Marks 2

NEPHROTIC SYNDROME:

1° / 2ndary (SLE, DM, Amyloid)

- r/w massive proteinuria.
- oncotic pressure ↓ → edema
→ excess loss of Albumin - HypoAlbuminaea:
→ nephrotic syndrome - fatty cast.
→ Hyperlipidemia.

PATHOGENESIS:

Liver compensate

APH. Activated

- Thrombotic episodes.

due excess loss of protein → ↑ chance of infection.

Diagram imp Refer / after etiopathology

Investigation

Urine sample show proteinuria.

- urinary cast

- Autoimmune markers

VQ S.N

1° Minimal change disease (podocytopathy) → seen in children → null deposit. (Lipoid nephrosis)

(1) seen only in immuno electron microscopy.

→ Immune dysfunction ↪ Fusion of podocytes

↓

elaboration of factors → Damage to visceral epithelium

↓

Proteinuria.

→ Associated with Hodgkin's lymphoma

→ LM - -ve, Immunofluo - -ve

(2) Membranous nephropathy → most common seen in elderly Age group

→ Associated with Malignancy

↪ colon
↪ lung
↪ Melanoma
} Ca

→ deposit containing Ig in subepithelial Regions

→ Associated with → penicillin, NSAIDS, gold,

infection: hep B, syphilis (leprosy, schistosomiasis)

Pathway

Causes:

Phospholipase A2 Receptor is mutated

Complement activation (C5b-C9)

Membranous GN

1) Light microscope: Normal: Early stage

GBM

→ granular, spike and dome Appearance

(5) Focal segmental glomerulosclerosis (FGSS) → seen in HIV → seen in Adult

Epithelial cell damage is the hall mark.

cause – HIV, Heroin Abuse, Hypertensive, SCD, Reflux nephropathy.

Protein → nephrin

podocin

α-Actin

TRPC6.

MORPHOLOGY:

Light microscope:

↑ in mesangial Area.

Hyalinosis

Immunofluorescence:

EM → Degradation of GM

IgM & C3 deposit seen in sclerotic Area of mesangium.

→ Fibrin, leukocytes, epithelial cell (parietal cell)

→ see content of segment shape

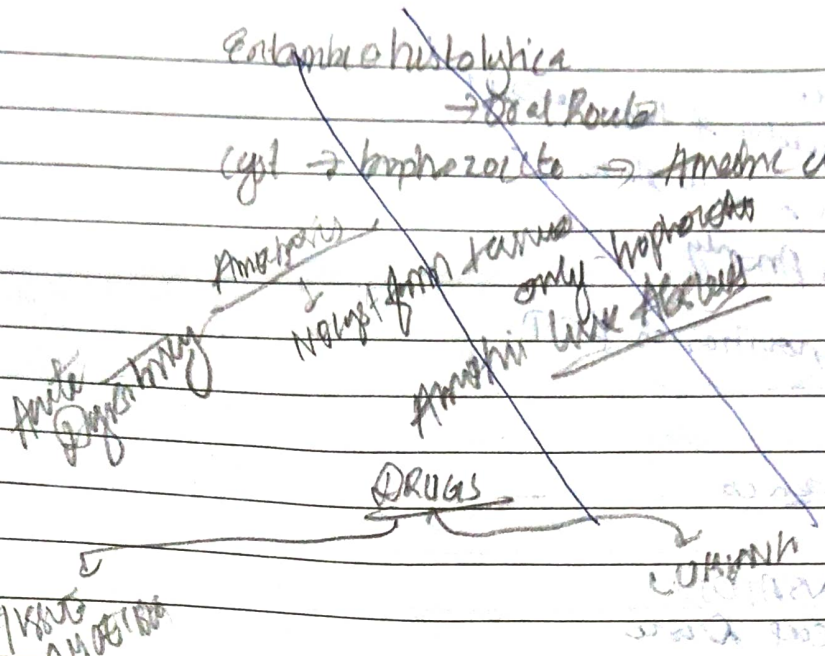
variant of FSGS → Collapsing glomerulopathy.

in kidney.

→ proliferation & Hypertrophy of epithelial cells.

(6) Membrano proliferative Glomerulonephritis (MPGN)

Type I: involve subendothelial cells → both classical & Alternate pathway.



→ Light microscopy: (MPGN Type I)

Thickened GBM - Tram track Appearance

- Coexist ++++

Electron microscopy: subendothelial electron dense deposit.

Double contour of.

Type II - MPGN:

LM → Mesangial proliferation

Inflammation

Coexists ++++

deposit of C3++ in GBM

↳ n/w with hypocellular.

Ribbon-like appearance.

SN

IgA nephropathy (Berger disease)

- Mild hematuria, Mild proteinuria, IgA deposit in mesangium.

↳ HSP (skin disease)

SN → DIABETIC NEPHROPATHY