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Founding Member, Mass General Brigham

Proteinuria, Hematuria & Glomerular Disease

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DISCLOSURES

Please state any/all financial disclosures for past 24 months.

Scientific Advisory Board: Vera Therapeutics, Travere Therapeutics

Learning Objectives:

Workup of hematuria and proteinuria

Mechanisms and etiologies of glomerular disease

Correlate presentations to workup of glomerular disease

Hematuria

Glomerular	Structural	Systemic
IgA nephropathy	RCC, Prostate Ca, Stone	Anticoagulant or antiplatelet therapy
ANCA vasculitis	Pyelonephritis/cystitis/prostatitis/urethritis	Coagulopathy/bleeding disorder
Lupus nephritis	Ureteral or urethral stricture	Sickle cell trait/disease
Anti-GBM disease	Benign prostatic hyperplasia	Renal infarction (embolic/thrombotic)
Post-infectious GN	Polycystic kidney disease	Pelvic radiation (radiation cystitis)
Alport syndrome	Arteriovenous malformation	Renal/pelvic trauma/Instrumentation
Thin basement membrane disease	Papillary necrosis	Exercise-induced (march) hematuria
	Schistosomiasis	Loin pain–hematuria syndrome

Pseudohematuria (-Dipstick/No RBCs)

Dietary Sources

- Beets (beeturia)
- Blackberries
- Rhubarb
- Foods with synthetic red/pink dyes

Medications

- Phenazopyridine (urinary analgesic)
- Rifampin (antibiotic)
- Phenytoin
- Chloroquine
- Deferoxamine
- Laxatives containing senna

Metabolic & Endogenous Factors

- Porphyria (can result in wine-colored urine)
- Alkaptonuria (urine turns black upon standing)
- High concentrations of urates

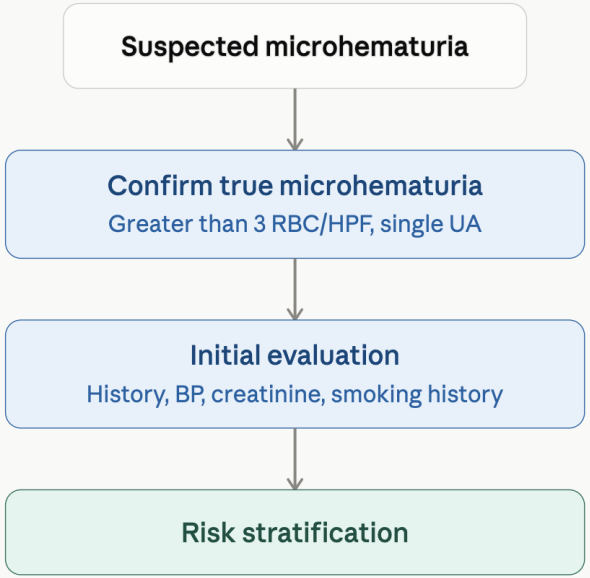
RBCs vs Hemoglobin vs Myoglobin

	Hematuria	Hemoglobinuria	Myoglobinuria
Dipstick	Positive for blood	Positive for blood	Positive for blood
Urine microscopy	**RBCs present (>3/HPF)**	No RBCs (or rare)	No RBCs (or rare)
Centrifuged urine	Sediment red, **supernatant clear/yellow**	Sediment clear, supernatant red/pink	Sediment clear, supernatant red/brown
Centrifuged serum	Clear (normal color)	**Pink/red (hemolyzed)**	Clear (normal color)
Serum haptoglobin	Normal	**Low/decreased**	Normal
CK	Normal	Normal	**Markedly elevated**
Causes	Glomerular disease, stones, tumors, infection, trauma, coagulopathy	Intravascular hemolysis: hemolytic anemia, transfusion reaction, G6PD deficiency, malaria, mechanical heart valve, TTP/HUS	Rhabdomyolysis: crush injury, extreme exertion, statins, seizures, prolonged immobilization

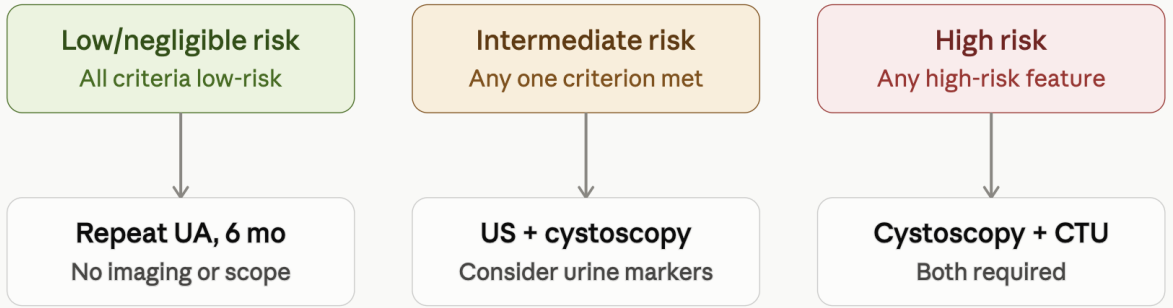
Hematuria risk stratification

	Low/negligible (0–0.4%)	Intermediate (0.2–3.1%)	High (1.3–6.3%)
Criteria needed	**All** must apply	**Any one** applies	**Any one** applies
RBC/HPF	3–10	11–25 or Persistent low risk MH	>25, or history of gross hematuria
Age	Women <60, men <40	Women ≥60, men 40–59	Men ≥60 (women not high-risk by age alone)
Smoking	Never or <10 pack-yrs	10–30 pack-yrs	>30 pack-yrs
Other risk factors	None	Any	One or more, plus a high-risk feature

Barocas, AUA/SUFU Guideline 2025



Once risk category is assigned, the workup diverges into three distinct pathways:



Barocas, AUA/SUFU Guideline 2025

Hematuria Evaluation – Take Home

Confirm first → 3 RBC/HPF on a single, properly collected UA before proceeding.

Risk-stratify — low, intermediate, or high; low-risk requires meeting *all* criteria.

Low risk → repeat UA at 6 months; no imaging or cystoscopy yet.

Intermediate risk → ultrasound + cystoscopy; urine markers can help defer the scope.

High risk → cystoscopy + CT urography, both required.

Hereditary risk overrides category — RCC family history, genetic syndromes, or Lynch syndrome need upper tract imaging regardless of risk tier.

Markers are adjuncts, not screens — not routine for low or high risk, and not routine after a normal cystoscopy.

Think glomerular? — dysmorphic RBCs, casts, or proteinuria mean nephrology referral, not this pathway.

Proteinuria

Normal excretion <150 mg/24 h

- 60% is filtered plasma protein (20-40 mg of albumin)
- 40% are glycoproteins -- Immunoglobulins, uromodulin

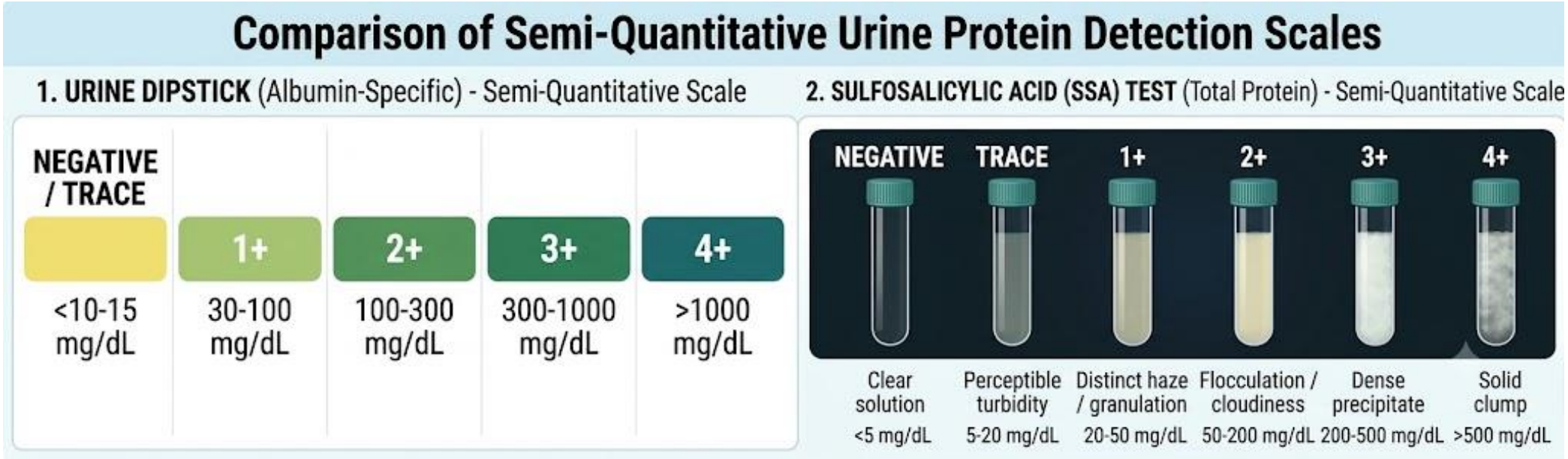
Detected by urine dipsticks (specific to albumin)

- Detects >10-15 mg/dL; almost always (+) if urine alb > 30 mg/dL

Moderately increased = 30-300 mg/24h
Severely increased = >300 mg/24h

Estimate protein (and albumin) excretion by measuring the ratio of protein to creatinine (to account for urine volume)
Proteinuria is one of the best predictors of progression of renal disease

Dipstick vs SSA



Dipstick uses a pH-indicator dye (tetrabromophenol blue) that changes color only when it binds **albumin**. It's fast and convenient, but structurally blind to other proteins.

