

Nephrotic Syndrome

The kidney's filter broke and now protein leaks out, so the blood loses what it needs to hold fluid inside vessels.

THE STORY IN ONE SENTENCE

The kidney's filter broke and now protein leaks out, so the blood loses what it needs to hold fluid inside vessels.

“Nephrotic syndrome means the kidney filter is leaking protein into the urine. Every weird finding after that—swelling, high cholesterol, clots, infections—comes from the same upstream break.”

Normal anatomy — the body's job

The kidney filter—called the glomerulus—has three layers that work together like a fence: endothelial cells (the inner lining), the basement membrane (a mesh in the middle), and podocytes (cells with little feet on the outside). The podocyte feet fit together tightly and carry a negative electrical charge, so they block albumin (which is also negative and relatively large) from leaving the blood. Normally almost no albumin gets into the urine.

BODY'S JOB

Albumin is the most important protein in your blood. It acts like a sponge, holding fluid inside your blood vessels so you don't swell. It also carries hormones, medicines, and nutrients around your body. The glomerulus keeps albumin in the blood while letting water, salt, and waste pass through into the urine. If the filter works right, your urine has almost no protein.

1. Blood flows into the glomerulus (a tiny ball of capillaries in the kidney).
2. The three-layer filter—endothelium, basement membrane, and podocyte feet—blocks albumin and other big proteins because of size and negative charge.
3. Water, salt, urea, and other small waste molecules pass through into the tubule and become urine.
4. Clean blood with all its albumin flows back out into the body.

5. Result: blood albumin stays normal (around 3.5–5 g/dL) and no protein appears in the urine (under 150 mg per day).

What breaks

The podocytes—those cells with little feet that form the outer layer of the kidney filter—get damaged. The feet flatten, fuse together, or pull apart, so gaps open in the fence. Albumin and other proteins pour through into the urine. Blood albumin drops below 3 g/dL and urine protein rises above 3.5 grams per day (that's the definition of nephrotic-range proteinuria).

CLINICAL TERM

Nephrotic syndrome is defined by heavy proteinuria (≥ 3.5 g per day or urine protein-to-creatinine ratio ≥ 3 –3.5 mg/mg), hypoalbuminemia (serum albumin < 3 g/dL), swelling (edema), and hyperlipidemia. The primary glomerular diseases are minimal change disease (most common in children, podocyte foot-process effacement on electron microscopy but normal light microscopy), focal segmental glomerulosclerosis (FSGS, scarring in some glomeruli), membranous nephropathy (thickened basement membrane with subepithelial immune deposits), and membranoproliferative glomerulonephritis (MPGN, thick walls and mesangial expansion). Secondary causes include diabetic nephropathy (the most common cause in adults, from chronic high blood sugar (hyperglycemia) damaging the filter), systemic lupus erythematosus, amyloidosis, infections (hepatitis B and C, HIV, syphilis), drugs (NSAIDs, gold, penicillamine), and malignancy (especially solid tumors and Hodgkin lymphoma). All share the final common pathway: podocyte injury disrupts the glomerular filtration barrier.

How the body compensates

The body tries to restore blood volume and pressure by holding sodium and water (via the renin-angiotensin-aldosterone system and ADH), making more cholesterol and clotting factors in the liver, and shifting fluid around. Vital signs and kidney function can look stable at first, but the patient is on a knife edge—fluid overload, clots, infections, and acute kidney injury can appear suddenly.

1. Low albumin $\rightarrow$ low oncotic pressure $\rightarrow$ fluid leaks into tissues $\rightarrow$ the body senses low effective blood volume.
2. The kidney releases renin, triggering aldosterone and ADH release, which tells the kidney to hold onto sodium and water, trying to refill the vessels.
3. But because albumin is still low, the extra fluid just leaks out again into the tissues, worsening swelling (edema) (this is the underfill hypothesis).

