Nephritic Syndrome and Glomerulonephritis

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THE STORY IN ONE SENTENCE

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“Nephritic Syndrome and Glomerulonephritis”

Normal anatomy — the body's job

Each kidney holds about one million glomeruli (tiny ball-shaped filters made of looped capillaries wrapped in a capsule). Blood arrives under pressure, water and waste squeeze through the filter wall into the capsule, and clean blood leaves. The filtered fluid (now called urine) flows down a tube to the bladder. At rest the kidneys have huge reserve—you can lose half your filters and still feel fine—so early trouble hides until stress (infection, salt load, or immune attack) eats into that reserve.

BODY'S JOB

The glomeruli are the kidney's front gate: they decide what stays in your blood (protein, blood cells, nutrients) and what leaves as waste (urea, creatinine, extra salt and water). By balancing pressure and choosing what passes, they keep your blood volume steady, your blood pressure in range, and your chemistry clean. Every heartbeat pushes about one liter of blood through both kidneys, so even a small filter problem adds up fast.

1. Blood under pressure enters the glomerulus (a tuft of capillaries inside a cup-shaped capsule called Bowman's capsule).
2. The capillary wall has three layers: endothelial cells (the inside lining), a basement membrane (a mesh in the middle), and podocytes (foot-shaped cells on the outside that wrap around the capillaries like fingers).

3. Filtered fluid (water, urea, creatinine, glucose, electrolytes) collects in Bowman's capsule and flows into the renal tubule.
4. Clean blood—still carrying protein, red cells, and white cells—leaves the glomerulus through the efferent arteriole and heads back to the heart.
5. The kidneys also release renin (a hormone) when blood pressure or volume drops, starting a cascade (the renin-angiotensin-aldosterone system) that raises pressure and holds onto salt and water.

What breaks

In nephritic syndrome and glomerulonephritis, the immune system attacks the glomerular capillaries and basement membrane. Inflammation punches holes in the filter wall, so red blood cells and protein leak into the urine. Swelling and scar tissue clog the capillaries, cutting blood flow and raising pressure upstream. The damaged kidneys can't clear salt and water fast enough, so fluid backs up into your tissues (causing swelling) and blood vessels (raising blood pressure). Every finding—cola-colored urine, puffy eyes, high blood pressure, rising creatinine—traces back to that one broken filter.

CLINICAL TERM

Glomerulonephritis is inflammation of the glomeruli, triggered by immune-complex deposition (antibodies and antigens clump on the filter), anti-glomerular basement membrane antibodies (antibodies directly attack the filter wall), or pauci-immune injury (white blood cells attack with few visible antibodies). The inflamed endothelium and basement membrane become leaky (hematuria and proteinuria), and proliferating cells and infiltrating neutrophils or monocytes crowd the capillary loops (reducing filtration surface area). Loss of filtration area drops the glomerular filtration rate (GFR), so creatinine rises. Salt and water retention activates the renin-angiotensin-aldosterone system, worsening high blood pressure (hypertension) and swelling (edema). If inflammation persists, fibrosis replaces functioning nephrons—irreversible chronic kidney disease.

How the body compensates

At first, undamaged glomeruli work overtime—they filter more per minute (hyperfiltration) to make up for the broken ones. Blood vessels in the kidney clamp down (vasoconstriction) to keep pressure high enough for filtration. The renin-angiotensin-aldosterone system turns on to hold salt and water, trying to keep blood volume and pressure steady. These moves buy time and can mask trouble for weeks, which is why early labs may look almost normal. But if inflammation drags on, hyperfiltration scars the remaining healthy filters, high pressure

damages vessel walls, and constant immune attack replaces kidney tissue with scar. The compensations themselves become the problem.