SSA works by a completely different principle — sulfosalicylic acid denatures and precipitates **any** protein in solution (albumin, globulins, and immunoglobulin light chains), read out as turbidity rather than color.

Proteinuria Pearls

- Very high (or low) creatinine production can change the protein estimation as the formula assumes 1g creatinine production daily
- LMW proteins are usually absorbed in the proximal tubule so a high ratio of total protein to albumin may suggest either over-production of light chains (myeloma) or a proximal tubular disorder
- There is large intra-individual variation in the albumin/creatinine ratio even in a single day

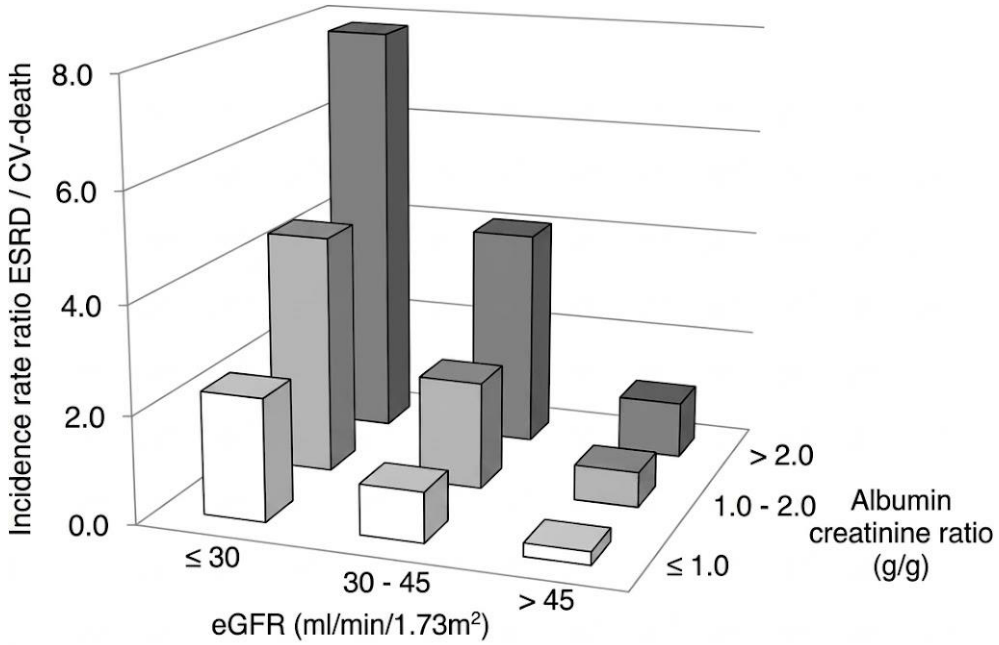
Tubular Proteinuria

- Results from **impaired proximal tubular reabsorption** of normally filtered low-molecular-weight proteins (β2-microglobulin, retinol-binding protein) rather than glomerular damage
- Typically **mild** — usually <1-2 g/day, rarely nephrotic-range
- Causes: ATN, interstitial nephritis, Fanconi syndrome, heavy metal toxicity, drug-induced tubular injury, reflux nephropathy
- Urine protein electrophoresis shows predominance of low-molecular-weight proteins rather than albumin
- Often accompanied by other proximal tubular dysfunction: glucosuria (with normal serum glucose), aminoaciduria, phosphaturia

Orthostatic Proteinuria

- Proteinuria that occurs only in the **upright position** and normalizes when supine - a benign condition
- Most common in adolescents and young adults, often incidentally found on routine screening
- Usually <1g/day
- Diagnosed by comparing a first-morning urine sample (after overnight recumbency) to a daytime sample: first-morning is protein-negative or minimal, daytime shows proteinuria. Or Split 12-hour collections.
- Typically mild, non-progressive, with normal renal function, no hematuria
- Usually resolves spontaneously with age; no treatment needed, just periodic monitoring to confirm it stays benign

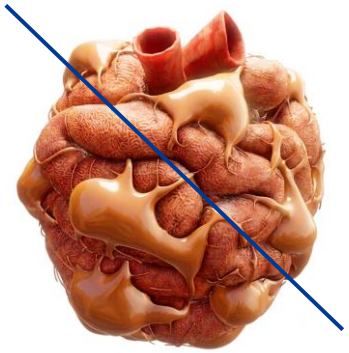
Risk for ESRD increases as proteinuria increases and GFR decreases

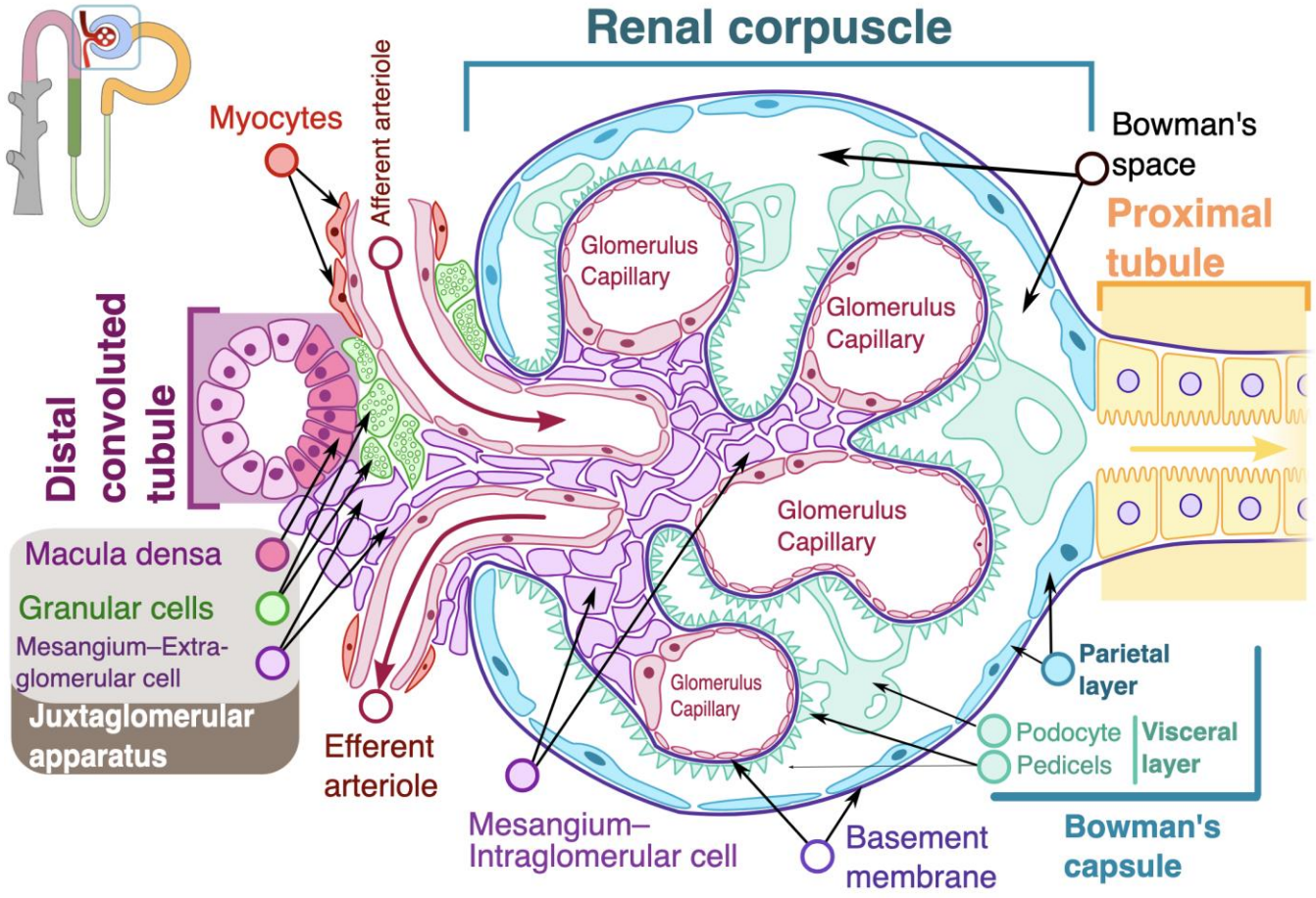


ACR (g/g)	eGFR (ml/min/1.73m²) ≤30	30 - 45	>45
> 2.0	12.87 (5.97-27.74)	7.46 (3.63-15.33)	7.40 (3.32-16.47)
1.0 – 2.0	7.12 (3.16-16.04)	3.47 (1.63-7.40)	2.80 (1.18-6.64)
≤1.0	3.61 (1.49-8.73)	1.49 (0.64-3.48)	1.00 (reference)

Umanath, AJKD 2018

Glomerulus Structure





The Glomerulus: Where Injury Drives Presentation

Functional structures

- Capillary tuft — site of filtration
- Mesangium — structural support
- Glomerular basement membrane (GBM)
- Podocytes — visceral epithelial cells
- Bowman's capsule — parietal epithelium

Site of injury → syndrome

Endothelial / mesangial	**Nephritic**
Podocyte	**Nephrotic**
GBM (immune)	**Nephritic / RPGN**
Crescentic (any cause)	**RPGN — emergency**

