

CKD · Chronic Kidney Disease

Chronic kidney disease — slow the decline, treat the downstream effects, and prepare for dialysis or transplant.

THE ONE-LINE TRUTH

The kidneys have been losing their cleaning power slowly, over months or years, and they cannot heal back.

The chain — how it unfolds

1. Nephrons die from diabetes, high blood pressure, or another cause. — why: High blood sugar damages the glomerular filters by thickening their walls and making them leaky. High blood pressure scars kidney tissue by forcing blood through at too much pressure. Once nephrons are lost, they never grow back.
2. The remaining nephrons hyperfiltrate to compensate. — why: Surviving nephrons increase their workload to keep waste clearance steady. This keeps GFR from crashing immediately, but the extra pressure inside these nephrons scars them and speeds their death.
3. GFR falls and waste products (creatinine, urea) build up in the blood. — why: As more nephrons die, the kidneys cannot filter enough blood. Toxins that normally leave in urine start to accumulate, causing severe waste buildup in the blood (uremia) (toxin buildup symptoms like nausea, confusion, and itching).
4. Erythropoietin production drops, causing anemia. — why: Damaged kidney tissue cannot make enough erythropoietin, so bone marrow slows red blood cell production. Low red blood cells mean less oxygen delivery, causing fatigue, weakness, and shortness of breath.
5. Vitamin D activation fails, causing low calcium and high PTH. — why: Without active vitamin D, the gut cannot absorb calcium from food. Serum calcium drops, and the parathyroid glands crank out PTH to pull calcium from bones into the blood. This prevents dangerous low calcium but causes bone pain, fractures, and vascular calcification.
6. Phosphate excretion fails, causing hyperphosphatemia. — why: Damaged kidneys cannot clear phosphate, so it builds up in the blood. High phosphate binds calcium and deposits in arteries and soft tissues, making blood vessels stiff and brittle.

7. Acid excretion fails, causing metabolic acidosis (acid building up in the blood). — why: The kidneys cannot remove hydrogen ions or regenerate bicarbonate, so acid builds up in the blood. This makes the body work harder, worsens bone loss, and increases muscle breakdown.
8. Fluid and salt retention causes high blood pressure (hypertension) and swelling (edema). — why: Damaged kidneys cannot adjust urine output to match intake, so extra salt and water stay in the body. This raises blood pressure, causes leg and lung swelling, and eventually leads to heart failure.

Spot it — what you see, and why

- Creatinine level in the blood rises slowly over months or years: The kidneys filter creatinine (a waste made by muscles) out of the blood. When the kidneys are damaged, they can't clean creatinine out as well, so it builds up in the blood like trash piling up when the garbage truck stops coming.
- Estimated glomerular filtration rate (eGFR) drops slowly over time: eGFR measures how much blood the kidneys can filter each minute. Damaged kidneys have fewer working filter units (nephrons), so less blood gets cleaned — like a coffee filter with holes in it that lets dirty water through.
- Urine shows rising amounts of albumin (protein) compared to creatinine: Healthy kidney filters keep big molecules like albumin (a blood protein) inside the blood. When the filters are damaged, albumin leaks into the urine like sand falling through a torn screen.
- The person becomes anemic (low red blood cell count): Healthy kidneys make a hormone called erythropoietin that tells bone marrow to make red blood cells. Damaged kidneys make less of this hormone, so fewer red cells are produced — like a factory slowing down when the order sheet is lost.
- Blood pressure goes up and is hard to control: Damaged kidneys hold onto extra salt and water, which raises the volume of blood in the vessels and pushes pressure up. The kidneys also release chemicals that tighten blood vessels, making pressure climb even higher — like overfilling a balloon.
- No symptoms at all in the early stages: The kidneys have a lot of extra capacity. Even when half the filter units are damaged, the remaining ones can still do enough work to keep waste from piling up to noticeable levels — like losing half your workers but still finishing the job on time.

Red flags — act now

- Sudden sharp drop in urine output or no urine at all for several hours

- Severe swelling all over the body, trouble breathing, or fluid in the lungs (pulmonary swelling (edema))
- Confusion, seizures, or uncontrollable muscle twitching (signs that waste toxins are reaching dangerous levels in the blood)
- Chest pain or irregular heartbeat (potassium may be dangerously high, which can stop the heart)
- Blood in the urine that appears suddenly or gets much worse quickly

Treat it — in this order

1. ACE inhibitor (such as lisinopril) or ARB (such as losartan), aiming for blood pressure below 130/80 mmHg → These medicines block a hormone called angiotensin II. That hormone squeezes blood vessels inside the kidney and raises pressure in the filters. Blocking it relaxes those vessels, lowers the pressure, and cuts down protein leaking into the urine. This slows down how fast the kidney disease gets worse.
2. SGLT2 inhibitor (such as empagliflozin or dapagliflozin), even if the person does not have diabetes → This medicine blocks the kidney from pulling sugar back into the blood. That lowers blood sugar and lowers pressure inside the filters. Studies show it slows kidney failure in both people with diabetes and people without diabetes.
3. Erythropoiesis-stimulating agent (ESA, such as epoetin alfa or darbepoetin), after fixing low iron, aiming for hemoglobin between 10 and 11.5 g/dL → This replaces the erythropoietin hormone the kidneys no longer make. It tells the bone marrow to make more red blood cells, which raises oxygen in the body and reduces tiredness.
4. Phosphate binder (such as calcium acetate or sevelamer) taken with meals, plus active vitamin D (calcitriol) if needed → The binder sticks to phosphate in food so it cannot be absorbed into the blood. This lowers blood phosphate. Calcitriol replaces the active vitamin D the kidneys no longer make, which helps control calcium and parathyroid hormone. Together they protect bones and blood vessels from hardening.
5. Sodium bicarbonate (baking soda) tablets when serum bicarbonate falls below 22 mEq/L → This neutralizes the acid that builds up when the kidneys cannot get rid of it. It stops muscle breakdown, protects bones, and may slow kidney disease progression.

Your top priorities at the bedside

1. Check potassium level immediately and get a 12-lead ECG if the patient reports chest pain, palpitations, muscle weakness, or tingling, or if potassium is above 5.5 mEq/L. — why: High potassium (Hyperkalemia) (high potassium) happens when kidneys cannot excrete potassium; levels above 6.0 mEq/L can cause deadly arrhythmias (irregular heartbeats) like ventricular fibrillation and cardiac arrest within minutes, so urgent treatment with insulin-dextrose, calcium gluconate, or dialysis is needed.
2. Measure strict intake and output every shift, weigh the patient daily at the same time with the same clothing, and assess for pitting swelling (edema) in ankles, sacrum, and lungs (crackles on listening with a stethoscope (auscultation)). — why: Damaged kidneys cannot remove extra fluid, so fluid overload builds up in the tissues (edema) and lungs (pulmonary swelling (edema)), causing heart failure and respiratory distress; tracking fluid balance guides diuretic dosing and tells us when dialysis is needed to pull off fluid.
3. Hold NSAIDs (ibuprofen, naproxen, ketorolac) and review the medication list to stop or adjust nephrotoxic drugs (drugs that poison the kidneys) like aminoglycosides, vancomycin, and contrast dye. — why: NSAIDs reduce blood flow to the kidneys by blocking prostaglandins (chemicals that open kidney blood vessels), causing acute worsening of CKD; other nephrotoxic drugs accumulate to dangerous levels when kidneys fail, so doses must be reduced or stopped to prevent further damage.