4. The liver cranks up production of albumin, lipoproteins (LDL, VLDL), and clotting factors (fibrinogen, factors V and VIII) to try to thicken the blood and restore oncotic pressure.
5. Result: hyperlipidemia (total cholesterol often >300 mg/dL, LDL high, sometimes triglycerides high) and a hypercoagulable state.
6. Loss of antithrombin III, protein C, and protein S in the urine plus higher clotting-factor synthesis tips the balance toward clot formation—renal vein clot formation (thrombosis) and pulmonary embolism (a clot lodging in a lung artery) are real risks.
7. Loss of immunoglobulins (IgG) in the urine weakens opsonization, so encapsulated bacteria (pneumococcus, H. influenzae) cause serious infections, especially peritonitis and pneumonia in children.
8. If diuretics are pushed too hard to treat swelling (edema), intravascular volume can drop sharply → prerenal acute kidney injury and shock.
9. Watch the trend, not the snapshot: stable blood pressure and creatinine today do not mean safe—look for rising weight, worsening swelling (edema), sudden leg pain or chest pain (clot), fever (infection), or rising creatinine (AKI).
10. The dangerous middle is when compensation is holding but about to fail—catch it early by monitoring daily weights, urine output, albumin, and signs of clot or infection.

Domino effect

1. Podocyte damage opens gaps in the glomerular filter. — The podocytes (foot cells on the outside of the glomerular capillary) lose their tight foot-process structure and negative charge. Gaps appear, and the barrier that normally blocks albumin from leaving the blood is gone.
2. Albumin pours into the urine (proteinuria ≥ 3.5 g/day). — Albumin is negatively charged and normally repelled by the podocyte charge barrier. Once that barrier breaks, albumin floods through into the urine faster than the liver can replace it.
3. Serum albumin drops below 3 g/dL (hypoalbuminemia). — You lose more albumin in the urine every day than your liver can make, so blood albumin level falls. Albumin is the main protein holding fluid inside your vessels (oncotic pressure), so oncotic pressure drops.
4. Fluid leaks out of blood vessels into tissues → swelling (edema). — With low oncotic pressure, the balance between hydrostatic pressure (pushing fluid out) and oncotic pressure (pulling fluid in) tips outward. Fluid shifts into the interstitial space, causing puffiness around the eyes (periorbital swelling (edema), worst in the morning), swollen legs and ankles, fluid buildup in the belly (ascites) (fluid in the belly), and sometimes pleural a fluid collection (effusion) (fluid around the lungs).

5. The liver makes more cholesterol and lipoproteins → hyperlipidemia. — The liver senses low albumin and tries to help by ramping up synthesis of all proteins, including lipoproteins (VLDL, LDL). Total cholesterol often goes above 300 mg/dL. You may see lipiduria—oval fat bodies and fatty casts in the urine under the microscope.
6. Antithrombin III, protein C, and protein S leak out → hypercoagulable state. — These natural anticoagulant proteins are small enough to leak through the damaged filter. At the same time, the liver makes more clotting factors (fibrinogen, factors V, VIII). The balance tips toward clotting. Renal vein clot formation (thrombosis) (sudden flank pain, blood in the urine (hematuria), worsening kidney function) and pulmonary embolism (a clot lodging in a lung artery) (chest pain, shortness of breath) are serious risks.
7. Immunoglobulins (IgG) leak out → increased infection risk. — Antibodies that normally coat bacteria (opsonize them) are lost in the urine. Encapsulated bacteria—especially *Streptococcus pneumoniae*—become dangerous. Children with nephrotic syndrome are at high risk for spontaneous bacterial peritonitis and pneumonia.
8. Every symptom you see comes from one upstream break: podocyte injury causing low albumin. — Trace it backward: foamy urine, swelling, high cholesterol, clot risk, infection risk—all point to the same source. Fix the filter (treat the underlying cause—steroids for minimal change, control sugar in diabetes, stop the offending drug) and the dominoes reverse. The body was never broken randomly; every sign is the emergency response to losing albumin.

Symptoms · what the patient feels, and why

- Swelling around the eyes and in the legs (edema): The kidneys leak huge amounts of albumin (the main protein that keeps water inside blood vessels) into the urine, so water seeps out of the blood and pools in the tissue under the skin, especially in loose areas like the eyelids and ankles where gravity or tissue stretch let fluid collect easily.
- Foamy or frothy urine: Protein spilling into the urine acts like soap — it traps air bubbles when you pee, making the toilet water look foamy or sudsy, just like shaking a bottle of dish soap and water.
- Weight gain (often several pounds in a few days): Fluid is building up in the body's tissues faster than you can pee it out, so the extra water weight shows up on the scale before you even notice visible swelling.
- Fatigue and low energy: Losing albumin drains the fuel your cells need to work well, and carrying extra fluid weight is like hauling around heavy bags all day — it wears you out.
- Loss of appetite or belly fullness: Fluid can leak into the lining of the belly (ascites), pressing on the stomach and intestines so you feel stuffed even when you haven't eaten much.