1. {'step': 'Uninjured glomeruli hyperfiltrate (each one cleans more blood per minute) to keep total GFR near normal.', 'why': 'This protects waste clearance in the short term, but chronic high flow and pressure inside those glomeruli stretch and scar them—eventually they burn out, and GFR falls further.'}
2. {'step': 'Afferent and efferent arterioles (the vessels feeding and draining each glomerulus) constrict to raise pressure inside the capillary tuft and maintain filtration.', 'why': 'Higher pressure squeezes more fluid through damaged filters, keeping urine output up. Over time, though, that pressure injures the capillary walls, worsening leakage and scarring.'}
3. {'step': 'The kidneys release renin, which converts angiotensinogen (a liver protein) to angiotensin I; an enzyme (ACE) then makes angiotensin II, a powerful vessel constrictor. Angiotensin II also triggers aldosterone (an adrenal hormone) release, which tells the kidneys to reabsorb sodium and water.', 'why': 'This system raises blood pressure and expands blood volume so the brain and heart get enough flow. But in nephritic syndrome, it overshoots: you end up with high blood pressure, swelling (edema), and even more strain on damaged filters.'}
4. {'step': 'The bone marrow ramps up red blood cell production (driven by erythropoietin from the kidneys) if blood loss into urine is heavy, and the liver makes more clotting proteins to try to seal leaks.', 'why': "These responses try to keep oxygen delivery and hemostasis normal, but they can't fix the underlying filter damage, so blood in the urine (hematuria) persists."}
5. {'step': 'If GFR drops low enough (below about 60 mL/min/1.73 m^2), the kidneys stop making enough erythropoietin, and you become anemic (low red blood cell count)—fatigue and shortness of breath appear.', 'why': 'Chronic inflammation and loss of nephrons shift the kidneys from compensation to failure; the reserve is gone, and every system depending on the kidneys starts to suffer.'}

Domino effect

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. — 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). — 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. — 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. — 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. — 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. — 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). — 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. — 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.

Symptoms · what the patient feels, 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).
- Shortness of breath (dyspnea): Excess fluid that the kidneys cannot remove backs up into the lungs (pulmonary swelling (edema)), making it harder to breathe—like trying to breathe through a wet sponge.
- Sudden weight gain: Retained salt and water add pounds quickly—sometimes 5 to 10 pounds in a few days—even though the person is not eating more.

Focused exam

- Blood pressure (both arms, sitting): Damaged kidney filters cannot flush salt and water, so fluid backs up inside the blood vessels and blood pressure climbs. High blood pressure (systolic ≥140 or diastolic ≥90 in adults, or above the 95th percentile in children) drives more scarring in the kidneys and raises the risk of heart and brain damage. Catching high blood pressure early tells you how urgently you need to lower the pressure and protect the kidneys.
- Swelling (Edema) (puffiness in the face, around the eyes, hands, legs, and ankles): When the kidneys cannot get rid of salt and water, fluid leaks from blood vessels into tissues and causes swelling. Facial swelling (periorbital swelling (edema)) is especially noticeable in the morning. Tracking where and how much swelling exists tells you how overloaded the body is and whether diuretics are working.
- Urine color (dark, tea-colored, or cola-colored): Red blood cells leaking through damaged glomeruli clump together and turn urine brown or tea-colored. This visible sign confirms active bleeding inside the kidney and points directly to glomerulonephritis rather than other kidney problems. The color change is often the first clue a patient or parent notices.
- Skin exam for recent infection signs (healing strep throat, impetigo sores, or other rashes): Many cases of glomerulonephritis start 1 to 3 weeks after a throat or skin infection (especially group A strep). Finding healing infection sites confirms post-infectious glomerulonephritis as the likely cause and guides whether you need antibiotics or just immune support. Lupus rashes (malar butterfly rash) or purple bleeding blotches (purpura) (small purple spots from bleeding under the skin) point to other immune causes like lupus nephritis or vasculitis.

- Lung sounds (crackles at the bases): Severe fluid overload from failing kidneys can push fluid into the lungs (pulmonary swelling (edema)) and make breathing hard. Crackles heard with a stethoscope at the bottom of the lungs signal that the patient is dangerously volume-overloaded and needs urgent diuretics or even dialysis.
- Heart sounds (S3 gallop or muffled heart sounds): An S3 gallop (an extra whoosh sound after the normal lub-dub) means the heart is overfilled with fluid and struggling to pump. Muffled heart sounds can signal fluid around the heart (pericardial a fluid collection (effusion)) from severe kidney failure or lupus. Either finding raises the urgency for aggressive fluid removal.

