ACUTE GLOMERULONEPHRITIS & HEMATURIA

1. Hematuria Fundamentals

- **Definition:** Presence of **> 5 RBCs/HPF** in 3 out of 3 consecutive centrifuged specimens, obtained at least 1 week apart.
- **Crucial Finding:** The presence of **RBC Casts** is ALWAYS pathological and points directly to a **Glomerular** source of bleeding.

Glomerular Causes

- IgA Nephropathy (Most common GN)
- Systemic Lupus Erythematosus (SLE)
- Membranoproliferative GN (MPGN)
- Alport Syndrome
- Goodpasture Disease (Anti-GBM)
- Hemolytic Uremic Syndrome (HUS)
- Rapidly Progressive GN (RPGN)

Non-Glomerular Causes

- **Infection:** Bacterial UTI, Viral, Tuberculosis.
- **Hematologic:** Coagulopathy, Thrombocytopenia, Sickle Cell, Renal Vein Thrombosis.
- **Structural:** Stones, Hypercalciuria, Polycystic Kidney, Trauma, Tumors.

2. Acute Nephritic Syndrome

Glomerulonephritis is an immune-mediated inflammation of the glomerulus. The classic presentation of **Acute Nephritic Syndrome** involves:

- **Hematuria** (gross or microscopic, with RBC casts)
- **Hypertension** (HTN)
- **Azotemia / Oliguria** (High Cr and BUN)
- **Edema** (Due to salt/water retention from decreased GFR)

3. Post-Streptococcal GN (PSGN)

PSGN is the classic acute nephritic syndrome. Peak age is **5-15 years** (rare before age 3). 95% of cases resolve completely.

- **Pathology:** Immune-complex mediated. On electron microscopy (EM), you see **Subepithelial "Humps"** (electron-dense deposits).
- **Diagnosis:** Confirmed by throat culture or positive ASO titer / Anti-DNAase B.
- **Complications:** Volume overload, Heart Failure, HTN Encephalopathy, Hyperkalemia.
- **Treatment:** Supportive (Diuretics, CCBs, ACE-inhibitors). A 10-day course of penicillin limits spread but **does NOT change the natural history** of the nephritis.

MEMORY AID: PSGN SEROTYPES & SEASONS

Group A Beta-Hemolytic Streptococcus has specific "nephritogenic" strains:

- **T**hroat Infection = Serotype **12** (Occurs in Winter)
- **S**kin Infection (Impetigo) = Serotype **49** (Occurs in Summer)

Mnemonic: T-12 (Winter) | S-49 (Summer).

MCQ TRAP: RENAL BIOPSY IN PSGN

Renal biopsy is **NOT INDICATED** in a classical PSGN presentation.
Only biopsy if presentation is atypical: 1. Acute Renal Failure (RPGN) 2. Nephrotic syndrome 3. No evidence of preceding strep infection 4. Normal complements 5. **Persistent low C3/C4 for > 3 months** (points toward SLE or MPGN instead).

4. IgA Nephropathy (Berger Disease)

The most common cause of glomerulonephritis worldwide (especially Japan, France, Italy). Peaks in the 2nd/3rd decades. Male:Female ratio is 2:1.

- **Pathophysiology:** Galactose-deficient IgA1 acts as an auto-antigen. Immune complexes deposit in the renal **mesangium**. *Unlike HSP nephritis, this occurs WITHOUT systemic symptoms.*
- **Diagnosis:** **Definitive diagnosis requires Renal Biopsy.** LM shows mesangial proliferation. EM/IF shows IgA deposits.
- **Treatment:** Fish oil, steroids, ACEi/ARBs (for HTN & proteinuria), Tonsillectomy.

PROGNOSIS IN IGA NEPHROPATHY

30% progress to ESRD. Persistent microscopic hematuria does **NOT** correlate with poor prognosis.
Poor Prognostic Signs: 1. Hypertension, 2. Heavy proteinuria (>1g/L), 3. Crescents on biopsy.

5. The Ultimate MCQ Differentiators: PSGN vs. IgA Nephropathy

Examiners love testing the differences between PSGN and IgA Nephropathy. Memorize these two critical differentiators:

Feature	Post-Streptococcal GN (PSGN)	IgA Nephropathy
Latency Timing (CRITICAL)	**1-2 WEEKS** after pharyngitis. **3-4 WEEKS** after skin infection.	**1-2 DAYS** after URTI (Synpharyngitic).
Complement Levels (C3/C4)	**Low C3**, but **Normal C4**. *(Returns to normal in 2-3 months)*	**Normal C3** and **Normal C4**.
Classic Biopsy Finding	Subepithelial electron-dense "humps".	Mesangial IgA deposits.
Resolution	Complete recovery in >95%. Recurrence is extremely rare.	Chronic course. 30% progress to ESRD. Recurrences are common.