

# Acute GN

Acute glomerulonephritis (GN) means the glomerulus—the kidney's tiny filter unit—is inflamed. Red blood cells and protein leak through holes in the damaged filter, turning urine dark brown or tea-colored.

## THE ONE-LINE TRUTH

The glomerulus (kidney filter) is under immune attack, and its leaky barrier is letting blood and protein spill into the urine.

## The chain — how it unfolds

1. Immune complexes or autoantibodies deposit in the glomerular capillaries or mesangium. — why: These deposits activate complement (C3, C5b-9 membrane attack complex), which punches holes in cell membranes, and recruit neutrophils and monocytes that release proteases, elastase, and reactive oxygen species—all of which chew through the basement membrane and podocyte foot processes.
2. Glomerular inflammation swells the capillary tuft, narrows the lumen, and damages the three-layer filter. — why: Endothelial cells swell and lose their fenestrations (small pores). The basement membrane becomes irregular, thickened, or split. Podocyte foot processes flatten and detach (effacement). The filter barrier is now leaky and clogged.
3. Red blood cells leak through the broken filter into the urine (hematuria). — why: Gaps in the basement membrane and missing podocyte bridges allow whole red cells to squeeze into Bowman space. As they travel down the acidic tubules, hemoglobin oxidizes, turning the urine dark brown, tea-colored, or cola-colored (gross blood in the urine (hematuria)). Under the microscope, you see dysmorphic RBCs (twisted, fragmented shapes from squeezing through damaged glomeruli) and RBC casts (clumps of red cells stuck in Tamm-Horsfall protein molds from the tubule), which prove the bleeding is glomerular, not from the bladder or urethra.
4. Albumin and other plasma proteins leak into the urine (proteinuria). — why: The damaged filter can no longer block large molecules. Proteinuria can range from mild (less than 1 g/day) to nephrotic-range (greater than 3.5 g/day). Severe protein loss drops serum albumin, reducing oncotic pressure (the force that pulls fluid back into capillaries), which worsens swelling (edema) and raises infection risk (immunoglobulins are lost in urine).

5. Glomerular filtration rate (GFR) falls as capillaries swell, collapse, or scar. — why: Fewer functioning glomeruli and smaller capillary surface area mean less blood is filtered per minute. The kidney cannot clear creatinine, urea, potassium, phosphate, and acid as fast as they are produced, so they accumulate in the blood.
6. Low urine output (Oliguria) (urine output drops below 400 mL per day). — why: Reduced GFR means less filtrate is made. Tubules also clog with RBC casts, cellular debris, and protein, raising back-pressure and further choking off urine flow. The kidneys hold onto salt and water to defend blood volume, cutting urine output even more.
7. Serum creatinine rises (normal ~0.7–1.2 mg/dL in adults; can climb to 2, 4, 8 mg/dL or higher). — why: Creatinine is a waste product from muscle breakdown, filtered only by the glomeruli and not reabsorbed. When GFR falls, creatinine clearance falls, so creatinine piles up in the blood. A doubling of creatinine means GFR has roughly halved. Rising creatinine is your bedside measure of worsening kidney function.
8. Salt and water retention trigger high blood pressure (hypertension) (high blood pressure) and swelling (edema) (swelling). — why: Falling GFR activates the renin-angiotensin-aldosterone system. Aldosterone tells the distal tubule to reabsorb sodium; water follows sodium, expanding blood volume. Higher volume raises blood pressure (often 140–180 systolic or more). Increased hydrostatic pressure (fluid-pushing force) in capillaries, plus low serum albumin (reduced oncotic pressure, fluid-pulling force), drives fluid into tissues—first around the eyes (periorbital swelling (edema), worst in the morning when lying flat) and ankles (pedal swelling), then sometimes the lungs (pulmonary swelling with trouble breathing (dyspnea) and crackles on exam) or abdomen (ascites).

## Spot it — what you see, and why

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- Cola-colored or tea-colored urine (blood in urine you can see): The immune system attacks the kidney's tiny filters and tears holes in them, so red blood cells slip through into the urine. As the cells sit in acidic urine, their iron-carrying protein (hemoglobin) rusts and turns the urine dark brown—like cola or tea.
- Puffy face and swollen ankles (edema): The damaged kidneys cannot get rid of salt and water, so both pile up in the blood and push fluid out into the tissues—especially around the eyes and feet where gravity and loose tissue make swelling easier. Also, protein leaks out in urine, so blood has less pulling power to keep fluid inside vessels.
- New high blood pressure or worsening of existing high blood pressure: Swollen, inflamed kidney filters cannot clean blood well, so the kidneys panic and hold onto extra salt and water to try to keep pressure up and keep filtering. But this extra volume raises blood pressure throughout the body.