Diagnostics · every test answers a question

- Urinalysis with microscopy → Is there blood, protein, and casts in the urine that prove the glomeruli are inflamed and leaking?
- Serum creatinine and estimated glomerular filtration rate (eGFR) → How much kidney filtration ability has been lost, and is kidney function getting worse?
- Complement levels (C3 and C4) → Is the immune system using up complement proteins to attack the kidney, and which type of glomerulonephritis is this?
- Anti-streptolysin O (ASO) titer or anti-DNase B → Did a recent strep infection trigger the kidney inflammation?
- Antinuclear antibody (ANA) and anti-double-stranded DNA (anti-dsDNA) → Is lupus (systemic lupus erythematosus) attacking the kidneys?
- Antineutrophil cytoplasmic antibodies (ANCA: c-ANCA/PR3 and p-ANCA/MPO) → Is a small-vessel vasculitis (granulomatosis with polyangiitis, microscopic polyangiitis) causing the glomerulonephritis?
- Kidney biopsy (percutaneous needle biopsy with light, immunofluorescence, and electron microscopy) → What is the exact type and severity of glomerular damage, and how aggressively should we treat?

Treatment logic

- 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.

- 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.
- 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.
- 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.
- 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.
- Treat anemia with oral or IV iron (target ferritin >100 ng/mL, transferrin saturation >20%) and erythropoiesis-stimulating agents (epoetin alfa, darbepoetin) if hemoglobin drops below 10 g/dL → Restores red blood cell production and oxygen-carrying capacity, which reduces fatigue, improves exercise tolerance, and may slow heart strain. Anemia in chronic kidney disease happens because damaged kidneys make less erythropoietin (the hormone that tells bone marrow to make red cells), so replacing it helps.

- Initiate renal replacement therapy (hemodialysis or peritoneal dialysis) if severe severe waste buildup in the blood (uremia), refractory fluid overload, high potassium (hyperkalemia) >6.5 mEq/L unresponsive to medical therapy, or too much acid in the blood (acidosis) pH <7.1 develops → Dialysis mechanically removes toxins, excess fluid, and potassium that the failing kidneys cannot clear. It buys time for the kidneys to heal (in acute glomerulonephritis) or sustains life if chronic kidney disease has reached end-stage (eGFR <15 mL/min). Prevents death from pulmonary edema (fluid flooding the lungs), high potassium (hyperkalemia)-induced cardiac arrest, or uremic encephalopathy.

Medications

Prednisone or methylprednisolone (corticosteroid) · Glucocorticoid (steroid hormone)

Turns down the immune system's attack on the kidney filters (glomeruli). The immune system mistakenly attacks its own tissue in many forms of glomerulonephritis; steroids quiet that overactive response and reduce inflammation inside the tiny kidney filters so blood can be cleaned properly again.

1. The steroid enters immune cells and binds to a receptor inside the cell.
2. This tells the cell to stop making inflammatory chemicals (cytokines like TNF-alpha and IL-6).
3. Fewer inflammatory signals reach the glomeruli, so the attack on the kidney filters slows down.
4. Swelling drops, the filter pores become less leaky, and less protein and red blood cells spill into the urine.
5. Over weeks to months, the kidney's ability to filter waste improves.

