

Nephrotic Syndrome

A plain-English guide for you and your family

WHAT IS HAPPENING

Your kidneys have tiny filters called glomeruli. Each filter is made of cells with little feet, called podocytes, that fit together like fingers laced in a fence. Normally this fence only lets water and waste through but keeps big important proteins—especially albumin—inside your blood. Albumin is the main protein that acts like a sponge, holding fluid inside your vessels so you don't swell up. When the podocytes get damaged, gaps open in the fence and albumin pours into the urine. Your blood albumin level drops. Without enough albumin sponge-power, fluid leaks out of your vessels into your tissues—that's why you see puffiness around the eyes in the morning, swollen ankles, and sometimes a swollen belly. Your liver tries to help by making more cholesterol to thicken the blood, so cholesterol shoots up. But other protective proteins also leak out: antithrombin (which keeps clots from forming) and immunoglobulins (antibodies that fight germs). So now your blood clots more easily and you catch infections more easily, especially pneumonia in kids. In children, the most common cause is minimal change disease, where the podocyte feet just flatten out (you can't even see it under a regular microscope). In adults, the top causes are focal segmental glomerulosclerosis (FSGS, where some filters scar), membranous nephropathy (where the filter wall thickens), and diabetes (high sugar damages the filter over time). Other triggers include lupus, infections like hepatitis B or C and HIV, some pain medicines (NSAIDs), and rarely cancer. The key idea: one break upstream—podocyte damage—explains every strange finding you see downstream. Trace it back and it all connects.

What you might feel

- Swelling around the eyes and in the legs (edema)
- Foamy or frothy urine
- Weight gain (often several pounds in a few days)
- Fatigue and low energy
- Loss of appetite or belly fullness
- High cholesterol and fats in the blood
- Infections happen more often (especially pneumonia or skin infections)

- Blood clots in legs or lungs (deep vein clot formation (thrombosis) or pulmonary embolism (a clot lodging in a lung artery))

Call the doctor right away if...

- 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)

What the care team will do

- Weigh the patient daily at the same time with the same clothing and record strict intake and output every shift
- Monitor blood pressure every 4 hours and report systolic over 140 or diastolic over 90 immediately
- Check albumin and protein levels and watch for signs of blood clots like sudden leg swelling, chest pain, or shortness of breath
- Teach and enforce a low-sodium diet under 2 grams per day and restrict fluids if swelling (edema) worsens
- Inspect skin daily for breakdown, especially over swollen areas like ankles, sacrum, and eyelids, and reposition every 2 hours
- Monitor urine for protein with dipstick every void and send 24-hour urine collection for total protein measurement
- Watch for infection signs like fever over 100.4 Fahrenheit, cough, burning with urination (dysuria) (painful urination), or redness and report immediately
- Educate the patient to avoid NSAIDs, get pneumococcal vaccine, and recognize symptoms needing emergency care like facial swelling or breathing trouble

Medicines you may be given — and why

Lisinopril or enalapril (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.

Losartan or valsartan (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.

Furosemide (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.

Atorvastatin or simvastatin (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.

Warfarin or low-molecular-weight heparin (anticoagulant)

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)).

Prednisone or other corticosteroid

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.

Cyclophosphamide (in steroid-resistant or relapsing cases)

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).

Rituximab (in certain resistant or relapsing cases)

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.

Calcineurin inhibitors: cyclosporine or tacrolimus (in steroid-resistant cases)

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.

Where this information comes from

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

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