Four Presentations of Glomerular Disease

Nephrotic

Nephritic

RPGN

Asymptomatic urinary abnormalities

Four Presentations of Glomerular Disease

Nephrotic

Proteinuria >3.5 g/day · albumin <3.0 · edema · hyperlipidemia

Nephritic

Hematuria + RBC casts · HTN · moderate proteinuria · ↑ Cr

RPGN

≥50% eGFR decline in days–weeks · crescents on biopsy

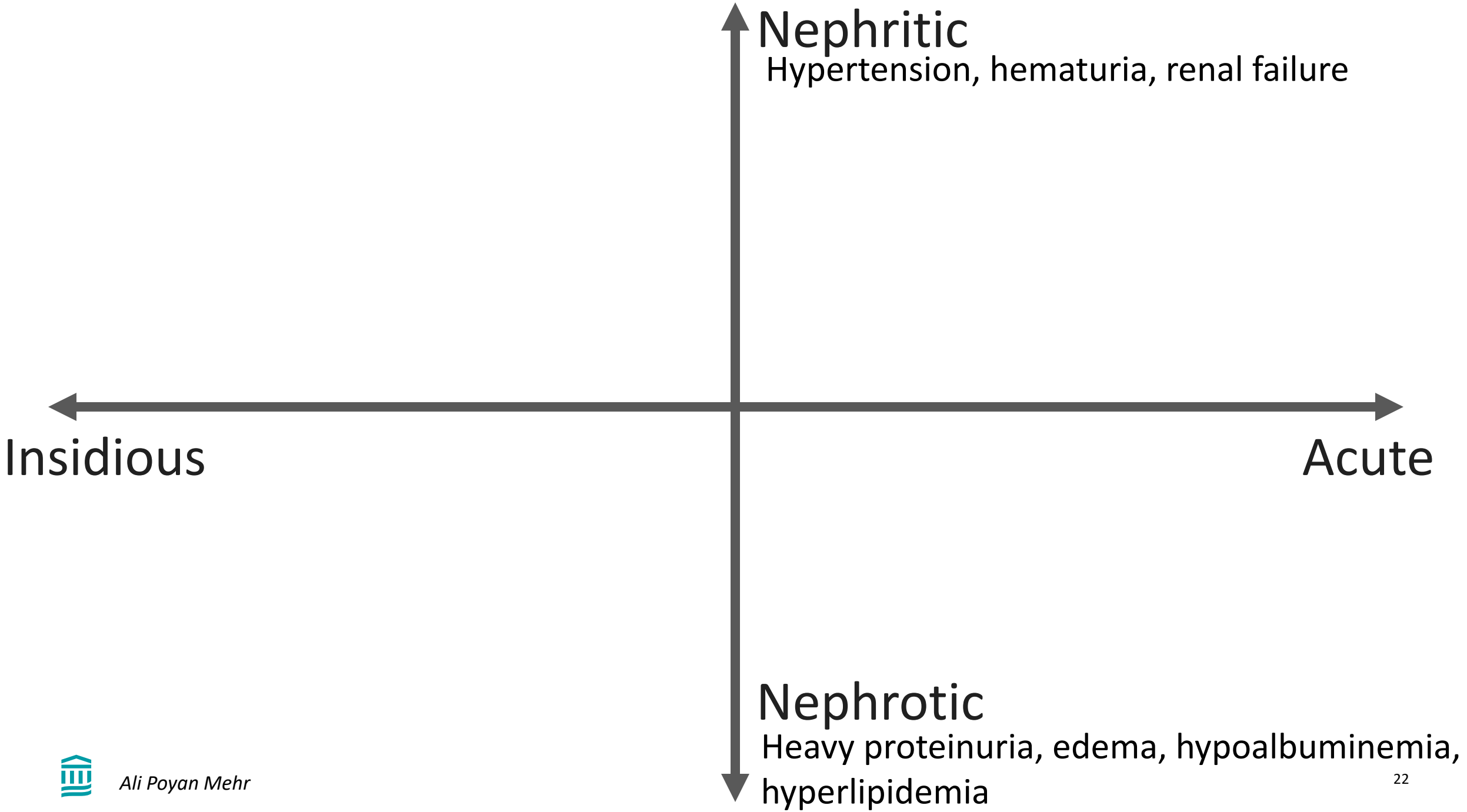
Asymptomatic urinary abnormalities

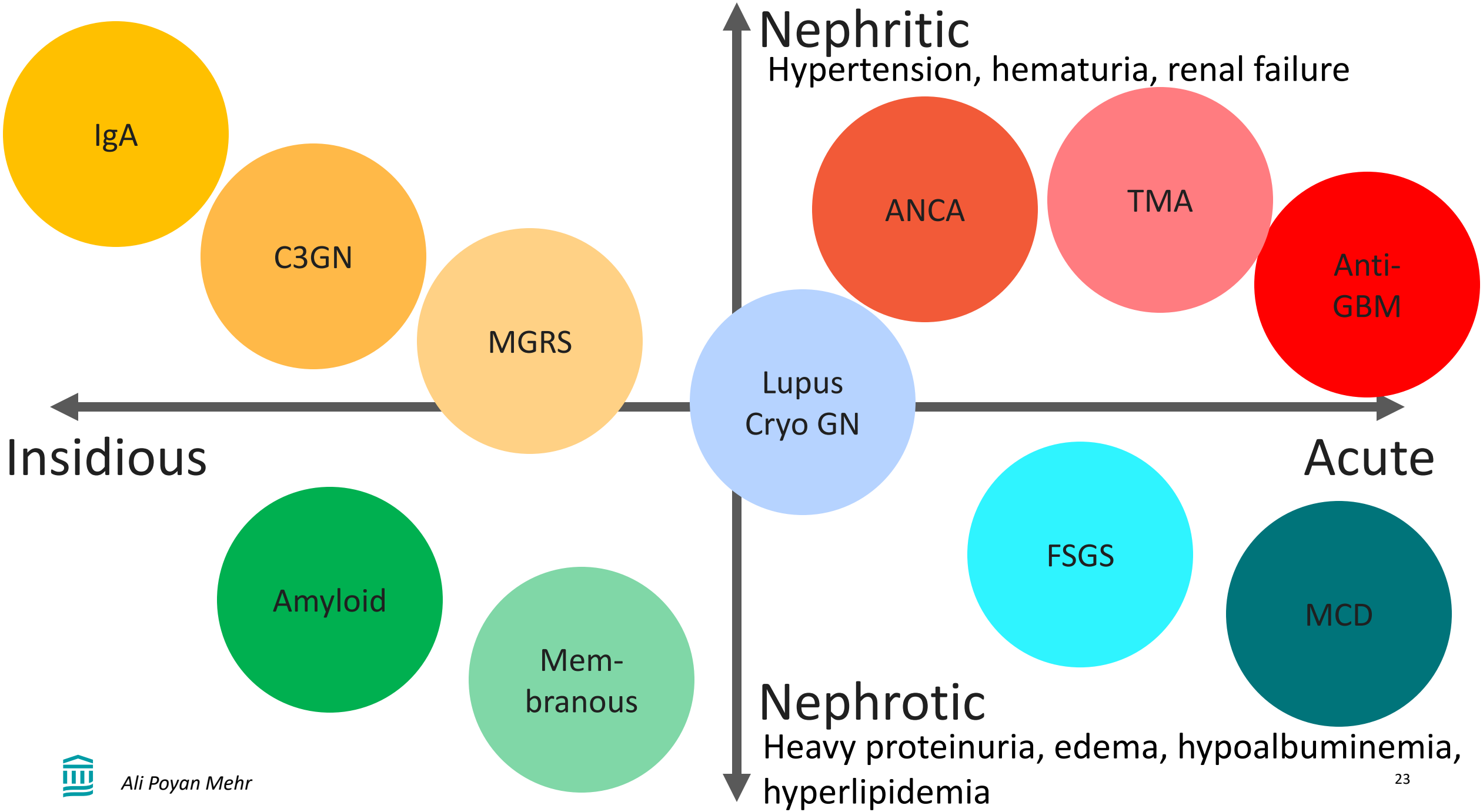
Incidental hematuria and/or proteinuria · most common presentation

Glomerular Disease: Clinical syndromes

	Nephrotic	Nephritic
Edema	++++	++
BP	Normal	Raised
JVP	Normal/Low	Raised
Proteinuria	++++	++
Hematuria	+/-	+++
RBC Casts	Absent	Present
Albumin	<3.0g/dL	Normal/Slight ↓

Adapted from Comprehensive Clinical Nephrology 3rd ed, Feehally et al.


Nephritic
Hypertension, hematuria, renal failure
Insidious
Acute
Nephrotic
Heavy proteinuria, edema, hypoalbuminemia, hyperlipidemia


Nephritic
Hypertension, hematuria, renal failure
IgA
C3GN
MGRS
ANCA
TMA
Anti-GBM
Lupus Cryo GN
Insidious
Acute
Amyloid
Mem-branous
FSGS
MCD
Nephrotic
Heavy proteinuria, edema, hypoalbuminemia, hyperlipidemia

Four Presentations of Glomerular Disease

Nephrotic

Proteinuria >3.5 g/day · albumin <3.0 · edema · hyperlipidemia

Classic causes

MCD, FSGS, membranous, diabetic, amyloid

Nephritic

Hematuria + RBC casts · HTN · moderate proteinuria · ↑ Cr

Classic causes

IgAN, post-infectious GN, lupus nephritis

RPGN

≥50% eGFR decline in days–weeks · crescents on biopsy

Classic causes

ANCA vasculitis, anti-GBM, immune-complex

Asymptomatic urinary abnormalities

Incidental hematuria and/or proteinuria · most common presentation

Classic causes

Thin basement membrane, mild IgAN, early disease

Start With the Urine

Urinalysis & sediment is the single most important first test.