SAFETY WATCH

High blood sugar (steroids make the body resist insulin, so check glucose regularly; people with diabetes need closer monitoring). · Weight gain and puffy face (steroids increase appetite and cause the body to hold salt and water). · Bone thinning (osteoporosis) with long-term use—doctors often add calcium, vitamin D, and sometimes a bone-protection drug (bisphosphonate) if steroids will be used for months. · Higher infection risk (the immune system is turned down, so infections like pneumonia or fungal infections can sneak in more easily). · Mood swings, trouble sleeping, or feeling jittery (steroids rev up the nervous system). · Stomach irritation or ulcers (take with food; some doctors add a stomach-protection drug like omeprazole). · Eye problems (cataracts and glaucoma) with prolonged use—regular eye checks are needed. · NEVER stop steroids suddenly after weeks of use; the dose must be tapered slowly or the adrenal glands (which make the body's own cortisol) may not restart properly, causing dangerous low blood pressure and weakness.

Cyclophosphamide · Alkylating agent (chemotherapy-type immune suppressant)

Kills or stops rapidly dividing immune cells (the cells launching the attack on the kidney). Used in severe glomerulonephritis (such as ANCA-associated vasculitis or lupus nephritis) when steroids alone are not enough. It is stronger than steroids and helps prevent the kidney from being destroyed by ongoing immune inflammation.

1. Cyclophosphamide is activated in the liver into a form that damages DNA.
2. It enters fast-growing cells (especially immune cells) and cross-links their DNA strands.
3. The cells cannot divide or make new proteins, so they die or stop multiplying.
4. Fewer immune cells are available to attack the kidney filters.
5. Inflammation in the glomeruli drops over weeks to months, and kidney function stabilizes or improves.

SAFETY WATCH

Low white blood cell count (makes infections much more dangerous—fever over 100.4°F requires urgent medical attention). · Bladder irritation and bleeding (hemorrhagic cystitis)—drink plenty of water and urinate frequently; a drug called mesna is often given to protect the bladder. · Infertility (can damage eggs and sperm; discuss egg or sperm banking before starting if future pregnancy is desired). · Nausea and vomiting (anti-nausea medicines are given with each dose). · Hair thinning or loss (temporary; hair grows back after treatment stops). · Increased cancer risk with long-term use (especially bladder cancer and blood cancers; risk is small but doctors weigh benefit vs. risk carefully). · Avoid pregnancy during treatment and for months after (the drug can harm a developing baby; reliable birth control is essential).

Mycophenolate mofetil (CellCept) or mycophenolic acid (Myfortic) · Inosine monophosphate dehydrogenase (IMPDH) inhibitor (immune suppressant)

Blocks a key enzyme (IMPDH) that immune cells (especially B-cells and T-cells) need to multiply. Because these cells rely heavily on this enzyme to make DNA building blocks, blocking it slows the immune attack on the kidney filters without hitting other fast-growing cells as hard as cyclophosphamide does.

1. Mycophenolate enters the cell and blocks the IMPDH enzyme.
2. Without IMPDH, B-cells and T-cells cannot make guanosine (a DNA building block).
3. These cells stop dividing and producing antibodies (proteins that mark the kidney for attack).
4. Fewer antibodies and immune cells reach the glomeruli.
5. Inflammation drops, and kidney function stabilizes over weeks to months.

SAFETY WATCH

Diarrhea, stomach cramps, and nausea (common; taking the drug with food helps, and the dose can be split or reduced). · Higher infection risk (especially viral infections like cytomegalovirus and herpes; doctors watch white blood cell counts). · Low white blood cell count (mild compared to cyclophosphamide, but still monitored with regular blood tests). · Avoid pregnancy (the drug can cause miscarriage and birth defects; women of childbearing age must use reliable birth control). · Slightly higher risk of skin cancers with long-term use—wear sunscreen and avoid tanning beds.

Azathioprine (Imuran) · Purine analog (immune suppressant)

Mimics a building block of DNA and RNA. When immune cells try to use it, their DNA gets scrambled and they cannot divide properly. This slows the production of new immune cells

attacking the kidney. It is an older drug, often used as a maintenance (long-term) therapy after stronger drugs like cyclophosphamide bring the disease under control.

1. Azathioprine is converted in the body into 6-mercaptopurine.
2. This fake DNA building block gets inserted into the DNA of dividing immune cells.
3. The DNA becomes unstable and the cells die or stop dividing.
4. Fewer new immune cells are available to attack the kidney.
5. Inflammation stays low, helping prevent flares of glomerulonephritis.