- High cholesterol and fats in the blood: The liver tries to make up for lost albumin by ramping up all protein production, but it accidentally also churns out extra cholesterol and triglycerides (fats), flooding the bloodstream with them.
- Infections happen more often (especially pneumonia or skin infections): Albumin carries antibodies (infection-fighting proteins), so when you lose albumin in the urine you also lose some of your immune defense; plus, swollen tissue is easier for bacteria to invade.
- Blood clots in legs or lungs (deep vein clot formation (thrombosis) or pulmonary embolism (a clot lodging in a lung artery)): The kidneys leak proteins that normally prevent clotting (like antithrombin), and the liver overproduces clotting proteins to compensate, tipping the balance so blood clots too easily inside veins.

Focused exam

- Pitting swelling (edema) around the eyes and legs: When you press your finger into the swollen skin and it leaves a dent that stays for a few seconds, that is pitting swelling (edema). It happens because albumin (the main protein that keeps water inside blood vessels) is low, so water leaks out into the tissues. This tells you the kidney is losing a lot of protein.
- Foamy urine in the toilet bowl: Foamy or frothy urine means protein is pouring out through the urine. The kidney's filter (the glomerulus) is damaged and letting albumin escape. This is the signature sign of nephrotic syndrome and confirms the protein leak is real.
- Weight gain of several pounds over days or a week without eating more: The body is holding onto salt and water because albumin dropped. The weight goes up from fluid building up in tissues, not from fat. Tracking weight helps you see how bad the leak is and whether treatment is working.
- Pale skin, poor appetite, or feeling very tired: Low albumin means the blood cannot carry nutrients and building blocks well. The patient feels weak and sick because the body is losing its fuel. This tells you the kidney problem is affecting the whole body.
- Swollen belly (ascites) or shortness of breath: Fluid can build up in the belly or the lungs (pulmonary swelling (edema)) when albumin is very low. If the patient is short of breath or breathing fast, fluid may be pressing on the lungs. This is a danger sign that means the swelling is severe and the patient needs urgent help.
- Warm, swollen calf or sudden chest pain with shortness of breath: Nephrotic syndrome makes the blood clot more easily because the kidney loses antithrombin (a protein that stops clots) in the urine. A clot in a leg vein (deep vein clot formation (thrombosis)) or in the lung (pulmonary a traveling clot lodging in a vessel (embolism)) is life-threatening and needs emergency treatment.

Diagnostics · every test answers a question

- Urine protein-to-creatinine ratio (spot urine) or 24-hour urine protein → Is the kidney leaking protein, and how much protein is escaping?
- Serum albumin → How low is the albumin in the blood, and is it low enough to cause swelling?
- Serum cholesterol and triglycerides (lipid panel) → Is the liver making extra cholesterol in response to low albumin?
- Serum creatinine and estimated glomerular filtration rate (eGFR) → Is the kidney still filtering waste well, or is kidney function dropping?
- Urinalysis with microscopy → What else is in the urine, and are there clues about the cause?
- Kidney biopsy (if cause is unclear, patient does not respond to steroids, or patient is an adult) → What is the exact disease damaging the kidney filter?
- Complement levels (C3, C4) and autoantibody panel (ANA, anti-dsDNA, hepatitis B and C, HIV) → Is an infection or autoimmune disease causing the kidney damage?

