NGN NCLEX 2026 • RENAL/UROLOGICAL SYSTEM • HIGH-YIELD REFERENCE

Glomerulonephritis

A spectrum of immune-mediated inflammatory disorders affecting the glomeruli — the kidney's filtering units. Classified by onset, cause, and histology. Mastery covers six clinical types, the classic triad, labs, priority interventions, and discharge teaching.

PRIORITY TOPIC | RENAL SYSTEM | ACUTE & CHRONIC | PEDIATRIC + ADULT | IMMUNE-MEDIATED

SOURCE TRANSPARENCY: This topic was not present in the uploaded reference flashcards. Content compiled from standard NCLEX references — *Saunders Comprehensive Review for the NCLEX-RN (9th ed.), ATI RN Adult Medical-Surgical Nursing (12th ed.), Lippincott Illustrated Reviews: Pharmacology (8th ed.), KDIGO 2024 Clinical Practice Guideline for Glomerular Diseases, and CDC group A strep guidance.* Aligned with the NGN NCLEX 2026 test plan.

SECTION 01

1 Definition & Overview

What it is

Inflammation of the **glomerular capillaries** — the tiny blood-filtering tufts inside the nephrons. Most cases are **immune-mediated**, triggered by antibody–antigen complexes that lodge in the glomerular basement membrane (GBM).

Why it matters

Damaged glomeruli leak protein and blood (**proteinuria + hematuria**) and lose filtration capacity, leading to **fluid overload, hypertension, and acute or chronic kidney disease**. Untreated, it can progress to end-stage renal disease (ESRD).

Hallmark triad

1. Hematuria (tea/cola-colored urine) | **2.** Edema (periorbital in kids, dependent in adults) | **3.** Hypertension

Two clinical patterns

Nephritic syndrome: hematuria + RBC casts + mild proteinuria + HTN (inflammation pattern). **Nephrotic syndrome:** massive proteinuria (>3.5 g/day) + edema + hypoalbuminemia + hyperlipidemia.

SECTION 02

2 Pathophysiology — Simplified

STEP 1 **Trigger:** Infection (group A strep), autoimmune disease (lupus, IgA), or anti-GBM antibody (Goodpasture) activates the immune system.

▼

STEP 2 **Antigen-antibody complexes** form in circulation and deposit in the glomerular basement membrane — OR antibodies attack the GBM directly.

▼

STEP 3 **Complement cascade activates** → inflammation, neutrophil infiltration, release of cytokines and proteolytic enzymes that damage the capillary walls.

▼

STEP 4 **Glomerular permeability ↑** → RBCs and proteins leak into Bowman's capsule → hematuria + proteinuria + RBC casts in urine.

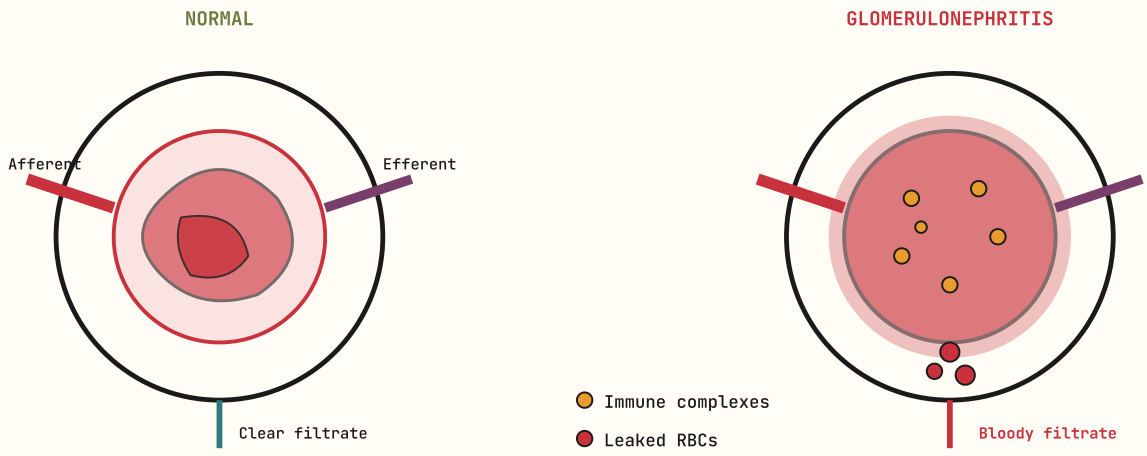
▼

STEP 5 **GFR drops** → sodium and water retention → fluid overload → hypertension, edema, oliguria, and rising BUN/creatinine.