On the dipstick & sediment

- Dysmorphic RBCs → glomerular bleeding
- RBC casts → pathognomonic
- Heavy proteinuria → podocyte disease
- Lipiduria, oval fat bodies → nephrotic
- WBC casts → interstitial nephritis (not GN)

Quantifying proteinuria

- Spot UPCR (g/g) — fast, well-validated
- 24-hour urine — gold standard but cumbersome
- Nephrotic range = >3.5 g/day (UPCR >3.5)
- First morning specimen reduces orthostatic noise

Send the Full Serologic Panel — Upfront

C3 / C4	Low C3+C4 → lupus, cryo. Low C3 only → post-infectious, C3GN, MPGN
ANA, anti-dsDNA	Lupus nephritis
ANCA (MPO, PR3)	Pauci-immune crescentic GN
Anti-GBM	Anti-GBM disease / Goodpasture
Anti-PLA2R	Primary membranous nephropathy
Hep B, Hep C, HIV	Secondary GN, MPGN pattern, HCV-cryoglobulinemia
SPEP / SFLC	Patients >50 — exclude monoclonal gammopathy
ASO / anti-DNase B	Post-streptococcal GN
Cryoglobulins, RF	Cryoglobulinemic GN (often with HCV)
CBC, CMP, UA	Always — establishes baseline and severity

Kidney Biopsy: The Gold Standard

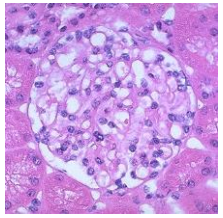
What the biopsy answers

- Etiology — via immunofluorescence pattern
- Severity — pattern and extent of injury
- Chronicity — degree of sclerosis & fibrosis
- Prognosis — guides aggressiveness of therapy

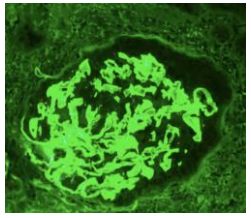
When to biopsy

- Adult-onset nephrotic syndrome
- Hematuria + proteinuria >0.5 g/day
- Unexplained AKI without obvious cause
- Any RPGN
- Serologic workup inconclusive

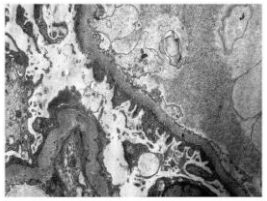
> *Generally avoided: longstanding diabetic with retinopathy + slow progressive proteinuria — clinical diagnosis usually suffices.*



Light microscopy



Immunofluorescence



Electron microscopy

Pathogenesis-Based Classification (by IF)

#	Category	IF / mechanism	Examples
1	**Immune-complex GN**	Granular polyclonal Ig deposits	*IgA nephropathy, lupus nephritis, post-infectious GN*
2	**Pauci-immune**	Little or no Ig staining; ANCA-associated	*GPA, MPA, EGPA*
3	**Anti-GBM**	Linear IgG along GBM	*Anti-GBM disease, Goodpasture*
4	**Monoclonal Ig GN**	Monoclonal heavy/light chain deposits	*PGNMID, MIDD*
5	**C3 glomerulopathy**	Complement-dominant, Ig-negative	*C3GN*

Nephrotic Syndrome

Defining features

- Proteinuria >3.5 g/24 hr (or UPCR >3.5)
- Serum albumin <3.0 g/dL
- Peripheral edema, often anasarca
- Hyperlipidemia
- Bland sediment (no RBC casts)

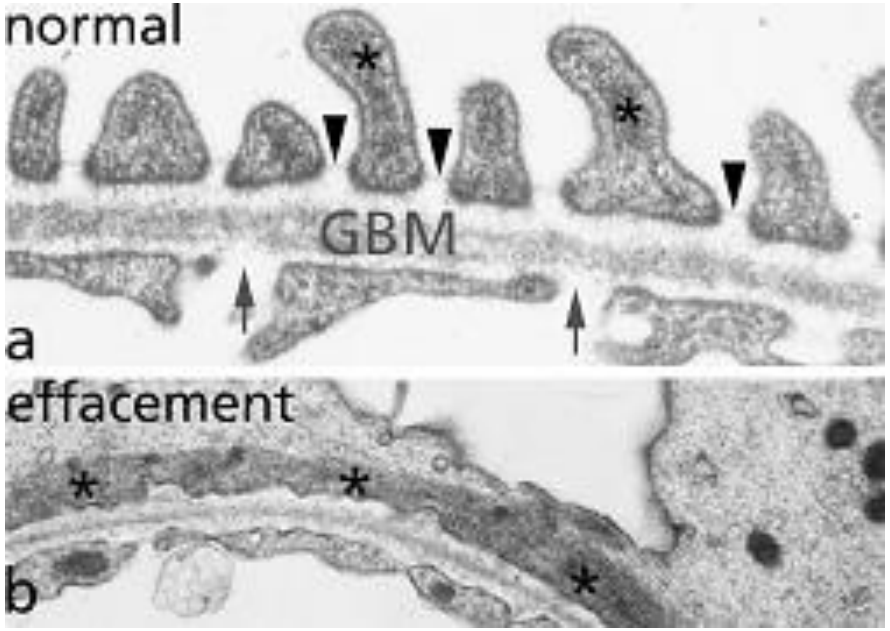
Mechanism

Podocyte injury → loss of size/charge selectivity → massive albumin loss.

Classic causes by epidemiology

- Children → minimal change disease
- Adults (white) → membranous nephropathy
- Adults (Black) → FSGS
- All adults → diabetic nephropathy, amyloid

