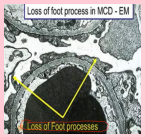
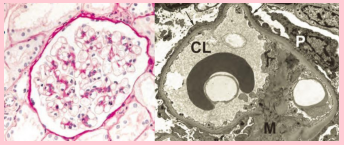
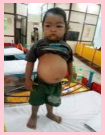
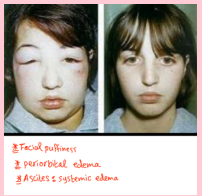
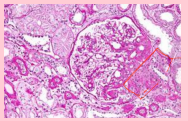
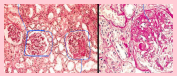
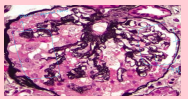
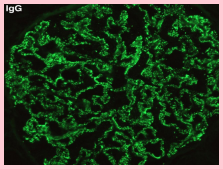
Nephrotic Syndrome/primary dz

	Cause	In microscope	Related	CLINICAL PRESENTATION	📸
MCD	(Lipoid nephrosis , nil change disease) fusion of the podocytes (foot processes) of epithelial cells 	LM : normal IF : normal EM : fusion of podocyte 	most common cause of NS in Children (2 - 6y) y children 65%, adults (10%) may follow URTI or immunization	-selective proteinuria -respond to steroids -renal function normal -Prognosis =excellent 	
FSGS	Sclerosis of some, but not all glomeruli and only part of the glomerulus is involved. mutations affecting cytoskeletal or related proteins expressed in podocytes (e.g., nephrin); APOL1 gene on CH.22 in individuals of African descent	LM : Sclerotic Segments Show Collapse Of B.M. Deposition Of Hyaline Masses (HYALINOSIS). Increased Mesangial Matrix IF : IgM & C3 IN Sclerotic Segments In affected glomeruli ,negative or nonspecific trapping of immunoglobulins EM : NON -Sclerotic Segments Show Loss Of Podocytes(exhibit effacement of foot processes as in MCD). Focal denudation of Epithelial cells.	35% Adults 10% Children Can occur in: (1) Association with known conditions: HIV, Heroin addiction, sickle cell disease and Obesity. (2)Glomerular scarring in other forms of GN. e.g IgA nephropathy. (3) as a maladaptation after nephron loss.	* NEPHROTIC SYNDROME -Nonselective proteinuria - poor response to steroids. - A higher incidence of hematuria, reduced GFR, and HT. -Prognosis=50% will develop end-stage renal failure in 10 yrs -DIFFERENTIAL DIAGNOSIS(MCD&MGN) *Collapsing glomerulopathy- FSGS morphologic variant Collapse glomerular tuft & epithelial cell hyperplasia. severe formwith worse prognosis Can be: idiopathic, ass/with HIV infection, or drug-induced toxicities	  
MGN	uniform thickening of the capillary wall due to diffuse deposition of electron dense deposits on epithelial aspect of GBM. 	LM: Thick capillary wall ; No proliferation. "SPIKES " by SILVER stain IF : granular deposits IgG &C3 EM : Subepithelial deposits, which nestle against the GBM & are separated from each other by small, spike-like protrusions of GBM matrix that formin reaction to the deposits(spike&dome pattern) 	30% Adults(30-60yrs) 5% children. 85% Idiopathic, 15% Secondary. 	->60%of patients have(Nonselective proteinuria) - poor response to steroids. - 33% stable with proteinuria -30-33% benign course(partial or complete remission of proteinuria) -33-40% progress to renal failure (end- stage renal disease)over 2 to 20 years.