- Much less urine than usual (oliguria—less than about 2 cups a day): The tiny filter units in the kidneys are swollen and clogged with debris from damaged cells, so fluid cannot pass through easily. Less fluid filtered means much less urine comes out.
- Feeling very tired and generally unwell (malaise): When kidneys fail to clean the blood, waste chemicals (creatinine, urea, acids) build up and poison every cell in the body, slowing down energy factories (mitochondria) and making you feel exhausted and sick.
- Dull ache in the back or sides (flank pain): The kidney swells rapidly from inflammation and fluid buildup, stretching the thin, tight capsule (outer shell) that wraps it. That capsule is packed with pain nerves, so stretching hurts.

## Red flags — act now

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- Coughing up blood along with blood in urine—warns of pulmonary-renal syndrome; needs urgent kidney biopsy and immune-suppressing drugs to save kidneys and lungs
- Kidney waste number (creatinine) rising fast (more than 0.3 in 48 hours or doubling in a week)—signals rapidly progressive GN; permanent kidney damage can happen in days without treatment
- Very little or no urine output (oliguria or no urine output (anuria))—means acute kidney injury; needs urgent kidney specialist and may need dialysis to clean the blood
- Uremic symptoms (confusion, pericardial rub, seizures)—show life-threatening toxin buildup from kidney failure; emergency dialysis may be needed
- Severe high blood pressure with headache, vision changes, or chest pain—with high blood pressure (hypertensive) emergency damaging brain, eyes, or heart; must lower pressure quickly in hospital

## Treat it — in this order

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1. ACE inhibitor (such as lisinopril) or ARB (such as losartan) to control blood pressure and reduce protein leak → Lowers pressure inside the glomeruli by relaxing the blood vessel leaving the filter. This reduces protein leakage and slows progression of chronic kidney disease. Per KDIGO 2021, target blood pressure < 120/80 mmHg if tolerated and proteinuria present.
2. Loop diuretics (such as furosemide) and salt restriction for volume overload → Removes excess salt and water that the inflamed kidneys cannot excrete. This lowers blood pressure, reduces swelling, and prevents fluid in the lungs that blocks oxygen exchange. Restrict sodium to < 2 grams per day.

3. Corticosteroids (methylprednisolone IV 500–1000 mg daily for 3 days, then oral prednisone 1 mg/kg/day) for immune suppression → Suppresses the immune system so it stops making antibodies and immune complexes that deposit in the glomeruli. This reduces inflammation, lets swelling resolve, and allows the glomerular filter to recover function.
4. Cyclophosphamide (oral 2 mg/kg/day or IV 0.5–1 g/m<sup>2</sup> monthly) or rituximab (375 mg/m<sup>2</sup> weekly for 4 doses or 1 g on days 1 and 15) for severe or rapidly progressive GN → Cyclophosphamide kills rapidly dividing immune cells. Rituximab wipes out B cells that make harmful antibodies. Both stop the immune attack at its source. Per 2021 KDIGO and 2021 ACR/Vasculitis Foundation guidelines, used in ANCA vasculitis, lupus nephritis, and other aggressive GN when kidney function is falling rapidly.
5. Plasmapheresis (plasma exchange, typically 7 sessions over 14 days, 60 mL/kg per session) for anti-GBM disease or severe ANCA-associated rapidly progressive GN → Physically removes circulating harmful antibodies and immune complexes from the blood before they deposit in glomeruli. Buys time for steroids and cyclophosphamide or rituximab to stop new antibody production. Per KDIGO 2021, plasmapheresis is standard for anti-GBM disease and considered in severe ANCA GN with dialysis-dependent AKI or pulmonary bleeding (hemorrhage).

## Your top priorities at the bedside

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1. Monitor urine output every hour using a Foley catheter or strict intake-and-output record, and report if less than 30 mL per hour (720 mL per day). — why: Low urine output (Oliguria) (low urine output) means the glomeruli are failing to filter waste and the patient is at immediate risk for high potassium (hyperkalemia) (high potassium that stops the heart), fluid overload with pulmonary edema (fluid flooding the lungs) (water in lungs), and severe waste buildup in the blood (uremia) (waste buildup causing confusion and seizures); hourly tracking catches rapid decline before life-threatening complications occur.
2. Check blood pressure every 4 hours and notify the provider immediately if systolic is above 160 or diastolic above 100. — why: Acute GN causes volume overload (too much fluid in blood vessels) and activates the renin-angiotensin system (hormone system that raises blood pressure); uncontrolled high blood pressure (hypertension) (high blood pressure) can trigger with high blood pressure (hypertensive) encephalopathy (brain swelling with seizures), heart failure, or stroke within hours.

3. Assess for signs of fluid overload every shift: listen to lung sounds for crackles, check for pitting swelling (edema) in ankles and sacrum, measure daily weight at the same time each morning. — why: Damaged glomeruli cannot excrete sodium and water, so fluid accumulates in tissues (edema) and lungs (pulmonary swelling (edema)); weight gain of more than 2 pounds in a day signals dangerous fluid retention, and lung crackles mean pulmonary edema (fluid flooding the lungs) requiring urgent diuretics and possible dialysis to prevent respiratory failure.