Nephrotic Syndrome

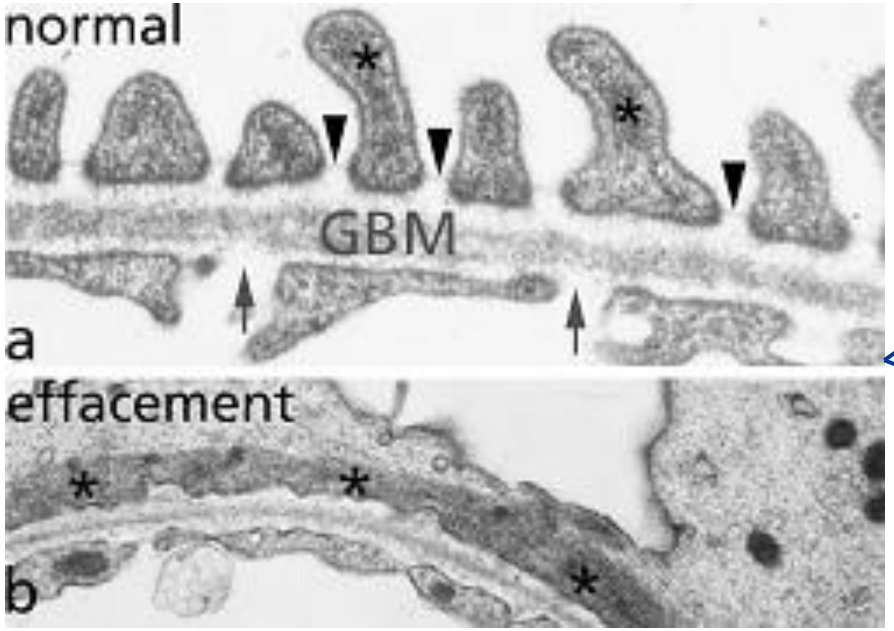


Nephrotic Syndrome

Normal
Podocytes

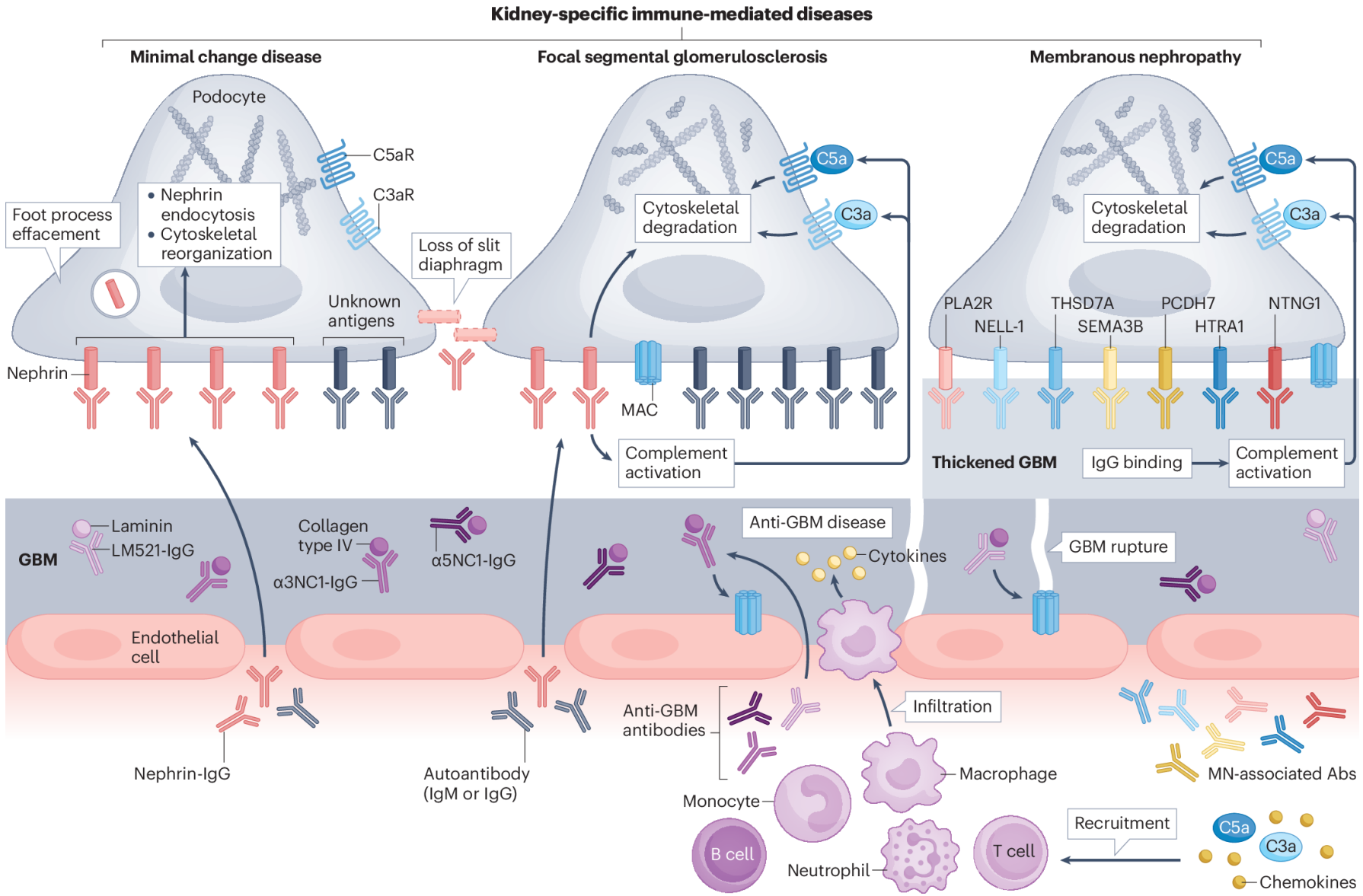
Effaced
Podocytes

Nephrotic Syndrome



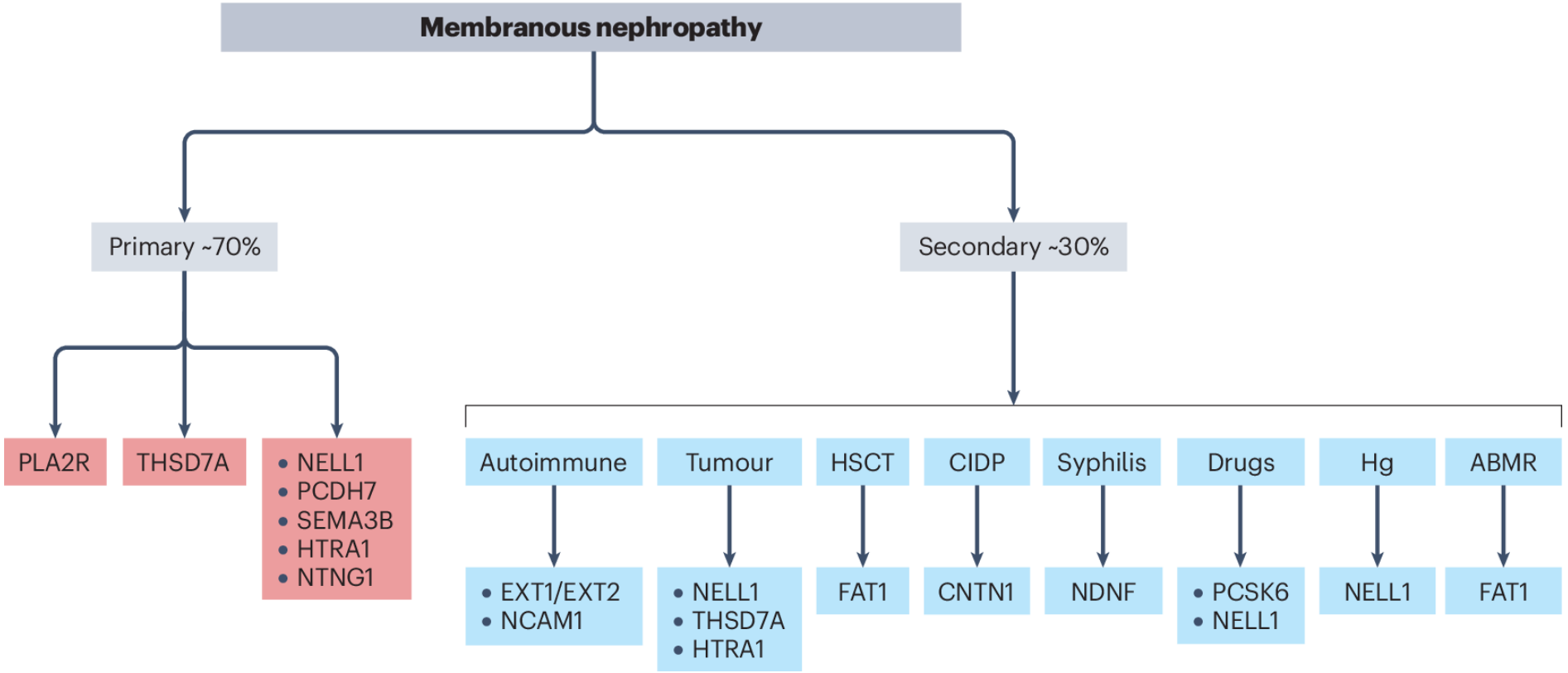
Immunogenic

Non-immunogenic



Cui, Nature Reviews Nephrology, 2024

Membranous Nephropathy (Causes)



Sethi, Nature Reviews Nephrology, 2025

Minimal Change Disease	FSGS
NSAIDs and other drugs (interferon, lithium, gold, bisphosphonates)	Heroin nephropathy and other drugs (interferon, anabolic steroids, pamidronate/bisphosphonates)
Hodgkin lymphoma and other lymphoproliferative disorders	Viral infections: HIV, parvovirus B19, CMV, SARS-CoV-2 (COVAN)
Allergic reactions / bee sting, food allergy	Obesity / adaptive hyperfiltration
Graft-versus-host disease (post-transplant)	Reduced nephron mass (renal agenesis, reflux nephropathy, low birth weight, prior nephrectomy, reflux/obstructive uropathy)
	Sickle cell disease, Chronic vesicoureteral reflux
	Genetic podocyte gene mutations (e.g., NPHS1/nephrin, NPHS2/podocin, ACTN4, TRPC6, INF2)
	APOL1 high-risk genotype (especially in individuals of West African ancestry)
	Chronic scarring from any prior glomerular injury ("secondary FSGS" as a common final scarring pathway)

Nephritic Syndrome

Defining features

- Hematuria with dysmorphic RBCs
- RBC casts (pathognomonic for glomerular bleeding)
- Subnephrotic proteinuria
- Hypertension, oliguria, edema
- Rising creatinine

Mechanism

Endocapillary or mesangial inflammation → leakage of RBCs and protein → ↓ filtration.

Classic causes

- IgA nephropathy (most common worldwide)
- Post-infectious GN (post-strep classic)
- Lupus nephritis
- MPGN pattern (cryoglobulinemia, HCV)

Rapidly Progressive Glomerulonephritis (RPGN)

Nephrologic emergency — initiate workup and consult same day

Definition

≥50% decline in eGFR over days to weeks. Histologic hallmark = crescent formation in Bowman's space.

ANCA-associated

Pauci-immune crescentic GN. MPO or PR3. Adults, often elderly.

