Nephritic Syndrome and Glomerulonephritis

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THE ONE-LINE TRUTH

In nephritic syndrome and glomerulonephritis, the team that catches the triad—blood in the urine (hematuria), high blood pressure (hypertension), and swelling (edema)—at the bedside wins before the biopsy returns.

The chain — how it unfolds

1. Immune trigger lands: infection (especially group A Streptococcus pharyngitis, but also staph, hepatitis B or C, HIV), autoimmune disease (lupus, IgA vasculitis, ANCA-associated vasculitis), or an unknown signal makes antibodies or immune complexes that target the glomerular basement membrane or get trapped there. — why: The glomeruli are innocent bystanders. Antibodies meant for bacteria or self-antigens cross-react with kidney tissue or circulate as clumps that lodge in the filter. Complement proteins (C3, C5b-9 membrane attack complex) activate, punching holes in endothelial cells and the basement membrane—the first break.
2. Inflammation floods the glomerulus: neutrophils and monocytes squeeze into capillary loops, the endothelium swells, and the basement membrane fragments. Red blood cells slip through the holes into Bowman's capsule and appear in urine (hematuria). The urine turns tea-colored or cola-colored, and the microscope shows red cell casts (clumps of red cells molded to the shape of the tubule). — why: Red cell casts are the smoking gun—they prove bleeding is happening inside the kidney (glomerulus or tubule), not from the bladder or urethra. The damaged filter also leaks some protein, though usually less than 3.5 g per day (the cutoff for nephrotic-range proteinuria).

3. Filtration surface area shrinks: swollen cells, deposited immune complexes, and fibrin clots clog capillary loops, cutting the effective filtering area. GFR drops, so creatinine and blood urea nitrogen (BUN) rise in the blood. Low urine output (Oliguria) (urine output below 400 mL per day) may appear because fewer glomeruli are working. — why: The kidneys can't clear waste and water as fast. Creatinine—a muscle breakdown product normally filtered and dumped—accumulates, signaling acute kidney injury. Patients feel tired, lose appetite, and may become confused if severe waste buildup in the blood (uremia) (waste buildup) is severe.
4. Salt and water pile up: damaged nephrons can't excrete sodium, so the body retains it; water follows sodium, expanding blood volume. Fluid leaks out of capillaries into the interstitial space (edema)—you see periorbital puffiness (especially in the morning), swollen ankles, and sometimes pulmonary edema (fluid flooding the lungs) (fluid in the lungs, causing shortness of breath). Blood pressure spikes because vessels are overfilled. — why: The kidneys sense low effective flow (because so many glomeruli are blocked) and activate the renin-angiotensin-aldosterone system, which holds even more sodium and water. This positive feedback loop—damage → retention → more pressure → more damage—drives high blood pressure (hypertension) and swelling (edema), the hallmarks of nephritic syndrome.
5. With high blood pressure (Hypertensive) injury compounds glomerular damage: high blood pressure batters vessel walls, thickening arterioles (arteriosclerosis) and scarring glomeruli (glomerulosclerosis). The left ventricle of the heart hypertrophies (thickens) to pump against high resistance, raising the risk of heart failure, stroke, and further kidney injury. — why: What started as an immune attack on the filter becomes a systemic vascular disease. Controlling blood pressure (with ACE inhibitors or angiotensin receptor blockers, which also reduce intraglomerular pressure) is critical to slow progression.
6. Resolution or scarring: if the immune trigger is removed (antibiotics for post-streptococcal glomerulonephritis, immunosuppression for lupus or vasculitis) and inflammation stops early, the glomeruli can heal—GFR recovers, blood in the urine (hematuria) clears, swelling (edema) and high blood pressure (hypertension) resolve. If inflammation persists for weeks, fibrosis replaces nephrons (chronic glomerulonephritis), GFR stays low, and the patient moves toward chronic kidney disease. — why: The kidneys cannot regenerate lost glomeruli reliably. Early aggressive treatment (steroids, cyclophosphamide, rituximab, or plasmapheresis, depending on the cause) stops new damage and gives surviving nephrons a chance to recover. Late treatment only manages complications—high blood pressure (hypertension), anemia, electrolyte shifts—and prepares for dialysis or transplant if GFR falls below 15 mL/min/1.73 m^2.

7. Complications of chronic disease: anemia (from low erythropoietin and chronic inflammation), secondary hyperparathyroidism (the damaged kidneys can't activate vitamin D, so calcium drops and parathyroid hormone rises, pulling calcium from bone), metabolic acidosis (acid building up in the blood) (kidneys can't dump acid), and severe waste buildup in the blood (uremia) (waste buildup causing nausea, confusion, pericarditis, and bleeding). — why: Once the kidneys lose reserve, every system they regulate starts to fail. The goal shifts from cure to preservation: slow further loss, treat complications, and plan renal replacement (dialysis or transplant) when GFR is critically low.
8. End-stage renal disease: if GFR falls below 15 mL/min/1.73 m^2 and symptoms (uremia, volume overload, refractory high potassium (hyperkalemia)) appear, the patient needs dialysis (hemodialysis or peritoneal dialysis) or kidney transplant to survive. — why: At this point the kidneys cannot sustain life. Dialysis does the job of the glomeruli—clearing waste, balancing electrolytes, removing extra water—but it is not a cure. Transplant offers the best long-term survival and quality of life, but the original disease (especially if autoimmune) can recur in the new kidney, so lifelong monitoring and immunosuppression are required.