Treatment logic

- ACE inhibitor (e.g., lisinopril) or ARB (e.g., losartan) → Relaxes the blood vessels inside the kidney filter (the glomerulus) and lowers the pressure squeezing protein out through the damaged podocytes (the filter cells). This slows the protein leak and protects the kidney from further damage.
- Loop diuretic (e.g., furosemide) plus low-sodium diet (under 2 grams of sodium per day) → Pulls extra water out through the kidneys so swelling in the legs, face, and lungs goes down and breathing gets easier. Limiting salt stops the body from holding onto more fluid.
- Disease-specific therapy: corticosteroids (e.g., prednisone) for minimal change disease, immunosuppressants (e.g., cyclosporine, tacrolimus, rituximab) for FSGS or membranous nephropathy, or tight diabetes and blood pressure control for diabetic nephropathy → Targets the actual cause damaging the podocytes or the immune attack destroying the filter, so protein stops leaking and albumin levels rise back toward normal.
- Statin (e.g., atorvastatin or simvastatin) → Lowers the high cholesterol that builds up when the liver over-produces it to try to replace lost albumin. This reduces the long-term risk of heart disease and blood clots.
- Anticoagulation (warfarin or low-molecular-weight heparin such as enoxaparin) if albumin is less than 2.0 to 2.5 g/dL or a clot is found → Prevents new blood clots from forming in veins and arteries because the kidney is losing antithrombin (a protein that stops clots) in the urine, making the blood sticky.

- Albumin infusion (intravenous) in severe, acute hypoalbuminemia (albumin less than 2.0 g/dL) with life-threatening swelling or low blood pressure → Temporarily puts albumin back into the bloodstream to restore oncotic pressure (the pull that keeps water inside vessels) and reduce severe swelling, fluid in the lungs, or organ dysfunction.

Medications

Lisinopril or enalapril (ACE inhibitor) · Angiotensin-converting enzyme (ACE) inhibitor

Blocks the body from making angiotensin II (a chemical that squeezes blood vessels tight). When blood vessels in the kidney stay more relaxed, the pressure inside the tiny kidney filters (glomeruli) drops. Lower pressure means the filters leak less protein into the urine.

1. The drug blocks ACE, an enzyme that normally turns angiotensin I into angiotensin II.
2. With less angiotensin II, the small artery leaving each kidney filter (efferent arteriole) relaxes and widens.
3. The pressure inside the filter drops.
4. Less protein is forced through the filter wall into the urine.

SAFETY WATCH

Can raise potassium — check blood potassium regularly, especially if the person also takes a diuretic that saves potassium or has kidney disease that is getting worse. · Can cause a dry cough in some people (about 10–20%). If the cough is bothersome, switch to an ARB. · Do not use during pregnancy — can harm the baby's kidneys and lower amniotic fluid. · Watch kidney function (creatinine) closely in the first few weeks. A small rise is okay; a jump over 30% means stop and talk to the doctor. · Rarely causes angioedema (sudden swelling of the face, lips, tongue, or throat). If this happens, stop the drug immediately and call 911.

Losartan or valsartan (ARB) · Angiotensin II receptor blocker (ARB)

Blocks the receptor (the lock on the cell) where angiotensin II normally plugs in. This stops angiotensin II from squeezing blood vessels. The result is the same as an ACE inhibitor: lower pressure inside the kidney filters and less protein leaking into urine.

1. The drug sits on the AT1 receptor (the spot where angiotensin II usually acts) and blocks it.
2. Angiotensin II can no longer squeeze the blood vessels tight.
3. The small artery leaving each kidney filter (efferent arteriole) relaxes and widens.
4. The pressure inside the filter drops.

5. Less protein is forced through the filter wall into the urine.

SAFETY WATCH

Can raise potassium — check blood potassium regularly. · Do not use during pregnancy — can harm the baby's kidneys. · Watch kidney function (creatinine) closely in the first few weeks. A jump over 30% means stop and talk to the doctor. · Does not usually cause a cough (unlike ACE inhibitors), so it is a good choice if cough was a problem. · Rarely causes angioedema, but less often than ACE inhibitors.

Furosemide (loop diuretic) · Loop diuretic

Blocks a pump in the kidneys that normally pulls sodium and water back into the blood. When that pump is turned off, sodium and water stay in the urine and leave the body. This shrinks the swelling (edema) in the legs, belly, and lungs.

1. The drug blocks the NKCC2 transporter (a pump that moves sodium, potassium, and chloride out of the urine and back into the blood).
2. Sodium and water stay in the urine tube and flow out of the body.
3. The blood volume shrinks a bit, and fluid that was stuck in tissues (swelling) gets pulled back into the blood and then out through urine.
4. Swelling in the legs, belly, and lungs goes down.