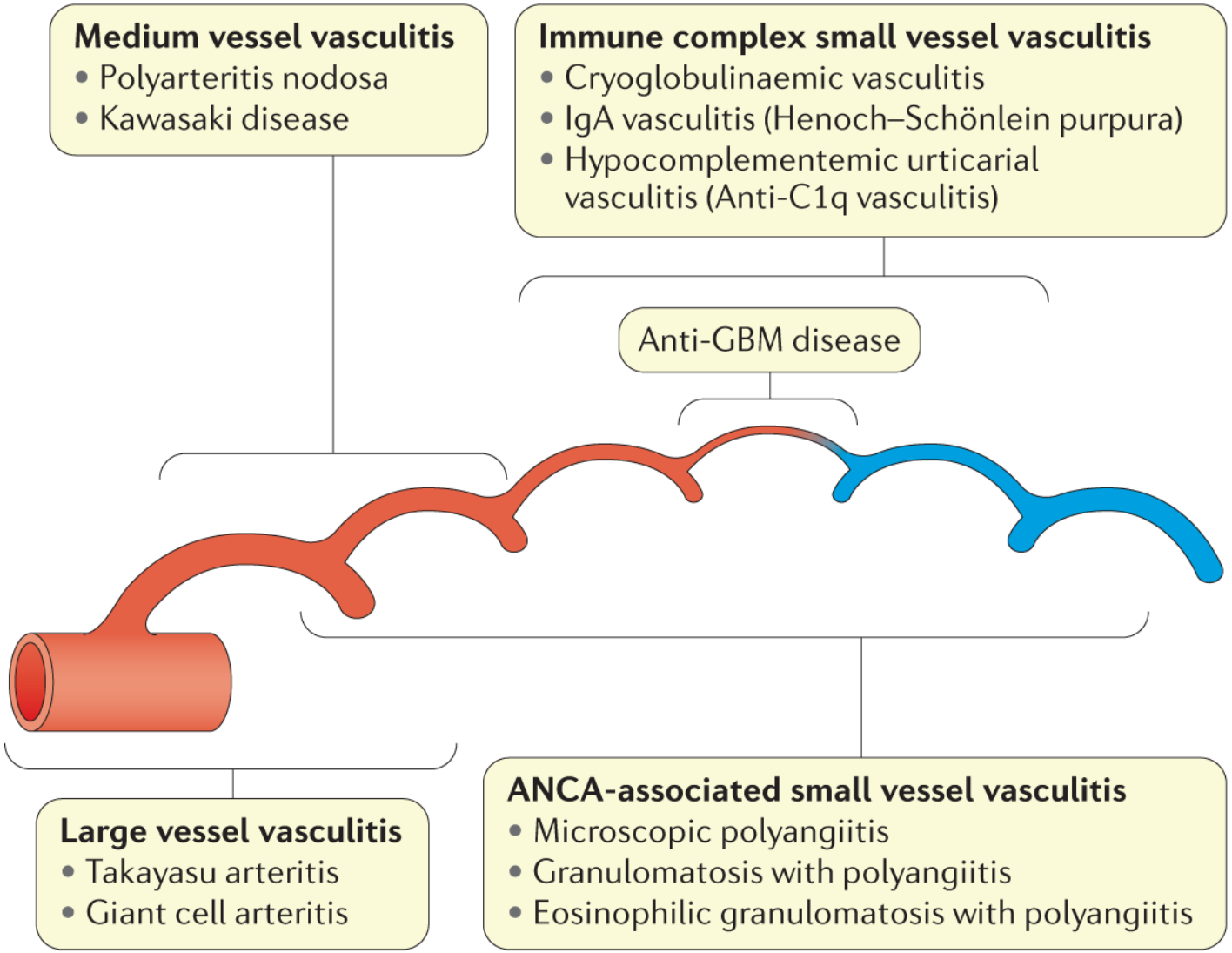
Anti-GBM

Linear IgG along GBM. ± alveolar hemorrhage (Goodpasture).

Immune-complex

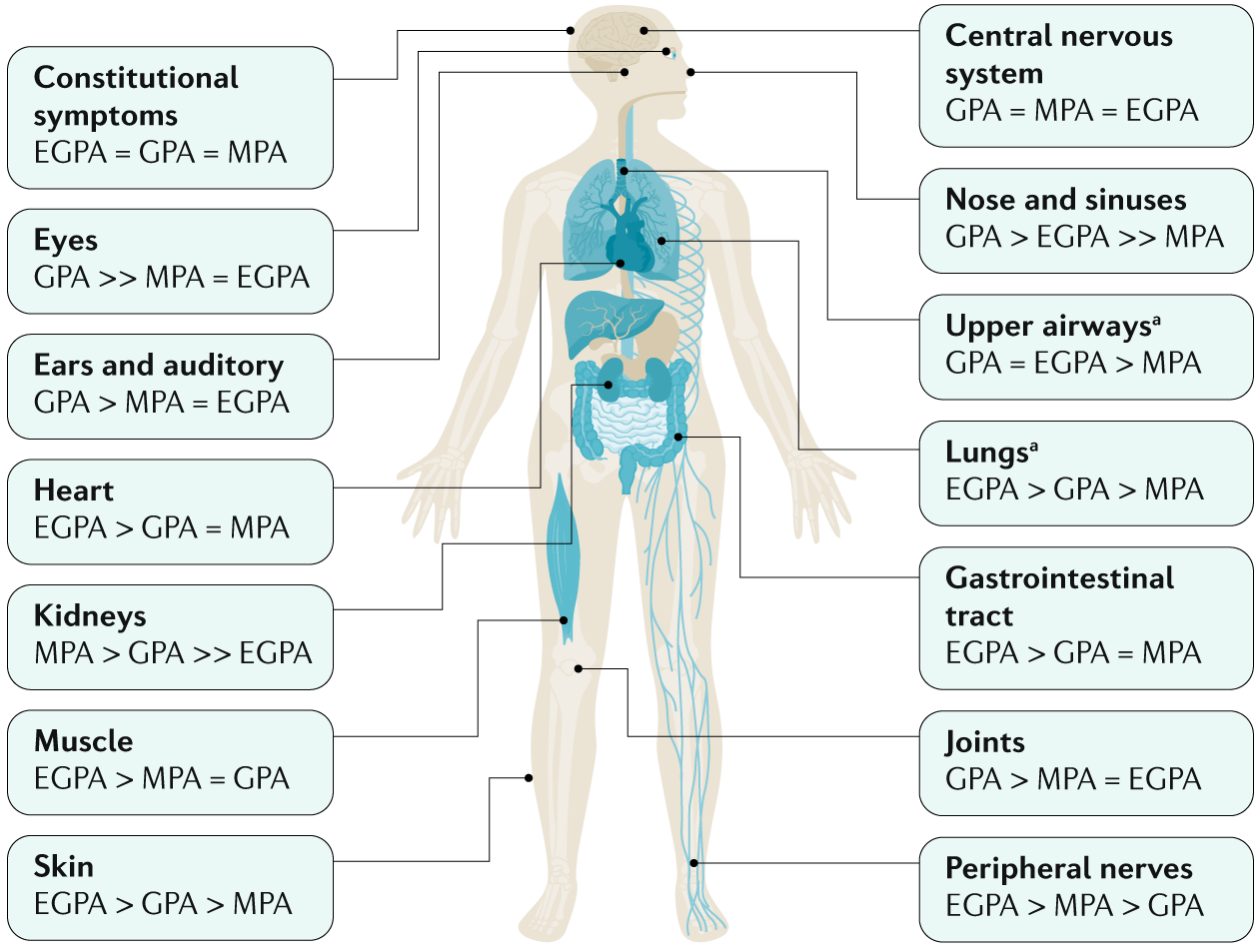
IgA, lupus, post-infectious, cryo, MPGN can all crescent.

ANCA



2012 Chapel Hill Consensus Conference Classification of Vasculitis

ANCA



granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA) and eosinophilic GPA (EGPA).

Kitching, Nature Reviews, 2020

Feature	ANCA Vasculitis	Anti-GBM Disease
Antibody target	Proteinase-3 (PR3) or myeloperoxidase (MPO) in neutrophils	Type IV collagen (α3 chain) in glomerular/alveolar basement membrane
Biopsy pattern	Pauci-immune (little/no immune deposits)	Linear IgG deposition along GBM
Systemic involvement	Multi-organ — sinuses, lungs, skin, nerves, eyes, GI (GPA, MPA, EGPA subtypes)	Largely limited to kidney and lung ("Goodpasture syndrome" when both involved)
Course	Often relapsing-remitting	Typically monophasic — one acute episode, less prone to relapse once treated
Age/epidemiology	Wider age range, often older adults	Bimodal: young adults (often with lung hemorrhage, smokers) and older adults (renal-limited)
Other clues	Sinusitis, nodules, mononeuritis multiplex, cavitary lung lesions, saddle-nose deformity	Isolated pulmonary-renal presentation, strong smoking/hydrocarbon exposure link for alveolar hemorrhage
Prognosis without overlap	Renal recovery more likely if caught early	Renal recovery poor once dialysis-dependent or with high % crescents

IgA Nephropathy

Most common immune-mediated GN worldwide.

Presentation

- Young adults, mean age 34–45
- Synpharyngitic hematuria (Few d post-URI)
- Asymptomatic micro to RPGN across spectrum
- Up to 50% progress to ESKD by 10 years

Treatment — KDIGO 2025

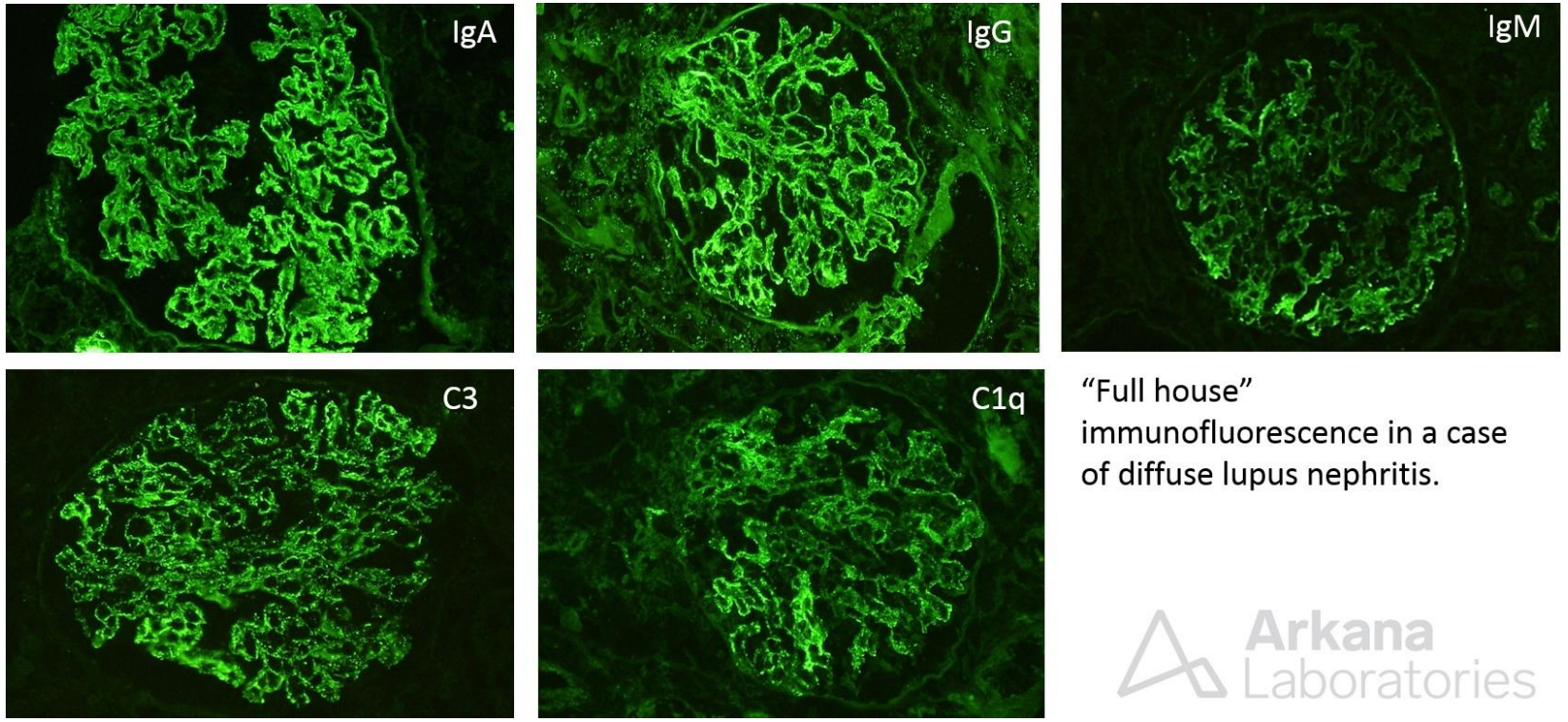
- BP target ≤120/70 · RAS blockade or sparsentan
- SGLT2 inhibitors, ERA
- Anti April/BAFF biologics, budesonide
- Complement inhibitors (iptacopan)
- Avoid systemic steroids in most patients