▼

STEP 6 **If unresolved:** chronic inflammation → glomerular scarring (sclerosis) → progressive nephron loss → chronic kidney disease → end-stage renal disease.

NORMAL vs INFLAMED GLOMERULUS



SECTION 03

3 Six Clinical Types — Cause • Findings • Management • Teaching

1. Acute Post-Streptococcal GN (APSGN)

MOST NCLEX-TESTED | PEDIATRIC PEAK 5-12 YR

CAUSE	KEY FINDINGS	MANAGEMENT	TEACHING
Group A beta-hemolytic strep	Tea/cola-colored urine	Penicillin if strep still active	Excellent prognosis: >95% recover
Onset 1–3 wk AFTER pharyngitis or impetigo	Periorbital edema (AM)	Antihypertensives, diuretics	Treat strep throat early next time
Type III hypersensitivity (immune complex)	↑ ASO titer, ↓ C3 complement	Na + fluid restriction	Daily weights, BP at home
	RBC casts on UA	Bed rest until BP & UOP normal	Return for ↓UOP or worsening edema

2. IgA Nephropathy (Berger Disease)

MOST COMMON WORLDWIDE | YOUNG ADULTS

CAUSE	KEY FINDINGS	MANAGEMENT	TEACHING
IgA deposits in mesangium	Gross hematuria with URI	ACE-I or ARB (1st-line, proteinuria)	Chronic — lifelong monitoring
Triggered by URI or GI infection	Microscopic hematuria between flares	Corticosteroids if progressive	20–40% progress to ESRD over 20 yr
Hematuria 1–3 DAYS after infection (synpharyngitic)	Normal complement levels	SGLT2 inhibitors (KDIGO 2024)	BP control critical (<130/80)
	↑ Serum IgA in ~50%	Sparsentan (newer agent)	Avoid NSAIDs

3. Rapidly Progressive GN (RPGN / Crescentic)

EMERGENCY | DAYS → ESRD

CAUSE	KEY FINDINGS	MANAGEMENT	TEACHING
Severe glomerular injury → crescent formation	Acute kidney injury over WEEKS	High-dose IV methylprednisolone	Aggressive, time-sensitive treatment
3 types: anti-GBM, immune complex, pauci-immune (ANCA+)	Hematuria + RBC casts	Cyclophosphamide or rituximab	Infection precautions (immunosuppressed)
	Oliguria/anuria	Plasmapheresis (esp. anti-GBM)	Strict adherence to therapy
	ANCA+, anti-GBM+, low C3	Dialysis if needed	Lifelong nephrology follow-up

4. Goodpasture Syndrome (Anti-GBM Disease)

PULMONARY-RENAL | YOUNG MEN

CAUSE	KEY FINDINGS	MANAGEMENT	TEACHING
Autoantibodies vs type IV collagen in GBM and alveolar BM	Hemoptysis (lung bleeding)	PLASMAPHERESIS (priority)	Avoid smoking absolutely
Attacks kidneys AND lungs	Hematuria + AKI	Corticosteroids	Report hemoptysis IMMEDIATELY
	Dyspnea, cough	Cyclophosphamide	Infection precautions
	Anti-GBM antibody positive	O_2, transfusion if hemorrhage	Avoid hydrocarbons (paint, fumes)

5. Lupus Nephritis (SLE-Related GN)

AUTOIMMUNE | WOMEN 20-40

CAUSE	KEY FINDINGS	MANAGEMENT	TEACHING
SLE immune complexes deposit in glomerulus	Proteinuria ± hematuria	Hydroxychloroquine (all SLE)	Sun protection (UV triggers flares)
6 ISN/RPS classes (I–VI)	+ANA, +anti-dsDNA, ↓ C3 & C4	Corticosteroids + MMF or cyclophosphamide	Annual eye exam (hydroxychloroquine)
Classes III, IV, V most clinically significant	Butterfly rash, joint pain	Belimumab, voclosporin (newer)	Infection precautions
	Edema, HTN	ACE-I/ARB	Pregnancy planning critical