SAFETY WATCH

Can lower potassium — check blood potassium regularly and add potassium-rich foods or a supplement if needed. · Can lower sodium and magnesium — watch for muscle cramps, weakness, or confusion. · Can raise blood sugar a little, especially in people with diabetes. · Can cause dehydration if the dose is too high or if the person does not drink enough. Watch for dizziness, dry mouth, and dark urine. · Can damage hearing if given in very high doses intravenously (IV) too fast, especially with other drugs that hurt the ears (like aminoglycoside antibiotics). · Does not work well if kidney function is very low (GFR under 20–30). May need a higher dose or switch to a different approach.

Atorvastatin or simvastatin (statin) · HMG-CoA reductase inhibitor (statin)

Blocks HMG-CoA reductase, the enzyme in the liver that makes cholesterol. With less cholesterol being made, the liver pulls more cholesterol out of the blood to use. This lowers LDL (bad cholesterol) and may slow damage to blood vessels and kidneys.

1. The drug blocks HMG-CoA reductase, the enzyme that runs the first big step in making cholesterol.
2. The liver makes less cholesterol.

3. The liver puts more LDL receptors (docking stations for cholesterol) on its surface to grab cholesterol from the blood.
4. LDL cholesterol in the blood drops.
5. Over time, this may protect the heart and kidneys from damage.

SAFETY WATCH

Can cause muscle pain or weakness (myopathy). If pain is severe or if urine turns dark (a sign of muscle breakdown called rhabdomyolysis), stop the drug and call the doctor immediately. · Check liver enzymes (ALT, AST) before starting and a few months later. A mild rise is common; a jump to more than three times normal means stop the drug. · Can raise blood sugar slightly — monitor in people with diabetes or prediabetes. · Avoid grapefruit juice with some statins (especially simvastatin and atorvastatin) — it raises the drug level and increases the risk of muscle problems. · Safe during most of life but do not use during pregnancy or breastfeeding.

Warfarin or low-molecular-weight heparin (anticoagulant) · Anticoagulant (blood thinner)

Slows the clotting cascade (the chain reaction that forms a blood clot). People with nephrotic syndrome lose proteins that normally prevent clots (antithrombin III and proteins C and S) in their urine, so their blood clots more easily. A blood thinner shifts the balance back and lowers the risk of a dangerous clot in a leg vein (DVT) or lung (pulmonary a traveling clot lodging in a vessel (embolism)).

1. Warfarin blocks vitamin K epoxide reductase, an enzyme that recycles vitamin K. Without enough active vitamin K, the liver cannot finish making several clotting factors.
2. Heparin binds to antithrombin III and makes it work much faster. Antithrombin III then shuts down thrombin and factor Xa (two key players in clot formation).
3. Either way, the blood takes longer to clot.
4. The risk of a new clot forming in a vein drops.

SAFETY WATCH

Bleeding is the main risk. Watch for unusual bruising, bleeding gums, blood in urine or stool, or coughing up blood. Call the doctor right away if any of these happen. · Warfarin requires regular blood tests (INR) to make sure the dose is right. The target INR is usually 2–3. Too low means not enough protection; too high means higher bleeding risk. · Many foods (especially leafy greens high in vitamin K) and drugs interact with warfarin. Keep vitamin K intake steady — do not suddenly start or stop eating a lot of greens. · Low-molecular-weight heparin (like enoxaparin) does not need daily blood tests but is given as a shot under the skin once or twice a day. · Do not use in active bleeding, recent surgery on the brain or spine, or very low platelets.

Prednisone or other corticosteroid · Corticosteroid (immune suppressant)

Turns down the immune system's attack on the kidney filters. In some types of nephrotic syndrome (especially minimal change disease), the immune system mistakenly damages the glomeruli. Prednisone quiets that immune response, lets the filters heal, and stops protein from leaking.

1. The drug enters immune cells and binds to a receptor inside.
2. It travels to the nucleus (the control center of the cell) and changes which genes are turned on or off.
3. Fewer inflammatory chemicals (cytokines like IL-2, TNF-alpha, and interferon-gamma) are made.
4. The immune attack on the kidney filters quiets down.
5. The filters begin to heal, and protein leak drops.

