NEPHRITIC SYNDROME:

→ Hematuria, dysmorphic Red cells, Red cell casts in urine, Azotemia And oliguria. mild to moderate hypertension

MAIN FEATURES:

- Hematuria
- Proteinuria
- Hypertension
- Azotemia - Biochemical Abnormality with elevated blood urea nitrogen And creatinine level due to ↓ GFR
- ↓ GFR of oliguria.

ACUTE PROLIFERATIVE GLOMERULONEPHRITIS:

⇒ diffuse proliferation of glomerular cell Accompanied by infiltration of glomerulus by leukocytes.

→ usually Immune complex diseases.

- Exogenous (post infectious glomerulonephritis)
- Endogenous. (SLE)

POSTSTREPTOCOCCAL GLOMERULONEPHRITIS:

Seen in b/w 6 & 10 years.

PATHOGENESIS:

Infection of pharynx by nephritogenic strain of β-hemolytic streptococci

Planting of streptococcal Antigen from the circulation in the subendothelial Region of GBM

↓

Specific Ab React with streptococcal Antigen to form immune complex

↓

Immune complex initiate inflammatory Response by Activating mediator of inflammation

↓

These complex detach migrate across the GBM

↓

Glomerular damage with glomerulonephritis.

Nephritis syndrome.

Evidence: to support immunological basis

→ Latent period - usually develop 1-4 weeks after 1° infection

→ Ab against Antigens

→ Hypocomplementemia

→ Immune complex deposits

→ Streptococcal Antigens in glomerulus.

↳ SpeB (main Antigen)

↳ NAPIR (Nephritis Associated streptococcal plasmin Receptor)

↳ GAPDH (streptococcal glyceraldehyde-3 PO4 dehydrogenase.

MORPHOLOGY:

GROSS - enlarged, pale capsular surface

MICROSCOPY:

→ Light Microscopy:

Glomeruli: enlarged & hypercellular.

↓ due to

- infiltration by leukocytes → neutrophil and monocyte
- Proliferation & swelling of endothelial & mesangial cells
- Crescent formation in severe cases

↓

Proliferation of parietal cell lining the Bowman capsule

Tubules: Red cell cast in the lumen

↳ or dysmorphic Red cell

Immunofluorescence Microscopy: ↓ cells with granular or fragmentation

IgG, C3, IgM all seen in mesangium along the GBM

↓

~~Appear as lumps and pin lumps~~

pleomorphic granular fluorescence

Electron Microscopy

Subepithelial deposit - distinct, Amorphous, electron dense

deposits on the epithelial side of GBM

→ Termed as humps or lumps

subendothelial deposits - commonly during early phase of the disease.

C.F

⇒ Oligouria, hematuria,

⇒ Pairbital edema, mild to moderate hypertension. → Post

→ color color wire

L.B

⇒ Raised Antistreptococcal Antibody titer. (ASO titer)

⇒ Hypocomplementemia.

⇒ Raised blood urea & serum creatinine.

PSGN

GOOD PASTURE SYNDROME:

Anti GBM Antibodies cross React with pulmonary Alveolar basement membrane causing pulmonary hemorrhage associated with a Renal failure.

PATHOGENESIS:

Genetic predisposition: HLA - DRB1

HLA - DRB1 - in these patient points towards genetic predisposition to Autoimmunity.

⇒ Anti GBM Antibodies are formed against Goodpasture Antigen.

↓

These Antigen result from conformational change in α3 chain of type IV collagen.

IV collagen → α3 chain

→ Anti GBM Antibodies cross React with target Antigen.
⇒ React with Alveolar capillary basement membrane
⇒ pulmonary hemorrhages & hemoptysis.

⇒ Ruptures in the GBM Allow leukocytes, protein And inflammatory mediators to leak into the urinary space initiate crescent formation.

MORPHOLOGY:

GROSS: Enlarged & pale kidney show petechial hemorrhages on the cortical surface (flea-bitten kidney)

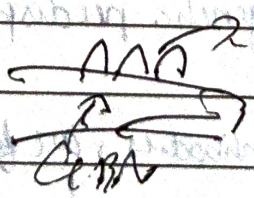
MICROSCOPY:

Glomeruli: Characteristic feature is crescents in most of the glomeli. Here the term CRESCENTIC GLOMERULONEPHRITIS.

CRESCENTS: formed by proliferation of the parietal epithelial cell lining the Bowman capsule And infiltration of monocytes And macrophages into the with urinary space.

Stain - H&E, silver methenamine stain

Stimulus: Fibrin leaked into the Bowman space found b/w the cellular layer.



Tubules → show casts And Necrosis.

Immunofluorescence Microscopy: