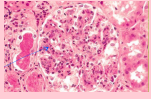

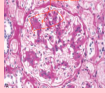
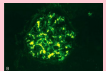
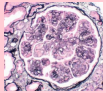
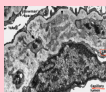
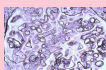



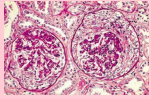
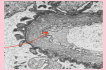


Nephritic

	Cause	In Microscope	Related	Clinical	Microscopy
ADPGN	<p>"POST STREPTOCOCCAL GN"</p> <p>presents 1 - 4weeks after a strep infection of throat or skin.</p> <p>Immune complex disease/ circulating or implanted Ag or both.</p> <p>Implicated Ags streptococcal exotoxin B (Spe B) and streptococcal glyceraldehyde-3- phosphate dehydrogenase (GAPDH) affinity for glomerular proteins and plasmin</p>	<p>LM : increased cellularity. (1) Diffuse proliferation & swelling of endothelial & mesangial cells. (2) infiltrating neutrophils& monocytes.</p> <p>EM : shows deposited immune complexes as subepithelial "humps"</p> <p>IF : scattered granular deposits of IgG& complement within the capillary walls&Mesangium.</p>	<p>Usually in children (6 - 10 years)</p> <p>Prognosis -children >95% recovery, 1% RPGN, 2% CRF -Adults 15-50% develop ESRD</p>	<p>Most commonly present as acute nephritic syndrome</p> <p>Fever,nausea,gross hematuria,&mild proteinuria</p> <p>Urine---- red cell casts</p> <p>-ASO titer increase -Decrease in serum complement</p>	 
IgA-N	<p>Commonest type of GN</p> <p>Usually 1 to 2 days after URTI</p> <p>Genetic or acquired abnormality leading to increase IgA synthesis by mucosal surfaces after antigenic stimulation [1] Circulating IgA aggregates or complexes entrapped in mesangium and activate alternative pathway.</p>	<p>LM : (different finding) normal/ mesangio prolif focal /diffuse prolif.</p> <p>EM : dense deposits in the mesangium</p> <p>IF : deposition of IgA andC3,intho mesangial region. (diagnostic)</p>	<p>Prognosis -initial benign course but slowly progressive -20 - 50% progress to CRF in 20 years -20 - 60% recur in transplants.</p> <p>Bad prognostic features: -old age -heavy proteinuria. -Hypertension -sclerosis</p>	<p>Increased frequency in individuals with celiac disease in liver disease (secondary IgA nephropathy).</p>	 
MPGN	<p>alterations in the basement membrane, proliferation of glomerular cells and leukocyte infiltration.</p> <p>Secondary cause: -chronic immune complex disorder (SLE, HCV, HIV) -malignant conditions (CLL, lymphoma, melanoma)</p> <p>Consider as : nephrotic syndrome, nephritic syndrome, proteinuria, hematuria or nephritic/nephritic.</p>	<p>Type</p> <ul style="list-style-type: none"> MPGN type I <ul style="list-style-type: none"> LM : enlarged glomeruli, proliferation + infiltration of inflammatory cells, lobular accentuation, thickening of capillary walls "reduplication" of glom capillary "tram-tracking", crescents may be seen.tubulointerstitial changes & vascular changes of HT. EM subendothelial deposits, circumferential mesangial interposition, increase in mesangial cells & matrix. IF C3 ,C1q,C4 in granular pattern in mesangial area. Type II (DDD) <ul style="list-style-type: none"> LM similar to type I EM Intramembranous dense deposits lamina densa transformation into an irregular , ribbon - like ,extremely electron dense structure IF granular mesangial & short or discontinuous linear capillary loop deposits of C3. No early complement components or Igs They have persistent low C3 >70% have C3 nephritic factor (C3NeF), an auto Ab that stabilizes C3 convertase leading to persistence of C3 degradation & hypo-complementemia Mutation of factor H, or autoantibodies to factor H. 	