6. Chronic Glomerulonephritis

END-STAGE PATH | SLOW PROGRESSION

CAUSE	KEY FINDINGS	MANAGEMENT	TEACHING
Final common pathway of unresolved GN	Small, shrunken kidneys on imaging	BP control (ACE-I/ARB)	CKD diet: low Na, K, P, protein
Progressive glomerular sclerosis	↑ BUN/Cr, ↓ GFR	Phosphate binders, EPO	Fluid restriction

- Years of subclinical damage
- Anemia, bone disease
- Uremic symptoms late
- Vitamin D, iron
- Prepare for dialysis/transplant
- Avoid nephrotoxins (NSAIDs, contrast)
- AV fistula prep when GFR < 20

SECTION 04

4 Signs & Symptoms — Early vs Late

⏱ EARLY / Acute

- **Tea/cola-colored urine** (gross hematuria)
- **Periorbital edema** — esp. on waking
- Mild flank or back pain
- Decreased urine output (oliguria <400 mL/day)
- Headache, malaise, low-grade fever
- New-onset hypertension
- Weight gain from fluid retention
- Foamy urine (proteinuria)

⚠ LATE / Severe

- **Pulmonary edema** — crackles, dyspnea, SpO_2 ↓
- **Hypertensive encephalopathy** — seizure, ↓LOC
- Anasarca (generalized body edema)
- Anuria (<100 mL/day)
- Uremic symptoms — N/V, pruritus, asterixis
- Heart failure signs (S3, JVD)
- Hyperkalemia → cardiac dysrhythmia
- Goodpasture: **hemoptysis** 🩸

🚩 RED FLAG: **Cola-colored urine + new HTN + periorbital edema in a child 1–3 weeks post-sore throat = APSGN until proven otherwise. SEIZURE in a GN patient = hypertensive encephalopathy — call provider STAT.**

SECTION 05

5 Diagnostic Labs & Studies

URINALYSIS

RBC Casts

Pathognomonic for GN. Plus proteinuria (1–3+), dysmorphic RBCs.

ASO TITER

↑ Elevated

Confirms recent strep infection in APSGN. Peaks 3–5 weeks post-infection.

COMPLEMENT

C3 ↓ Low

Low in APSGN, lupus nephritis, MPGN. Normal in IgA nephropathy.

BUN / CREATININE

↑ Both

Reflects ↓GFR. BUN > 20 mg/dL, Cr > 1.3 mg/dL signal renal impairment.

EGFR

↓ Reduced

Stages CKD. <60 = CKD, <15 = ESRD requiring renal replacement.

24-HR PROTEIN

>150 mg

>3.5 g/day = nephrotic-range. Spot UPCR also acceptable.

ANTI-GBM

Positive

Confirms Goodpasture syndrome. Urgent workup if hemoptysis present.

ANCA

Positive

Pauci-immune RPGN (granulomatosis with polyangiitis, MPA).

RENAL BIOPSY

Gold Std

Definitive diagnosis & classification. Hold pressure 30+ min, bed rest 24 hr, monitor for hematuria.

SECTION 06

6 Priority Nursing Interventions — ABC Framework

A

AIRWAY / BREATHING

- Auscultate lungs Q4H — crackles = pulmonary edema
- Monitor SpO_2; HOB at 30–45°
- Goodpasture: assess for hemoptysis & SOB
- O_2 as ordered; suction equipment at bedside

B

BP & CIRCULATION

- BP Q4H; report SBP >140 or rapid rise
- Daily weights — SAME time, scale, clothes
- Strict I&O; report UOP <30 mL/hr or <0.5 mL/kg/hr
- Monitor for HF: S3, JVD, dependent edema

C

CHEMISTRY / SAFETY

- Monitor K^+ — hyperkalemia → cardiac arrhythmia
- Seizure precautions if BP severely elevated
- Infection prevention (immunosuppressed)
- Skin care — edematous tissue is fragile

NCLEX Priority Action Sequence

1. **Assess airway/breathing first** — pulmonary edema is the immediate life threat.
2. **Monitor BP and neuro status** — hypertensive encephalopathy can occur rapidly.
3. **Strict I&O + daily weight** — 1 kg weight = 1 L fluid. The single best fluid-balance indicator.
4. **Restrict fluids and sodium** per order; protein restriction only if BUN markedly elevated.
5. **Administer prescribed meds** — diuretics (loop), antihypertensives, antibiotics, immunosuppressants.
6. **Bed rest during acute phase** until BP and UOP normalize; then gradual activity.
7. **Prepare for renal biopsy** if ordered — informed consent, NPO, baseline coags, type & screen.
8. **Plan for dialysis** if AKI severe (K^+ >6.5, fluid overload unresponsive, uremia).