Lupus Nephritis

50% of patients with Lupus
Full House IF pattern
Class drives treatment. Proliferative classes (III/IV) demand aggressive immunosuppression.

I	Minimal mesangial	Observation; supportive
II	Mesangial proliferative	Supportive ± low-dose steroid
III	Focal proliferative	Induction + maintenance immunosuppression
IV	Diffuse proliferative	Induction + maintenance immunosuppression
V	Membranous	Treat if nephrotic-range proteinuria
VI	Advanced sclerosing	Supportive; prepare for KRT

Lupus Nephritis: ‘Full House’



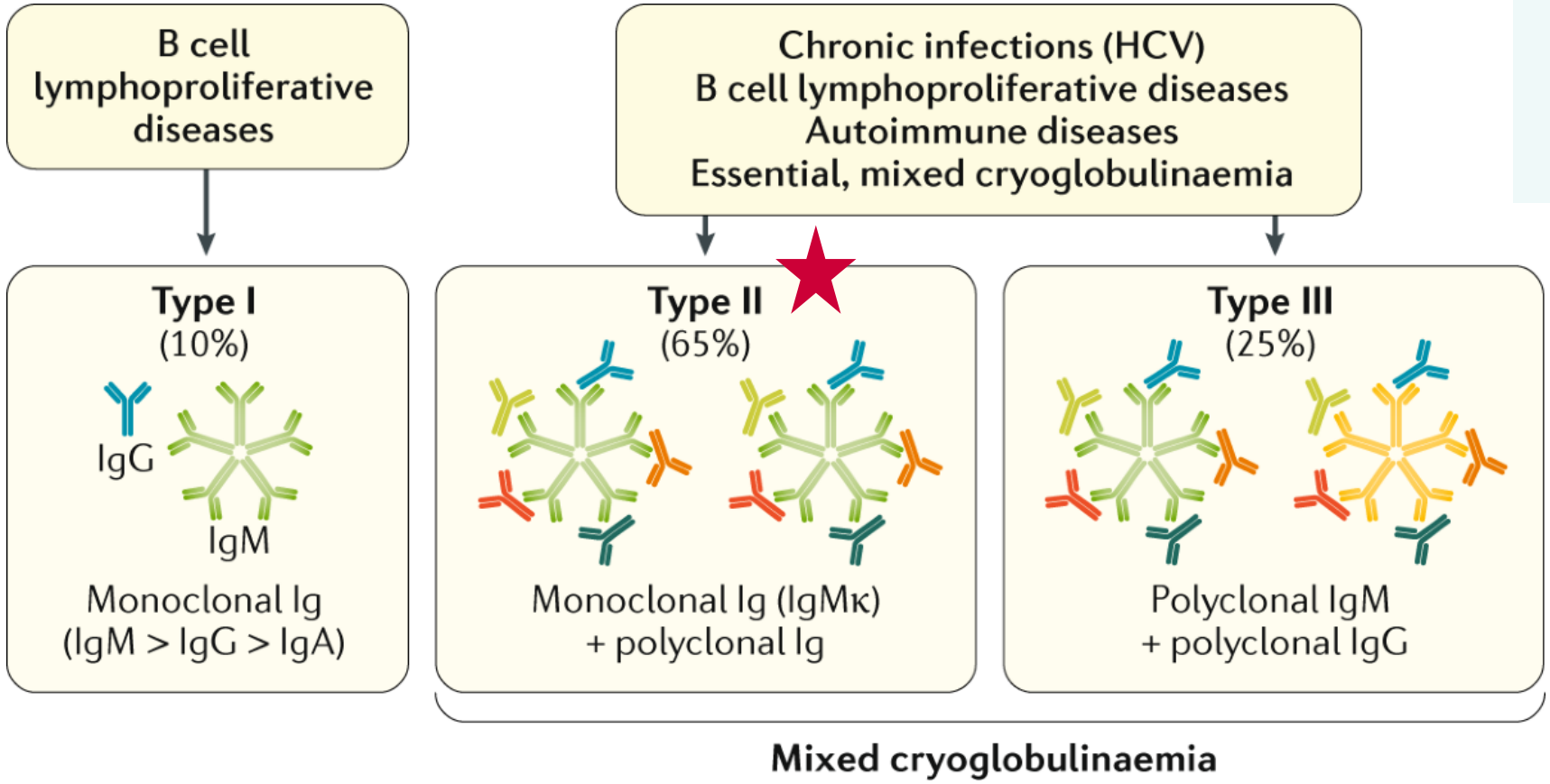
“Full house” immunofluorescence in a case of diffuse lupus nephritis.

Arkana Laboratories

Cryoglobulinemic Vasculitis

Meltzer's triad:
Palpable purpura
Arthralgia
Weakness

Low C4



Roccatello, Nature Reviews, 2018

MGRS (Monoclonal Gammopathy of Renal Significance)

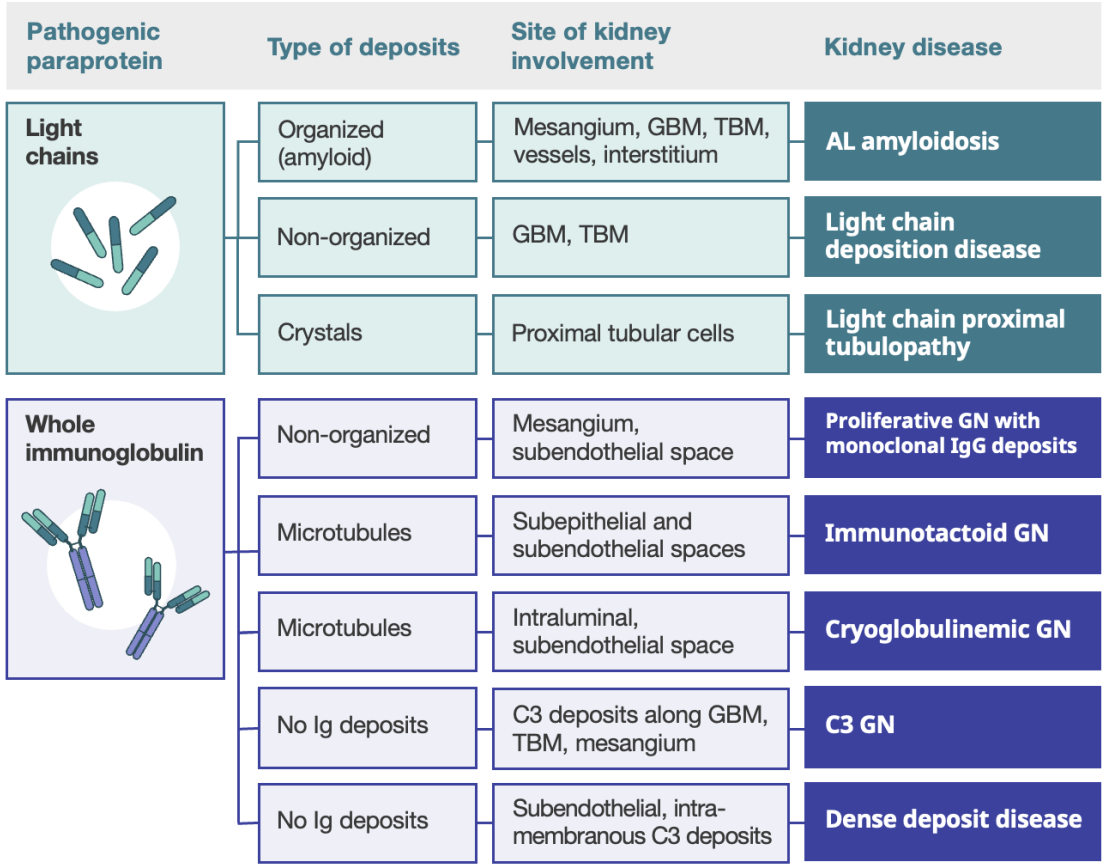


Figure 2. Spectrum of kidney pathology in monoclonal gammopathy of renal significance.
GBM – glomerular basement membrane; GN – glomerulonephritis; TBM – tubular basement membrane.

