RENAL PATHO

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	ETIOLOGY	**GROSS**	**LIGHT MICROSCOPY** **(for Histopathology)**	**ELECTRON MICROSCOPY** **(For location)**	**IMMUNO FLUORESCENCE** **(for composition)**	**CLINICAL FEATURE**
APGN/ PSGN	-Group A beta hemolytic streptococci -Immune complex mediated injury	Flea bitten kidney	Enlarged, hypercellular glomeruli	Subepithelial humps	IgG and complement C3 granular deposits	Young child, presenting with acute nephritic syndrome
RPGN Type 1: Anti GBM Disease **(Goodpasture Syndrome)**	-Antigen appears to be a component of collagen Type IV in GBM (Fixed Antigen) -Auto immune disease - Epi membranous deposits - in situ fixed deposition		Crescents (Composition of cresecents: epithelial cells + fibrin + macrophage)	Linear deposits along the GBM	IgG + C3	- Acute renal failure - Nephritic syndrome - Pulmonary hemorrhages (hemoptysis)
RPGN Type II	- PSGN or diffuse proliferative GN - Subepithelial deposits - in situ planted deposition		Crescents	Electron dense sub epithelial granular deposits	IgG + C3	- Acute renal failure - Nephritic syndrome
RPGN Type III	-pauci-immune (little or no glomerular immune deposit) - most common type E.g. Wegener's granulomatosis and microscopic polyarteritis nodosa - No complexes		Crescents	No deposits	Scanty or no deposits	- Acute renal failure - Nephritic syndrome

Minimal Change Disease	-usually idiopathic - may be associated with Hodgkin lymphoma - cell mediated	Normal	No change	Effacement of foot processes (Flattened podocytes)	No important findings	Nephrotic syndrome with normal biopsy
Membranous GN	- GBM thickened - Auto immune disease - Antigen appears to be gp 330, a component of podocyte (Fixed Antigen)		Thickening of basement membrane	Subepithelial deposits, spike and dome (basement membrane protrudes between deposits as spikes)	IgG + C3	Nephrotic syndrome
MPGN	- GBM thickened - increase cellularity		MPGN Type 1 - Tram track appearance (splitting of GBM)	MPGN I - Subendothelial deposits MPGN II- electron dense deposits in basement membrane	MPGN I - IgG + C3 MPGN II - IF show C3 but not IgG	
FSGN	**Focal** - some lobules are involved, some are not **Segmental** - half normal, half diseased **GN** - increased cellularity (proliferation)					
FSGS	**Focal** - some lobules are involved, some are not	Focal, segmental		Effacement of foot processes	- usually negative	

	Segmental - half normal, half diseased **GS** - sclerosis	lesions			- sometimes IgM, C3, C1	
IgA Nephropathy					IgA and C3	
Alport Syndrome	- Hereditary nephritis - XI linked dominant - Inherited defect in Type IV collagen		Diffuse GBM thinning	Alternate thickening and attenuation (thinning) pattern of GBM (basket weave appearance)		Alport triad 1. Sensorineural deafness 2. Ophthalmic complications 3. Proteinuria and hematuria

SIGNS IN NEPHRITIC SYNDROME:

- Microscopic Hematuria
- Mild Proteinuria
- Hypertension
- Edema
- Oliguria

Main problem in nephritic is sodium water retention (as sodium water is retained due to damaged filtration barrier of kidney)
Cause of edema: Sodium water retention

SIGNS IN NEPHROTIC SYNDROME

- Massive proteinuria
- Hypo albuminemia
- Edema
- Hyperlipidemia
- Lipiduria
- Hypercoagulability

Main problem in nephrotic is massive proteinuria.
Cause of edema: hypo albuminemia