SECTION 07

7 Pharmacology — Drug Class • Action • Nursing Pearls

DRUG / CLASS	MECHANISM	KEY SIDE EFFECTS	NURSING CONSIDERATIONS
Furosemide (Lasix) — Loop diuretic	Inhibits Na/K/Cl reabsorption in loop of Henle → diuresis	Hypokalemia, ototoxicity, hypotension, dehydration	Monitor K^+, BP, UOP, weights. IV push slowly (≤20 mg/min) to avoid hearing loss.
Lisinopril / Enalapril — ACE inhibitor	Blocks angiotensin II → ↓BP & ↓ glomerular pressure → renoprotective	Dry cough, hyperkalemia, angioedema, ↑Cr early	1st-line for proteinuric GN. Hold if K^+ >5.5 or Cr rises >30%. Avoid in pregnancy.
Losartan / Valsartan — ARB	Blocks AT1 receptor — same goals as ACE-I, less cough	Hyperkalemia, dizziness, angioedema (rare)	Alternative if ACE-I cough. Same K^+ & Cr monitoring rules.
Penicillin V — Antibiotic	Eradicates residual group A strep in APSGN	Rash, anaphylaxis, GI upset	Check allergy. Does NOT reverse existing GN — prevents further antigen load.
Prednisone / Methylprednisolone — Corticosteroid	Suppresses immune-mediated inflammation	↑BG, infection risk, osteoporosis, mood change, weight gain, peptic ulcer	Give with food. Never stop abruptly (taper). Monitor BG. Calcium + vit D. Infection precautions.
Cyclophosphamide — Alkylating immunosuppressant	Suppresses B/T-cell proliferation in RPGN, lupus, Goodpasture	Hemorrhagic cystitis, bone marrow suppression, infertility, malignancy	Push fluids 2–3 L/day. Give MESNA & void Q2H. CBC monitoring. Birth control.
Mycophenolate mofetil (MMF) — Immunosuppressant	Inhibits lymphocyte purine synthesis (lupus nephritis induction & maintenance)	GI upset, leukopenia, infection	Take on empty stomach. Pregnancy CATEGORY D — 2 forms contraception. CBC monitoring.
Rituximab — Anti-CD20 monoclonal	Depletes B cells (ANCA vasculitis, refractory disease)	Infusion reactions, infection, reactivation of hepatitis B	Pre-medicate (acetaminophen, diphenhydramine, steroid). Slow IV infusion. Screen HBV.
Hydroxychloroquine — Antimalarial / DMARD	Foundational therapy for all SLE incl. lupus nephritis	Retinal toxicity, GI upset, rash	ANNUAL eye exam. Take with food. Notify provider of visual changes.
Calcium polystyrene / patiromer — K^+ binder	Binds K^+ in GI tract to treat hyperkalemia	Constipation, hypokalemia, edema	Used for ongoing hyperkalemia. Separate from other oral meds by 3 hr.

SECTION 08

8 NCLEX Test Traps — Wrong vs Right

✗ WRONG	✓ RIGHT
Child with APSGN. The MOST important nursing assessment is:	
~~Monitor for sore throat recurrence~~	**Daily weights and strict I&O**
	Weight is the BEST indicator of fluid balance during acute GN. 1 kg = 1 L.

✗ WRONG	✓ RIGHT
APSGN typically develops:	
~~During an active strep throat~~	**1–3 weeks AFTER strep infection**
	There is a latent period for immune complexes to form. Hematuria during active strep = think IgA nephropathy.

✗ WRONG	✓ RIGHT
The classic urinalysis finding in glomerulonephritis is:	
~~Bacteria and WBCs~~	**RBC casts and proteinuria**
	Bacteria + WBCs = UTI. RBC casts = glomerular inflammation.

✗ WRONG	✓ RIGHT
A patient with Goodpasture suddenly coughs up blood. The priority is:	
~~Administer a cough suppressant~~	**Assess airway, apply O_2, notify provider**
	Hemoptysis = pulmonary hemorrhage = ABC priority. Plasmapheresis is the definitive treatment.