SAFETY WATCH

Low white blood cell count (regular blood tests—usually every month at first—are essential to catch this early). · Liver enzyme elevation (monitor liver function with blood tests). · Nausea and loss of appetite (taking the drug with food helps). · Higher infection risk (watch for fever, sore throat, or unusual tiredness). · Increased cancer risk with very long-term use (especially skin cancers and lymphomas; use sunscreen and get regular skin checks). · Drug interactions: allopurinol (a gout medicine) greatly increases azathioprine levels and can cause dangerous bone marrow suppression—these two drugs should NOT be combined without careful dose adjustment. · Genetic testing for TPMT enzyme activity is sometimes done before starting; people with low TPMT break down the drug slowly and need a lower dose to avoid toxicity.

Rituximab (Rituxan) · Monoclonal antibody (targeted immune suppressant)

A man-made antibody that latches onto a marker (CD20) found on the surface of B-cells (the immune cells that make antibodies). Once rituximab binds, the B-cells are marked for destruction and cleared away by the body's cleanup system. This is especially useful in glomerulonephritis driven by harmful antibodies (such as ANCA-associated vasculitis or membranous nephropathy).

1. Rituximab is given by IV infusion and circulates in the blood.
2. It finds and sticks to CD20 markers on B-cells.
3. The immune system's cleanup crew (complement proteins and natural killer cells) see the tagged B-cells and destroy them.
4. B-cell numbers drop to nearly zero within days to weeks.
5. Without B-cells, harmful antibodies stop being made, and the immune attack on the kidney fades.
6. B-cells slowly return over 6–12 months, but the disease often stays quiet during that time.

SAFETY WATCH

Infusion reactions (fever, chills, itching, low blood pressure during or right after the IV—pre-medications like acetaminophen and diphenhydramine are given to reduce this risk). · Higher infection risk, especially for unusual infections (such as reactivation of hepatitis B or a rare brain infection called PML)—doctors screen for hepatitis B before starting and monitor closely. · Low antibody levels for months (the body's ability to fight new infections is weakened; some people need extra vaccines or antibody infusions). · Very rare but serious: severe skin reactions (Stevens-Johnson syndrome) or lung problems (pneumonitis)—report new rash, trouble breathing, or chest pain immediately. · Avoid live vaccines (measles, chickenpox, yellow fever) during treatment and for months after.

ACE inhibitors (e.g. lisinopril, enalapril) or ARBs (e.g. losartan, valsartan) · Renin-angiotensin system blockers (blood pressure and kidney-protection drugs)

These drugs block angiotensin II (a hormone that squeezes blood vessels and raises blood pressure). In the kidney, angiotensin II especially squeezes the vessel leaving the glomerulus, which raises pressure inside the filter and forces more protein to leak out. By blocking angiotensin II, these drugs relax that outgoing vessel, lower the pressure inside the filter, and reduce protein spillage (proteinuria). They do NOT fix the immune attack, but they protect the kidney from further damage while other drugs work on the inflammation.

1. ACE inhibitors block the enzyme (ACE) that makes angiotensin II; ARBs block the receptor where angiotensin II would act—both paths reduce angiotensin II's effects.
2. With less angiotensin II, blood vessels relax and blood pressure drops.
3. The efferent arteriole (outflow vessel) relaxes more than the afferent arteriole (inflow vessel), so pressure inside the glomerulus drops.
4. Less pressure means less protein is forced through the filter into the urine.
5. Over weeks to months, proteinuria decreases and the risk of kidney scarring slows.

