Non immune nildren imary opear normal	complex Old primary	Immune complex (type III) Adult primary
imary		
·	primary	primary
ppear normal		
	 obliteration of capillary Lumina increased mesangial matrix hyalinosis 	Thickening of the capillary wall <u>Sliver stain:</u> GMB spike
fuse (effacement)of podocyte foot		Sub epithelial deposition of immune comp
ocess		(cause the thickening of BM)
	Trapping of IgM and complement (C3) in GMB and podocytes.	<u>Granular</u> <u>deposits</u> in Loop pattern of(IgG&C3) along GBM
esponds well to corticosteroid therapy	Responds to corticosteroid is poor	Does not usually respond to corticosteroid
	And 50% of patient develop end stage kidney disease	
	- May be primary or secondary :	*MGN caused by SLE ,and association sometime include hepatitis B, syphilis and
	1-Primary (idiopathic).	drugs (penicillamin ,captopril)
C		brease Trapping of IgM and complement (C3) in GMB and podocytes. sponds well to corticosteroid therapy Responds to corticosteroid is poor And 50% of patient develop end stage kidney disease - May be primary or secondary :

	Diabetic nephropathy	Renal amyloidosis
Causes of NS	Systemic	Systemic
LM	Will be either :	Massive amyloid deposits are present in glomeruli and
	*diffuse glomerulsclerosis: increase in mesangial	arterioles "hyalinization of blood vessels"
	matrix and mesangial cell	
		Polarized light with Special stain of amyloid (Congo
	*Nodular glomeulosclerosis :nodular accumulation	Red) : tubular involvement with amyloid is verified by
	of mesangial matrix called (kimmelstiel Wilson	apple-green birefringence.
	nodules)	
	*Arteriolar hyalinization. (detect the disease)	
	*Tubulointerstitial fibrosis.	
EM	Increase in thickness of GBM	characteristic fibrils appearance
Note	Most important cause	Two type of amyloid
		1 Amyloid AA :chronic inflammatory condition
		(reactive protein)like(TB,rheumatoid arthritis)
		2 Amyloid AL :tumors affect plasma
		cells,immunoglobin will effected (multiple myloma)

Nephritic syndrome

The diseases that cause nephritic syndrome have in common proliferation cells within the glomeruli, accompanied with inflammatory reaction.

-<u>Clinical manifestations</u> : oliguria ,azotemia, <u>Hematuria</u> with dysmorphic red cell and RBC cast and hypertension.

	Poststreptococcal GN	Alport syndrome	IgA Nephropathy
Allso called	Acute proliferative GN	hereditary nephritis	Berger disease
Most effect	Children	Children (especially male)	
LM	Neutrophil within capillaries . proliferation of glomeruli (mesangial& epithelial cells)		Mesangial widening.
EM	Dense subepithelial (hump)	Irregular BM thickening	Ideposition of IgA within mesangium.
IF	Granular deposition of IgG & C3 (lumpy bumpy)		dense deposition of IgA
Comment	Prognosis : Complete recovery in children, but some of them develop rapidly progressive crescentic GN or chronic renal disease In Adult depends on severity (worse than children)	Caused by mutation in gene for (α-5 chain) which form collagen IV which important of GBM and cause structural abnormality	Immune complex

	cent between bowman's capsule & glom		
	Anti-glomerular BM	Pauci immune GN	Immune complex mediated
Also called	Good posture syndrome	Wegener granulomatosis	
	Immune complex	Non Immune complex	
Pathogenesis	Formation of antibody which direct against glomerular & pulmonary alveolar BM	+ive ANCA serum	Complication of any immune complex like Post streptococcal GN. SLE. IgA nephropathy. Henoch-Schonlein purpura
LM	Characterized by formation of crescent		
IF	Linear deposition of IgG & c3		granular deposition

Membranoproliferative GN
Can be Nephrotic or Nephritic syndrome
Is marked by replication of GBM into 2 layer due to expansion of mesangial matrix
Disease occurs in two forms :
1)type I:LM: tram track appearance EM: accumulation of immune complex
2) type II Dense Deposit Disease: EM :irregular electron dense material