis (Buerger Disease)
→ segmental, thrombosing Acute & chronic Inflammation of Medium & small
especially tibial & Radial.
- microAbscess composed neutrophils

⇒ Takayasu Arteritis:

NON - HODKIN'S LYMPHOMA