✗ WRONG	✓ RIGHT
Which diet is appropriate during ACUTE GN with oliguria?	
~~High-protein, high-potassium~~	**Low-sodium, restricted fluids, moderate protein**
	Protein only severely restricted if BUN markedly elevated. K^+ restricted if hyperkalemic.

✗ WRONG	✓ RIGHT
A patient on lisinopril for IgA nephropathy. The MD adds spironolactone. The nurse should:	
~~Administer both as ordered~~	**Hold and clarify — risk of hyperkalemia**
	ACE-I + K-sparing diuretic = dangerous K^+ accumulation in renal patients.

✗ WRONG	✓ RIGHT
Patient is post-renal biopsy. Which finding requires immediate report?	
~~Pink-tinged urine 4 hours post-procedure~~	**Bright red urine with clots and ↓BP**
	Mild hematuria is expected post-biopsy. Frank bleeding + hemodynamic change = retroperitoneal hemorrhage.

✗ WRONG	✓ RIGHT
Patient on long-term prednisone for lupus nephritis. Which statement indicates need for more teaching?	
~~"I will take it with food every morning."~~	**"I'll stop the steroid when I feel better."**
	Abrupt steroid withdrawal causes adrenal crisis. Always taper.

SECTION 09

9 Patient & Family Education

Self-Monitoring

- ✓ Daily weights — same time, same scale, after voiding, before breakfast
- ✓ Report weight gain >2 lb/day or >5 lb/week
- ✓ Check BP at home; log readings
- ✓ Track urine output and color
- ✓ Recognize edema (rings tight? socks leaving deep marks?)

Diet

- ✓ Low-sodium: <2 g/day; read labels
- ✓ Fluid restriction per provider (often 1–1.5 L/day)
- ✓ Low potassium if hyperkalemic — avoid bananas, oranges, potatoes
- ✓ Moderate protein in acute phase; lower in CKD
- ✓ Eat small frequent meals if N/V from uremia

Medications

- ✓ Never stop steroids abruptly — adrenal crisis risk
- ✓ AVOID NSAIDs (ibuprofen, naproxen) — nephrotoxic
- ✓ Take ACE-I/ARB at same time daily; report dry cough or swelling
- ✓ Carry medication list; show every provider
- ✓ Annual eye exam if on hydroxychloroquine

Infection Prevention

- ✓ Treat sore throats early; complete antibiotic courses
- ✓ Annual flu vaccine; pneumonia & COVID per CDC
- ✓ Avoid crowds during immunosuppressive therapy
- ✓ Hand hygiene; report fever >100.4 °F immediately
- ✓ Dental hygiene; tell dentist about kidney disease

When to Call

- ✓ Decreased or absent urine output
- ✓ Worsening edema or shortness of breath
- ✓ Severe headache, confusion, vision changes
- ✓ Hemoptysis (Goodpasture patients)
- ✓ Fever, chills, flank pain

Long-Term Follow-Up

- ✓ Nephrology visits as scheduled — even when feeling well
- ✓ Lab work: BUN, Cr, eGFR, urinalysis, complement levels
- ✓ Smoking cessation — accelerates kidney damage
- ✓ Pregnancy planning — discuss with nephrologist
- ✓ Avoid IV contrast unless absolutely necessary

SECTION 10

10 Mnemonics & Memory Boosters

PHAROAH

- **P** Proteinuria
- **H** Hematuria (cola/tea urine)
- **A** Azotemia (↑BUN/Cr)
- **R** RBC casts
- **O** Oliguria
- **A** Antistreptolysin-O ↑
- **H** Hypertension

PEE BURST

Nephritic syndrome features:

- **P** Proteinuria (mild-mod)
- **E** Edema (periorbital)
- **E** Erythrocytes in urine
- **B** BP elevated
- **U** Urea & creatinine ↑
- **R** Reduced GFR
- **S** Sodium retained
- **T** Tea-colored urine

"1 to 3"

APSGN onset rule:

- 1–3 **WEEKS** after strep
- IgA: 1–3 **DAYS** after URI
- Same numbers, different units — different disease!