Achi, KI Reports Community, 2024

MGRS (Monoclonal Gammopathy of Renal Significance)

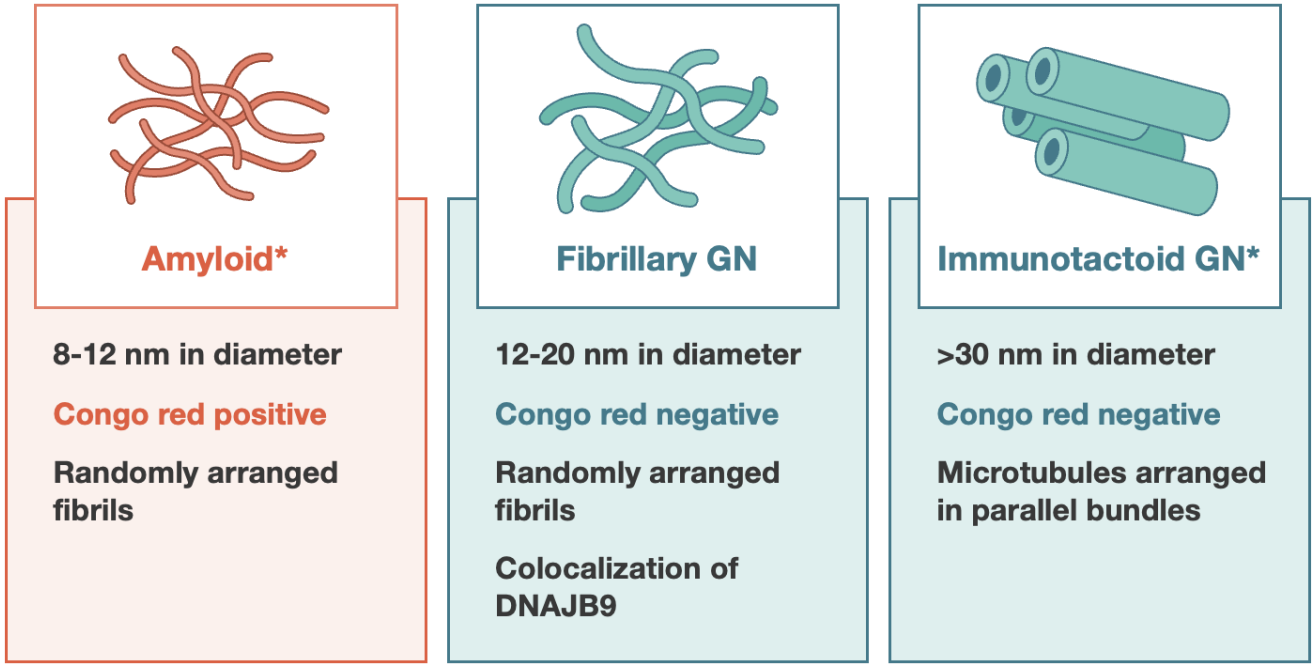


Figure 3. Organized deposits by diameter, orientation and Congo red staining. Deposits marked with (*) are associated with monoclonal gammopathies. DNAJB9 – DnaJ heat shock protein family (Hsp40) member B9.

Achi, KI Reports Community, 2024

Complications of Nephrotic Syndrome

Thromboembolism

7× VTE risk

Highest in membranous (20–30% renal vein thrombosis). Loss of antithrombin III. Anticoagulate if albumin <2.5.

Infection

Encapsulated organisms

Loss of immunoglobulins and complement opsonins → pneumococcal peritonitis, sepsis.

Hyperlipidemia

↑ CV risk

Hepatic compensatory synthesis. Statin therapy often indicated.

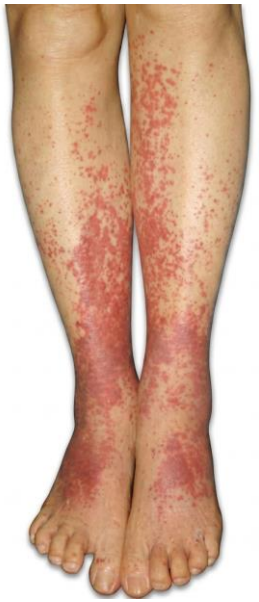
AKI

Hypovolemia or interstitial

Low oncotic pressure → reduced effective circulating volume. Watch with aggressive diuresis.

Clinical Case I

A 42-year-old man presents with a 5-day history of palpable purpuric lesions over his buttocks and lower legs, along with new-onset arthralgias in both ankles and colicky abdominal pain with one episode of melena. He reports a mild upper respiratory illness about 3-4 days earlier. He has no significant past medical history and takes no medications. Vital signs are normal. Exam shows symmetric, non-blanching purpuric papules, mild ankle swelling, and diffuse abdominal tenderness without peritoneal signs. Labs show a creatinine of 1.6 mg/dL (baseline unknown), urinalysis with 2+ blood and 2+ protein, dysmorphic RBCs and RBC casts on microscopy, and a spot urine protein-to-creatinine ratio of 1.8 g/g. Platelet count and coagulation studies are normal. Serum C3 and C4 are normal.



Clinical Case I

Which of the following is the most likely diagnosis?

A. Cryoglobulinemic vasculitis

B. Lupus Nephritis

C. IgA vasculitis (Henoch-Schönlein purpura) with nephritis

D. Post-infectious glomerulonephritis

E. Thrombotic thrombocytopenic purpura

Clinical Case I

Correct Answer:
C. IgA vasculitis (Synpharyngitic, Henoch-Schönlein purpura)

Other answers:
A. Cryoglobulinemic vasculitis: Arthralgia, rash +. Usually linked to HCV, C4 usually low, no abdominal symptoms

B. Lupus Nephritis: Unusual type of rash, C3/C4 normal here.

D. Post-infectious GN: low C3, more lag between infection and kidney involvement.

E. TTP: Thrombocytopenic rash similar, but platelets normal here. No neurological findings.

Clinical Case II

A 58-year-old woman presents with fatigue, hemoptysis, and dyspnea over the past week. She denies fever, joint pain, or rash. Labs show hemoglobin 8.9 g/dL, creatinine 6.4 mg/dL (baseline 0.9 mg/dL one month ago), and urinalysis with 3+ blood, 2+ protein, dysmorphic RBCs, and RBC casts. Chest imaging shows diffuse bilateral alveolar infiltrates. She is started on hemodialysis for volume overload and uremia, and is intubated for hypoxic respiratory failure. Serologic workup (anti-GBM antibody, ANCA, complement levels) has been sent but results are not yet available.

Clinical Case II

What is the most appropriate next step in management?

A. Wait for serologic results before starting any immunosuppressive therapy

B. Start pulse-dose corticosteroids alone; hold plasma exchange until ANCA/anti-GBM results return

C. Start empiric pulse-dose corticosteroids and plasma exchange now, without waiting for serologic confirmation

D. Start cyclophosphamide monotherapy and reassess once serologies result

E. Proceed directly to renal biopsy before initiating any treatment

Clinical Case II

Answer: C. Corticosteroids + Plasma Exchange (ANCA/Anti-GBM, Medical Emergency)

A. **Wait for serology** (could take days)

B. **B. Steroids without plasma exchange** – undertreats diffuse alveolar hemorrhage, one of the few settings where plasma exchange has benefit in *both* ANCA vasculitis (per PEXIVAS, reserved for severe DAH/severe kidney failure) and anti-GBM disease (standard for essentially all organ-threatening cases).

C. **D. Cyclophosphamide alone** – slow onset of effect

D. **E. Biopsy before treatment** – too sick for biopsy

Clinical Case III

A 45-year-old woman with no significant past medical history undergoes a routine urinalysis as part of an annual physical exam, which shows 15 RBC/HPF. She denies gross hematuria, dysuria, flank pain, or recent exercise, menses, or catheterization. She has never smoked, has no occupational chemical exposures, no history of pelvic radiation or cyclophosphamide use, and no family history of urologic malignancy. Blood pressure and serum creatinine are normal. A repeat urinalysis 3 weeks later confirms 18 RBC/HPF with no other abnormalities.

Clinical Case III

Which of the following is the most appropriate next step?

A. No further evaluation needed; recheck urinalysis in 1 year

B. Repeat urinalysis in 6 months only

C. Renal and bladder ultrasound plus cystoscopy

D. Cystoscopy plus CT urography

E. CT urography alone

Clinical Case III

Correct answer: C — Renal and bladder ultrasound plus cystoscopy (Intermediate Risk – only one criteria; could use urine cytology to defer cystoscopy)

A. **No further evaluation** - her RBC count is above the low-risk range (3–10/HPF)

B. **Repeat UA in 6 months only** - **low-risk** pathway; she doesn't qualify for it due to her RBC count (11–25/HPF)

D. Cystoscopy plus CT urography - **high-risk** pathway (reserved for >25 RBC/HPF, gross hematuria history, heavier smoking, or older age/male sex plus risk factors).

E. CT urography alone, no cystoscopy — incorrect on two counts: CT urography isn't the imaging of choice at the intermediate-risk tier tier.

Take Home Points

- Hematuria: Classify based on risk (choose wisely)
- Glomerular disease can be Ab-mediated (Lupus, ANCA, anti-GBM), abnormal Ig (IgA, MGRS – Amyloid/Cryo) or a systemic process (secondary MCD/FSGS)
- Low C3 + normal C4 → post-infectious GN or C3 glomerulopathy. Low C3 AND C4 → lupus or cryoglobulinemia.
- Catch-words: PLA2R = Membranous Nephropathy; Anti-nephrin = MCD/FSGS; Congo Red = Amyloid

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