

Acute GN

A plain-English guide for you and your family

WHAT IS HAPPENING

Your kidneys hold millions of tiny filters called glomeruli. Each glomerulus is a tuft of delicate capillaries (tiny blood vessels) wrapped in a thin protein sheet called the basement membrane, with special guard cells (podocytes) standing watch. Their job is to filter your blood cleanly—letting water and waste pass through, but blocking red blood cells and big proteins so they stay in your bloodstream. When acute glomerulonephritis happens, your immune system mistakenly attacks these filters. Sometimes clumps of antibodies and germs (immune complexes) get stuck in the filter walls; other times antibodies directly target and damage the filter itself. This immune attack causes inflammation—swelling, irritation, and holes punched through the barrier—so red blood cells and protein leak into your urine, turning it dark brown or cola-colored. The inflamed, swollen glomeruli also squeeze the capillaries inside them, slowing the flow of blood being filtered. Less fluid is cleaned, so urine output drops and waste (like creatinine) piles up in your blood. Meanwhile, your kidneys hold onto salt and water to try to keep blood pressure up, but that backfires—fluid overflows into your tissues, puffing up your face (especially around your eyes in the morning) and your ankles. Blood pressure climbs. You feel tired, you see dark urine, and you notice swelling. All these signs—dark urine, puffiness, high blood pressure, rising creatinine—point back to one upstream problem: the glomerular filter is under immune attack and can no longer do its job.

What you might feel

- Cola-colored or tea-colored urine (blood in urine you can see)
- Puffy face and swollen ankles (edema)
- New high blood pressure or worsening of existing high blood pressure
- Much less urine than usual (oliguria—less than about 2 cups a day)
- Feeling very tired and generally unwell (malaise)
- Dull ache in the back or sides (flank pain)
- Foamy or bubbly urine
- Coughing up blood (hemoptysis) along with blood in urine (pulmonary-renal syndrome)

- Kidney waste number (creatinine) climbing fast—up by more than 0.3 in 48 hours or doubling in a week
- Confusion, nausea, or a scratchy sound when the heart beats (pericardial rub)—signs of severe waste buildup in the blood (uremia)

Call the doctor right away if...

- 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

What the care team will do

- 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).
- Check blood pressure every 4 hours and notify the provider immediately if systolic is above 160 or diastolic above 100.
- 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.
- Draw blood for creatinine and potassium levels as ordered (usually daily), and report creatinine rise of 0.3 mg/dL or more in 48 hours, or potassium above 5.5 mEq/L.
- Place the patient on a strict low-sodium diet (2 grams or less per day) and fluid restriction (usually 1 to 1.5 liters per day or 500 mL above previous day urine output).
- Monitor for signs of severe waste buildup in the blood (uremia) (waste buildup): confusion, drowsiness, nausea, metallic taste, or asterixis (flapping hand tremor when wrists are extended).
- Inspect urine color each void and test for blood and protein using dipstick; notify provider if urine turns darker or protein level increases.

- Educate the patient to avoid NSAIDs (ibuprofen, naproxen), nephrotoxic drugs (aminoglycoside antibiotics, contrast dye), and to report any new rash, fever, or worsening symptoms immediately.

Medicines you may be given — and why

ACE inhibitor (lisinopril, enalapril) or ARB (losartan, valsartan)

These drugs block a hormone called angiotensin II (a chemical that squeezes blood vessels tight). When you block this hormone, the tiny artery leaving each kidney filter relaxes and opens wider. That drops the pressure inside the filter—like opening a valve to reduce pressure in a pipe. Lower pressure means less protein is forced through the damaged filter wall into the urine, and the filter is less likely to scar over time. They do NOT fix the immune attack; they only protect filters that are still working from being crushed by high pressure.

Corticosteroid (prednisone, methylprednisolone)

Steroids are synthetic versions of cortisol (a hormone your adrenal glands make to calm inflammation). They flood into immune cells—especially T cells and macrophages—and turn OFF the genes that tell those cells to attack. This stops new immune cells from being called into the kidney and stops the ones already there from releasing chemicals that punch holes in the glomerular filter. Think of it as hitting the emergency brake on the immune attack. The damaged filters stop getting worse, and some can heal if the attack is stopped early enough.

Cyclophosphamide

Cyclophosphamide is a drug that sticks to the DNA (the instruction manual) inside dividing cells and scrambles it, so the cell cannot copy itself and dies. Immune cells that make antibodies (B cells) divide quickly, so this drug kills them off. Fewer B cells means fewer new antibodies attacking the kidney. It is used in severe, rapidly progressive GN—especially ANCA vasculitis and lupus nephritis—where you need to shut down antibody production fast to save the kidneys.

Rituximab

Rituximab is a laboratory-made antibody (a protein designed to stick to one target). Its target is CD20 (a marker on the surface of B cells). When rituximab docks onto CD20, it tags the B cell for destruction—either the immune system's natural killer cells eat it, or the cell's membrane ruptures. Within days, nearly all B cells in the blood and lymph tissue are gone. No B cells means no new antibodies attacking the kidney. It is used in ANCA vasculitis, lupus nephritis, and some cases of membranous nephropathy (a different type of glomerular disease). It is an alternative to cyclophosphamide—fewer harsh side effects, especially for people who want to preserve fertility.

Plasmapheresis (plasma exchange)

Plasmapheresis is a machine process that separates your blood into cells (red cells, white cells, platelets) and plasma (the liquid part that carries antibodies, clotting factors, and other proteins). The machine keeps your cells, throws away the plasma (which contains the harmful antibodies), and replaces it with clean donor plasma or albumin (a safe protein solution). This physically removes circulating antibodies—especially anti-GBM antibodies (in Goodpasture disease) and ANCA antibodies (in severe vasculitis). It is used when the antibody level is so high and the kidney damage so fast that you cannot wait for immunosuppressant drugs to work.

Antibiotics (penicillin or amoxicillin for group A Streptococcus)

Antibiotics are used **ONLY** if the infection that triggered the acute GN is still active. In post-streptococcal GN (the most common infection-triggered type), group A Streptococcus (the bacteria that causes strep throat) infects the throat or skin 1–3 weeks before kidney symptoms appear. By the time you see the kidney problem, the infection is usually gone, and the GN is caused by immune complexes (clumps of antibody stuck to leftover bacterial antigen) depositing in the kidney—**NOT** by live bacteria in the kidney. So you give antibiotics **ONLY** if the throat or skin culture is still positive or if the patient still has active strep symptoms (sore throat, fever, skin sores). The antibiotic kills the bacteria and stops more antigen from being made, but it does **NOT** treat the kidney damage itself—that heals on its own over weeks to months.

Dialysis (hemodialysis or peritoneal dialysis)

Dialysis is a machine (hemodialysis) or a process using the belly lining (peritoneal dialysis) that does the kidney's three main jobs: (1) removes waste products (urea, creatinine) that build up and cause confusion, nausea, and pericarditis (inflammation of the heart sac), (2) removes extra fluid that floods the lungs and causes shortness of breath, and (3) fixes blood chemistry—lowers potassium (which can stop the heart) and raises blood pH if it is too acidic. Dialysis does **NOT** heal the kidney—it buys time while you treat the immune attack with steroids or immunosuppressants. In post-strep GN, most patients recover kidney function and come off dialysis; in rapidly progressive GN (ANCA, anti-GBM, lupus), some need dialysis permanently if too many filters are destroyed before treatment works.

Where this information comes from

- KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases
- American Society of Nephrology (ASN)
- National Kidney Foundation (NKF)
- Cochrane systematic reviews on glomerulonephritis

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