<p>Children and young adults</p>	<p>50% of cases is the nephrotic syndrome, may begin as acute nephritis or mild proteinuria.</p> <p>Prognosis: Generally poor. 40% progress to end-stage renal failure, 30% had variable degrees of renal insufficiency, 30% had persistent nephrotic syndrome without renal failure.</p> <p>MPGN type I: is more common than DDD.</p> <p>DDD has a worse prognosis, and it tends to recur in renal transplant recipients</p> <p>Type 1</p>    <p>Type 2</p>  	
RPGN	<p>rapid and progressive loss of renal function with severe oliguria and (if not treated) death from renal failure within weeks or months.</p> <p>not a single disease it is a syndrome which could be caused by a number of diseases both primary of kidney and systemic diseases.</p>	<p>LM : (CRESCENTIC GN) > 50 - 75% of glomeruli contain crescents obliterating Bowman capsule and compressing the underlying glomeruli which could show normal, or focal,proliferative changes.</p> <p>EM : rupture of GBM only or with electron dense deposits</p> <p>IF : linear, granular or none.</p> <p>ACCORDING TO IMMUNOFLUORESCENCE FINDING</p> <ol style="list-style-type: none"> 1- Anti-GBM antibody-mediated crescentic GN: <ul style="list-style-type: none"> -Linear pattern for IgG & C3 (12%) - Anti glomerular basement membrane disease - anti bodies to GBM, could cross react with pulmonary alveolar BM to produce the clinical syndrome of lung hemorrhage and renal failure (Good Pasture's syndrome). 2-Immune complex-mediated crescentic GN: <ul style="list-style-type: none"> -Granular pattern for IgG & C3 (44%) - post infect, SLE, IgAnN, HSP - idiopathic 3- Pauci-immune type crescentic GN: <ul style="list-style-type: none"> - Immunofl. Negative (44%) -most have ANCA in serum (ANCA Associated RPGN) -some associated with systemic vasculitis. - the rest no association (idiopathic). 		<p>presence of crescents in most glomeruli</p> <p>-Prognosis depends roughly on the fraction of the involved glomeruli. -Milder forms may subside but renal involvement is usually progressive leading to oliguria.</p> <p>Therapy - Plasmapheresis (immune complex-mediated crescentic GN usually doesn't respond) - steroids - cytotoxic drugs - Some patients requires long term dialysis, and renal transplant.</p>	
Hereditary Nephritis	<p>A group of heterogeneous hereditary - familial renal diseases associated with glomerular lesions.</p> <p>EX:Alport syndrome</p> <p>Defective GBM synthesis, mutation in encoding for alpha-5 chain of collagen type IV.</p>	<p>LM - Normal glomeruli early in the disease, secondary sclerosis later, Foam cells in the interstitium.</p> <p>EM ----GBM shows irregular thickening, lamination, splitting ("basketweave" appearance)</p>		<p>-Nephritis + nerve deafness + eye disorders. -Males > females - X-linked, AR or AD</p> <p>-Males 5 - 20 yrs (Gross hematuria). in 20 yrs (CRF)</p>	

(وَقُلْ رَبِّ أَدْخِلْنِي مُدْخَلَ صِدْقٍ وَأَخْرِجْنِي مُخْرَجَ صِدْقٍ
وَاجْعَلْ لِي مِنْ لَدُنْكَ سُلْطَانًا نَصِيرًا)

اجعل مداخلي ومخارجي كلها في طاعتك وعلى مرضاتك، وذلك لتضمنها بالإخلاص وموافقته الأمر.
﴿وَاجْعَلْ لِي مِنْ لَدُنْكَ سُلْطَانًا نَصِيرًا﴾ أي: حجة ظاهرة، وبرهاناً قاطعاً على جميع ما أتبه وما أذره.
وهذا أعلى حالة ينزلها الله العبد، أن تكون أحواله كلها خيراً ومقربة له إلى ربه، وأن يكون له - على كل
حالة من أحواله - دليلاً ظاهراً، وذلك متضمن للعلم النافع، والعمل الصالح، للعلم بالمسائل والدلائل!
- تفسير السعدي.