Spot it — what you see, and why

- Dark or tea-colored urine (hematuria): Tiny blood vessels inside the kidney filters (glomeruli) become inflamed and leaky, so red blood cells squeeze through and color the urine brown or rust-red.
- Puffiness around the eyes and ankles (edema): The inflamed kidney filters cannot get rid of salt and water as they should, so extra fluid pools under the skin—first in the face (especially mornings) and then in the legs.
- Foamy or bubbly urine (proteinuria): Damaged filter walls let protein leak from the blood into the urine; the protein creates foam when you urinate, like soap bubbles.
- High blood pressure (hypertension): The kidneys hold on to extra salt and water, which increases the volume of blood in the arteries and raises pressure—like overfilling a garden hose.
- Feeling tired or weak (fatigue): The kidneys are not cleaning the blood well, so waste products build up (uremia); also, fluid overload and rising blood pressure make the heart work harder, draining energy.
- Reduced urine output (oliguria): Inflamed, swollen filters clog and slow the flow of blood being cleaned, so less urine is made each day—sometimes dropping below 400 milliliters (less than 2 cups).

Red flags — act now

- Shortness of breath or chest pain (may mean fluid is backing up into the lungs or the heart is struggling)
- Very little or no urine for 12 hours or more (may signal kidney shutdown)
- Severe headache, blurred vision, or seizure (may mean dangerously high blood pressure is hurting the brain)
- Sudden confusion or drowsiness (may mean waste products are building to toxic levels in the blood)

Treat it — in this order

1. Identify and treat the underlying cause (post-infectious glomerulonephritis, IgA nephropathy, lupus nephritis, ANCA-associated vasculitis) → Stopping the immune attack on the glomeruli halts further destruction of the kidney filters and allows healing to begin. For post-infectious glomerulonephritis, supportive care and sometimes antibiotics (if active infection remains) let the immune reaction calm down naturally. For autoimmune causes (lupus, ANCA vasculitis), immunosuppression (steroids, cyclophosphamide, rituximab) directly shuts down the cells attacking the kidney.
2. Control blood pressure aggressively, targeting <130/80 mmHg in adults and <90th percentile in children, using ACE inhibitors (lisinopril, enalapril) or ARBs (losartan, irbesartan) as first-line agents → ACE inhibitors and ARBs relax the efferent arteriole (the blood vessel leaving each glomerulus), which lowers pressure inside the filter and reduces how much protein and blood leak out. Lower glomerular pressure slows scarring (glomerulosclerosis) and protects remaining kidney function. These drugs also block angiotensin II and aldosterone, which drive fibrosis.
3. Restrict sodium intake to <2 grams per day and limit fluid intake; give loop diuretics (furosemide 20–80 mg daily, titrated to response) if swelling (edema) or high blood pressure (hypertension) persists → Reducing salt and water intake decreases the volume of fluid in blood vessels, which lowers blood pressure and reduces swelling. Loop diuretics block sodium reabsorption in the kidney tubules (specifically the loop of Henle), forcing the kidneys to dump salt and water into urine. This takes strain off the damaged glomeruli and prevents fluid from backing up into the lungs.

4. Immunosuppression (corticosteroids such as prednisone 1 mg/kg/day, often combined with cyclophosphamide, rituximab, or mycophenolate depending on cause and biopsy findings) → Corticosteroids dampen the entire immune response by blocking inflammatory signals in T cells and B cells. Cyclophosphamide and rituximab specifically kill or deactivate B cells that make the antibodies attacking the kidney. Mycophenolate blocks lymphocyte multiplication. Together, these drugs shut down the immune attack on the glomeruli and give the kidney a chance to heal. Benefit is greatest when started before irreversible scarring occurs.
5. Monitor and correct electrolyte imbalances (especially high potassium (hyperkalemia), too much acid in the blood (acidosis), and low calcium (hypocalcemia)) with dietary potassium restriction, sodium bicarbonate, calcium supplements, and phosphate binders as needed → Keeps serum potassium in the safe range (<5.5 mEq/L) to prevent life-threatening heart arrhythmias. Correcting too much acid in the blood (acidosis) (bringing bicarbonate above 22 mEq/L) protects bones and muscles and slows kidney disease progression. Calcium and phosphate management prevents bone disease (renal osteodystrophy) from chronic kidney dysfunction.

Your top priorities at the bedside

1. Measure blood pressure every 2 to 4 hours and notify provider immediately if systolic is above 160 or diastolic above 100 — why: High blood pressure (Hypertension) (high blood pressure) occurs in 80 to 90 percent of nephritic syndrome because damaged glomeruli (kidney filters) retain salt and water; uncontrolled high blood pressure causes stroke, heart failure, and further kidney damage
2. Monitor strict intake and output every shift, measuring all oral intake and urine output to the milliliter — why: Low urine output (Oliguria) signals declining glomerular filtration rate (kidney filtering ability) and predicts fluid overload; tracking daily fluid balance guides diuretic dosing and identifies patients needing urgent dialysis before pulmonary edema (fluid flooding the lungs) develops
3. Weigh the patient daily at the same time with the same scale and minimal clothing — why: Rapid weight gain of 2 pounds in one day or 5 pounds in one week indicates fluid retention from failing kidneys; each kilogram of weight gain represents approximately one liter of retained fluid requiring diuretic adjustment