SAFETY WATCH

Raises blood sugar — check regularly, especially in people with diabetes or prediabetes. · Raises blood pressure — monitor at home and at every visit. · Increases appetite and can cause weight gain, especially in the face (moon face) and upper back (buffalo hump). · Weakens bones over time (osteoporosis) — add calcium (1200 mg/day) and vitamin D (800–1000 IU/day). Consider a bone-density scan if used for more than three months. · Increases infection risk because the immune system is turned down. Avoid live vaccines while on high doses. Watch for fever, cough, or any sign of infection. · Can cause mood swings, trouble sleeping, or (rarely) severe mental changes. If mood or behavior changes suddenly, call the doctor. · Never stop suddenly after more than a couple of weeks — taper the dose slowly to let the adrenal glands wake back up.

Cyclophosphamide (in steroid-resistant or relapsing cases) · Alkylating agent (immune suppressant and chemotherapy drug)

Damages the DNA inside fast-growing immune cells (especially B cells and T cells) so they cannot multiply. This shuts down the immune attack on the kidney filters in steroid-resistant or frequently relapsing nephrotic syndrome (for example, in some cases of minimal change disease or membranous nephropathy).

1. The drug enters cells and adds chemical groups (alkyl groups) onto DNA strands.
2. The DNA strands cannot unzip and copy themselves correctly.
3. Fast-dividing cells (like B cells and T cells) die or stop multiplying.
4. The immune attack on the kidney filters weakens.
5. Protein leak slows or stops.

SAFETY WATCH

Lowers white blood cells, red blood cells, and platelets — check blood counts regularly. Increases infection risk. · Can cause nausea and vomiting — anti-nausea medicine is often given at the same time. · Can damage the bladder and cause blood in the urine (hemorrhagic cystitis). Drink plenty of water and urinate often. Mesna (a protective drug) is sometimes given with high doses. · Can harm fertility in both men and women. Discuss sperm or egg banking before starting if future pregnancy is desired. · Increases the risk of certain cancers (especially bladder cancer and blood cancers) years later. Use only when benefit outweighs risk. · Do not use during pregnancy — can cause severe birth defects.

Rituximab (in certain resistant or relapsing cases) · Monoclonal antibody (B-cell-depleting agent)

Targets and destroys B cells (a type of immune cell) by locking onto CD20, a protein on their surface. In some types of nephrotic syndrome (especially steroid-dependent minimal change disease or membranous nephropathy), B cells make antibodies or release chemicals that damage the kidney filters. Removing B cells stops that attack and lets the filters heal.

1. The drug binds to CD20, a marker on the surface of B cells.
2. The immune system recognizes the drug as a flag and destroys the tagged B cells.
3. B-cell numbers drop sharply within days to weeks.
4. The immune attack on the glomeruli quiets.
5. Protein leak slows or stops, often allowing a reduction or stop of steroids.

SAFETY WATCH

Increases infection risk, especially viral infections, because B cells are gone for months. Screen for hepatitis B before starting — the virus can reactivate and cause severe liver damage. · Can cause infusion reactions (fever, chills, rash, low blood pressure) during or right after the IV infusion. Pre-medication with acetaminophen and an antihistamine is standard. · Rarely causes progressive multifocal leukoencephalopathy (PML), a serious brain infection. Watch for new confusion, weakness, vision changes, or trouble speaking. · Check immunoglobulin levels over time — some people develop low antibody levels and may need immunoglobulin replacement. · Do not give live vaccines while B cells are low.

Calcineurin inhibitors: cyclosporine or tacrolimus (in steroid-resistant cases) · Calcineurin inhibitor (immune suppressant)

Blocks calcineurin, an enzyme inside T cells that normally turns on genes needed to start an immune attack. Without calcineurin working, T cells stay quiet and stop releasing signals that damage the kidney filters. This helps in steroid-resistant nephrotic syndrome, especially FSGS and some cases of minimal change disease.

1. The drug enters T cells and binds to a protein inside (cyclophilin for cyclosporine, FKBP for tacrolimus).
2. This complex blocks calcineurin.
3. Calcineurin can no longer activate NFAT (a gene switch).
4. T cells cannot make IL-2 and other inflammatory signals.
5. The immune attack on the kidney filters weakens, and protein leak drops.

