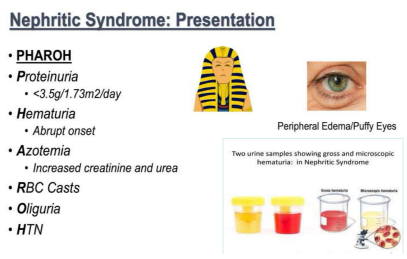
Nephrotic Syndrome

Topic	Details
Definition	Clinical complex due to glomerular disease.
Key Features	1. Massive proteinuria (>3.5g/day in adults) 2. Hypoalbuminemia (≤ 3g/dL) 3. Generalized edema 4. Hyperlipidemia and lipiduria 5. Little or no azotemia, hematuria, or hypertension
Causes	A. Primary Glomerular Diseases: - Minimal Change Disease (MCD) - Focal Segmental Glomerulosclerosis (FSGS) - Membranous Nephropathy - Membranoproliferative GN type 1 B. Secondary Causes: - Diabetes Mellitus - Amyloidosis - SLE - Drugs (gold, penicillamine, street heroin) - Infections (malaria, syphilis, HBV, HIV) - Malignancies (carcinoma, melanoma) - Others (bee-sting allergy)
Prevalence	In Children: - Minimal Change Disease (65%) - FSGS (10%) - Membranoproliferative GN (10%) - Membranous GN (5%) - IgA Nephropathy (10%) In Adults: - Membranous GN (30%) - FSGS (35%) - MCD (10%) - Membranoproliferative GN (10%) - IgA Nephropathy (15%)

Disease	Key Features	Morphology	Clinical Course & Treatment
Minimal Change Disease (MCD)	- Most common in children (ages 1–7) - Podocyte injury (T-cell factor?) - Selective albuminuria	LM: Normal IF: Negative EM: Effacement of foot processes, no immune deposits	- Nephrotic syndrome in healthy child - No hypertension - Renal function preserved - Excellent response to corticosteroids (90%) - Adults: slower response & frequent relapse
Focal Segmental Glomerulosclerosis (FSGS)	- Segmental sclerosis in some glomeruli - Primary (20–30% of NS) or secondary (e.g., HIV, heroin) - Non-selective proteinuria - Possible hematuria & hypertension	LM: Segmental sclerosis IF: Negative EM: Effaced foot processes Special: Collapsing variant (poor prognosis, linked to HIV)	- Poor response to steroids - ~50% develop renal failure in 10 years - Adults worse than children
Membranous Nephropathy	- Immune complex deposition - 85% primary (anti-PLA2R antibodies) - Secondary: infections (HBV, syphilis), tumors, SLE, drugs (NSAIDs, gold), toxins	LM: Diffuse GBM thickening IF: Granular IgG & complement deposits EM: Subepithelial deposits; “spike and dome” pattern	- Often nephrotic syndrome - Poor steroid response - 60%: persistent proteinuria - 40%: progressive to renal failure (2–20 yrs) - 30%: partial/complete remission

Nephritic Syndrome

Topic	Details
Definition & Pathogenesis 	- Inflammation of glomeruli - Leukocyte infiltration and proliferation - Capillary wall injury → RBCs in urine (hematuria, RBC casts) - ↓ GFR → oliguria, fluid retention, azotemia - Hypertension (from fluid overload + ↑ renin) - May have mild proteinuria
Common Diseases	1. Membranoproliferative Glomerulonephritis (MPGN) 2. Acute Postinfectious Glomerulonephritis (PSGN) 3. IgA Nephropathy (Berger Disease)

Membranoproliferative Glomerulonephritis (MPGN) .1

Type	Pathogenesis	Morphology	Clinical Course
Type I (80%)	- Immune complex disease - Associated with: HBV, HCV, SLE, infected A-V shunts	LM: Large lobular glomeruli, mesangial/ endothelial proliferation, leukocytes, “tram-track” GBM IF: IgG + complement (C1q, C4) – subendothelial deposits EM: Subendothelial electron-dense deposits	- Poor prognosis - No remission - 40% → end-stage renal disease - 30% → variable insufficiency
Type II (Dense Deposit Disease)	- Caused by autoantibody (C3 nephritic factor) that stabilizes C3 convertase → excessive alternative pathway activation → ↓ complement	LM: Similar to type I IF: C3 only in GBM EM: Dense ribbon-like intramembranous deposits	- Worse prognosis - Tends to recur in kidney transplant recipients

2. Acute Postinfectious (Poststreptococcal) Glomerulonephritis (PSGN)

Cause	Morphology	Clinical Course
- Immune reaction to previous infection (usually Group A strep) - Occurs 1–4 weeks after pharyngitis/skin infection	LM: Hypercellular glomeruli with endothelial, mesangial proliferation & neutrophils IF: IgG + C3 along capillary walls EM: Subepithelial “humps” (immune complexes)	- Mostly in children - Acute onset: fever, nausea, nephritic signs - Gross hematuria, mild proteinuria - Low complement (during active phase) - ↑ ASO titers - Usually complete recovery in children

3. IgA Nephropathy (Berger Disease)

Features	Morphology	Clinical Course
- Recurrent hematuria (microscopic or gross) - Common in children/young adults - Occurs 1–2 days after respiratory infections - Resolves then recurs periodically	LM: Variable IF: IgA + C3 in mesangium EM: Mesangial deposits	- Variable prognosis - Can progress slowly to chronic renal failure

Disease	Syndrome	Age	LM	IF	EM	Prognosis
MCD	Nephrotic	Children	Normal	Negative	Foot process effacement	Good
FSGS	Nephrotic	Adults	Segmental sclerosis	Negative	Foot process effacement	Progressive
Membranous GN	Nephrotic	Adults	Thick GBM	IgG + C3	Subepithelial “spikes & domes”	Progressive
MPGN Type I	Nephritic/ Nephrotic	Adults	“Tram track”	IgG + C1q/C4	Subendothe-lial deposits	Poor
MPGN Type II	Nephritic/ Nephrotic	Adults	“Tram track”	C3 only	Dense intramem-branous	Poor
IgA Nephropathy	Nephritic	Young	Variable	IgA + C3	Mesangial deposits	Variable
PSGN	Nephritic	Children	Hypercellular	IgG + C3	Subepithelial “humps”	Good