GOOD PASTURE

- **G** GBM antibodies
- **O** O_2 needs ↑
- **O** Oliguria
- **D** Dyspnea
- **P** Plasmapheresis
- **A** Anti-GBM titer
- **S** Steroids
- **T** Two organs (lung+kidney)
- **U** Urgent treatment
- **R** Renal failure
- **E** Eliminate smoking

SALT

Diet teaching:

- **S** Sodium restriction
- **A** Avoid NSAIDs
- **L** Limit fluids
- **T** Track weight & BP daily

"Berger Burgers"

IgA Nephropathy = Berger Disease. Imagine a burger with red juice dripping (hematuria) AFTER eating it — IgA hematuria appears WITH or right after a viral illness, not weeks later like APSGN.

11

SECTION 11 • NGN CLINICAL JUDGMENT

NCJMM 6-Step Scenario

STEP 1 — RECOGNIZE CUES

Recognize Cues

Scenario: A 9-year-old child arrives at the clinic with the parent. Parent reports, "His urine looks like Coke and his eyes have been puffy in the mornings for 3 days." History reveals strep pharyngitis treated 16 days ago with amoxicillin. **VS:** T 99.2°F, HR 96, RR 22, BP 142/90, SpO_2 97% RA. Weight up 1.4 kg from last visit. Periorbital edema 2+ bilaterally. Urinalysis: dark amber, 3+ blood, 2+ protein, RBC casts present. **Cues to note:** tea-colored urine, periorbital edema, hypertension (high for age — >95th percentile), recent strep infection 2–3 weeks prior, hematuria + proteinuria + RBC casts, weight gain.

STEP 2 — ANALYZE CUES

Analyze Cues

The combination of **cola-colored urine + periorbital edema + new hypertension + RBC casts** occurring 2–3 weeks after streptococcal pharyngitis fits the classic presentation of **Acute Post-Streptococcal Glomerulonephritis (APSGN)**. The latent period reflects the time required for antigen-antibody complexes to form and deposit in the glomerular basement membrane. Weight gain reflects sodium and water retention. UTI is ruled out — there are no WBCs or bacteria and the urine shows RBC casts (glomerular origin). IgA nephropathy is less likely because hematuria here developed weeks after, not 1–3 days after, the infection.

STEP 3 — PRIORITIZE HYPOTHESES

Prioritize Hypotheses

Most likely: APSGN with fluid overload and hypertension. **Greatest immediate risk:** hypertensive encephalopathy (seizure, stroke) and pulmonary edema if BP and fluid status are not controlled. **Differentials to rule out:** IgA nephropathy (timing differs), Henoch-Schönlein purpura nephritis (would expect purpuric rash and joint/abdominal pain), hereditary nephritis. Lab confirmation will include ASO titer (↑) and complement C3 (↓).

STEP 4 — GENERATE SOLUTIONS

Generate Solutions

Plan of care: (1) Admit for monitoring of BP, fluid status, and renal function. (2) Order serum BUN/Cr, electrolytes, ASO titer, complement levels, CBC. (3) Initiate strict I&O, daily weights, BP Q4H. (4) Fluid restriction and low-sodium diet. (5) Administer antihypertensive (e.g., nifedipine or hydralazine) and loop diuretic (furosemide) as ordered. (6) Penicillin if any residual strep activity. (7) Bed rest during acute phase. (8) Family teaching on home BP monitoring and follow-up.

STEP 5 — TAKE ACTION

Take Action

Place child on continuous BP monitoring and assess neuro status Q2H. **Administer furosemide IV** per order, monitoring UOP. Restrict fluids to calculated allowance; offer ice chips for thirst. Provide low-sodium, moderate-protein diet (e.g., unsalted toast, fresh fruit, lean chicken). Weigh daily on same scale, same time. Educate parents on the 1–3 week latent period and importance of early strep treatment in the future. Document baseline edema with photographs and measurements. Coordinate with nephrology if BP refractory.

STEP 6 — EVALUATE OUTCOMES

Evaluate Outcomes

Expected outcomes within 1–2 weeks: BP returns to age-appropriate range; periorbital edema resolves; UOP returns to baseline (≥1 mL/kg/hr); weight returns to pre-illness baseline; gross hematuria clears (microscopic hematuria may persist months). **Red flags requiring escalation:** persistent oliguria, BP unresponsive to antihypertensives, seizure activity, new dyspnea or crackles. **Long-term:** 95%+ pediatric APSGN patients fully recover. Schedule nephrology follow-up at 4–6 weeks with repeat UA and BMP. Educate that microscopic hematuria can linger up to 12 months without indicating relapse.