SAFETY WATCH

Can damage the kidneys (nephrotoxicity) — check creatinine and drug levels regularly. The doctor will adjust the dose to keep the blood level in a safe range. · Raises blood pressure — monitor closely and treat if needed. · Increases infection risk, especially viral infections (like CMV and BK virus). · Can cause tremor (shaking hands), especially with tacrolimus. · Raises the risk of certain cancers (especially skin cancer and lymphoma) with long-term use. Use sun protection and get regular skin checks. · Can raise blood sugar and potassium. · Many drug interactions — check before starting any new medicine.

Red flags

- Sudden chest pain or trouble breathing (may mean a blood clot traveled to the lungs)
- Fever with chills or a new cough (may mean pneumonia or another serious infection)

- Severe belly pain or swelling that comes on fast (may mean infection of fluid in the belly or a clot in a belly vein)
- Blood in the urine that you can see, or urine that turns cola-colored (may mean the kidneys are inflamed in a dangerous way, not just leaking)
- Swelling so bad you can't breathe well lying flat, or swelling in the face and throat (may mean fluid is backing up into the lungs or airway)

Nursing priorities

1. Weigh the patient daily at the same time with the same clothing and record strict intake and output every shift — Fluid retention from low albumin causes swelling (edema) (swelling); daily weights detect fluid overload before it becomes severe pulmonary edema (fluid flooding the lungs) (fluid in lungs), and tracking helps adjust diuretic (water pill) doses
2. Monitor blood pressure every 4 hours and report systolic over 140 or diastolic over 90 immediately — Damaged kidneys activate the renin-angiotensin-aldosterone system (a hormone chain that raises blood pressure), causing high blood pressure (hypertension) (high blood pressure) that worsens kidney damage; ACE inhibitors or ARBs lower pressure and reduce proteinuria
3. Check albumin and protein levels and watch for signs of blood clots like sudden leg swelling, chest pain, or shortness of breath — When albumin drops below 2.5 grams per deciliter, the liver overproduces clotting factors and blood becomes hypercoagulable; venous thromboembolism (clots in veins) happens in 25 percent of patients and needs immediate anticoagulation (blood thinners)
4. Teach and enforce a low-sodium diet under 2 grams per day and restrict fluids if swelling (edema) worsens — Low albumin triggers aldosterone (a hormone) release that makes kidneys hold sodium and water; restricting sodium reduces fluid retention and makes loop diuretics like furosemide work better to pull off extra fluid
5. Inspect skin daily for breakdown, especially over swollen areas like ankles, sacrum, and eyelids, and reposition every 2 hours — Swelling (Edema) fluid stretches skin tight and reduces oxygen delivery to tissues; pressure plus poor blood flow (perfusion) (blood flow) causes skin breakdown and cellulitis (skin infection), which spreads easily because immunoglobulins are lost in urine
6. Monitor urine for protein with dipstick every void and send 24-hour urine collection for total protein measurement — Proteinuria over 3.5 grams per day defines nephrotic syndrome; tracking urine protein quantifies how well treatment is working and guides steroid or immunosuppressant dose adjustments

7. Watch for infection signs like fever over 100.4 Fahrenheit, cough, burning with urination (dysuria) (painful urination), or redness and report immediately — Lost immunoglobulin G in urine leaves patients immunocompromised (weak immune system), especially vulnerable to *Streptococcus pneumoniae* and spontaneous bacterial peritonitis (infected belly fluid); early antibiotics prevent sepsis (life-threatening infection)
8. Educate the patient to avoid NSAIDs, get pneumococcal vaccine, and recognize symptoms needing emergency care like facial swelling or breathing trouble — NSAIDs worsen proteinuria and kidney function; vaccines prevent encapsulated bacterial infections; recognizing anasarca (total body swelling) or pulmonary edema (fluid flooding the lungs) empowers patients to seek help before respiratory failure

Sources

- KDIGO Clinical Practice Guideline for Glomerulonephritis 2021
- American Society of Nephrology
- National Kidney Foundation
- UpToDate Clinical Reference (Nephrology)

Last reviewed: 2026-07-07