SAFETY WATCH

High potassium (hyperkalemia)—the drug reduces the kidney's ability to dump potassium, so blood levels must be checked regularly; avoid potassium supplements and salt substitutes (which contain potassium). · Rise in creatinine (a marker of kidney function) right after starting—a small rise (up to 30%) is expected and acceptable, but a big jump may mean the dose is too high or kidney blood flow is too low; the doctor will recheck labs in 1–2 weeks. · Dry cough (with ACE inhibitors only; if bothersome, switch to an ARB). · Dizziness or lightheadedness (blood pressure may drop too low, especially in the first few days—stand up slowly). · NEVER use during pregnancy (these drugs can harm or kill the developing baby; stop immediately if pregnancy is detected). · Rare but serious: angioedema (sudden swelling of the face, lips, tongue, or throat that can block breathing—go to the emergency room immediately if this happens).

Furosemide (Lasix) or other loop diuretics · Loop diuretic (water pill)

Blocks a transporter (the Na-K-2Cl cotransporter) in the loop of Henle (a hairpin turn deep inside each kidney tubule) so the kidney cannot pull salt and water back into the blood. Salt and water stay in the urine and leave the body, which shrinks swelling (edema) and lowers blood pressure. This does NOT fix the kidney disease itself—it only treats the symptom of fluid overload that often comes with nephritic syndrome.

1. Furosemide is filtered into the urine by the glomerulus.
2. It travels down the tubule and reaches the loop of Henle.
3. It blocks the Na-K-2Cl transporter on the tubule cells.
4. Salt (sodium and chloride) and water cannot be pulled back into the blood, so they stay in the urine.
5. Urine volume increases (often within 30–60 minutes of taking the pill), and swelling in the legs, lungs, and belly decreases over hours to days.

SAFETY WATCH

Low potassium (hypokalemia)—the kidney dumps potassium along with the extra salt and water; blood levels must be checked regularly, and a potassium supplement or potassium-sparing diuretic (like spironolactone) is often added. · Low sodium (hyponatremia) if too much water is lost. · Dehydration and dizziness (especially if the dose is too high or the person is not drinking enough). · Hearing problems (rare, usually only with very high IV doses or in people with severe kidney failure). · Gout flares (the drug can raise uric acid levels). · Frequent urination (plan bathroom access, especially in the first few hours after taking the pill).

Red flags

- 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)

Nursing priorities

1. Measure blood pressure every 2 to 4 hours and notify provider immediately if systolic is above 160 or diastolic above 100 — 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 — 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 — 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
4. Assess for swelling (edema) by checking periorbital areas in the morning and ankles and sacrum in the afternoon; grade pitting swelling 1+ to 4+ — Periorbital swelling (edema) (swelling around eyes) appears early because facial tissue has low pressure; tracking swelling location and severity shows whether salt and water restriction plus diuretics are working or if the patient needs escalation
5. Restrict sodium intake to less than 2 grams per day and limit fluids to 1 to 1.5 liters daily if low urine output (oliguria) is present — Damaged glomeruli cannot excrete sodium normally so dietary sodium directly increases blood volume, raises blood pressure, and worsens swelling (edema); fluid restriction prevents volume overload when kidneys cannot eliminate water

6. Inspect every urine sample for color and clarity, reporting tea-colored, cola-colored, or red urine immediately — Gross blood in the urine (hematuria) (visible blood in urine) indicates active glomerular inflammation; changes in urine color help track disease activity and response to immunosuppressive therapy in real time at the bedside
7. Assess respiratory rate, oxygen saturation, and lung sounds every 4 hours, watching for crackles or increased work of breathing — Pulmonary edema (fluid flooding the lungs) from fluid overload causes crackles (wet lung sounds) and low oxygen (hypoxia) (low oxygen); early detection allows diuretic escalation before respiratory failure requiring intubation and mechanical ventilation
8. Hold all nephrotoxic medications including NSAIDs, aminoglycosides, and contrast dye until provider reviews the medication list — Nephrotoxic agents (substances poisonous to kidneys) worsen acute kidney injury by reducing blood flow to already inflamed glomeruli; even single doses of ibuprofen can push compensating kidneys into failure requiring dialysis

Sources

- KDIGO Clinical Practice Guideline for Glomerulonephritis 2021
- UpToDate: Overview of the classification and treatment of rapidly progressive (crescentic) glomerulonephritis
- American Society of Nephrology
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)

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