

CKD • Chronic Kidney Disease

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THE STORY IN ONE SENTENCE

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

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

Normal anatomy — the body's job

Your kidneys are two fist-sized organs in your back that clean about 50 gallons of blood every day. Each kidney holds about one million nephrons — tiny filters that pull waste out of your blood and keep the good stuff in. The cleaned blood goes back into your body, and the waste leaves as urine. Your kidneys also make hormones that control red blood cell production, activate vitamin D for bone health, and help manage blood pressure and acid balance. Keep this normal picture in your head, because every symptom of kidney disease makes sense as a loss of one of these jobs.

BODY'S JOB

If you do not know what kidneys normally do, the lab numbers and symptoms will seem like a random mess instead of a clear story.

1. Blood flows into the kidney through the renal artery.
2. Blood enters a nephron and passes through a glomerulus (a tiny ball of capillaries).
3. The filtered fluid travels down a long tubule.
4. What is left becomes urine and flows to the bladder.
5. Kidneys make erythropoietin and release it into the blood.
6. Kidneys activate vitamin D.
7. Kidneys sense blood pressure and release renin when it drops.

What breaks

In chronic kidney disease, nephrons die slowly and do not grow back. Once enough are lost, the remaining ones get overworked and fail faster, creating a downward spiral.

CLINICAL TERM

The most common cause is diabetic nephropathy (high blood sugar damages the glomerular filters). The second most common is with high blood pressure (hypertensive) nephrosclerosis (high blood pressure scars the kidney tissue). Other causes include glomerulonephritis (autoimmune attack on the filters), polycystic kidney disease (inherited cysts replace normal tissue), chronic obstruction (stones or prostate blockage damage the kidneys backward), and recovery from acute kidney injury that left permanent scarring. No matter the cause, loss of nephron mass triggers hyperfiltration in the remaining nephrons, which accelerates their decline.

How the body compensates

The body tries to keep balance as kidney function falls. Remaining nephrons work harder (hyperfiltration), the heart pumps harder to clear toxins, and bones release calcium to buffer acid. These tricks keep the patient stable for a while, but they speed up kidney damage and eventually fail.

1. {'step': 'Remaining nephrons increase their filtration rate.', 'why': 'When some nephrons die, the survivors filter more blood to compensate. This keeps waste clearance stable early on, but the extra pressure scars the remaining nephrons and speeds their decline.'}
2. {'step': 'The renin-angiotensin-aldosterone system (RAAS) turns on.', 'why': 'Damaged kidneys sense lower blood flow and release renin, which raises blood pressure to try to keep filtration going. This briefly helps, but high pressure inside the glomeruli damages them further.'}
3. {'step': 'The heart pumps harder and the blood vessels constrict.', 'why': 'The body tries to push more blood through damaged kidneys to clear toxins. This raises blood pressure and heart workload, leading to heart enlargement and heart failure over time.'}
4. {'step': 'Parathyroid glands release more PTH.', 'why': 'When kidneys cannot activate vitamin D, calcium levels drop. PTH pulls calcium from bones into the blood to prevent dangerous low calcium, but this weakens bones.'}
5. {'step': 'Bones release calcium and carbonate to buffer acid.', 'why': 'When kidneys cannot clear acid, bones dissolve to neutralize it. This prevents immediate acid toxicity but causes bone disease (renal osteodystrophy).')}

6. {'step': 'The body retains salt and water to maintain blood volume.', 'why': 'Damaged kidneys cannot excrete excess fluid, so the body holds onto it to keep blood pressure from dropping. This causes swelling and eventually heart failure.'}
7. {'step': 'Watch for the tipping point: sudden high potassium (hyperkalemia), pulmonary edema (fluid flooding the lungs), or uremic encephalopathy.', 'why': 'Compensation works until GFR drops below 15 or a stressor (missed dialysis, ACE inhibitor, infection) pushes potassium or fluid over the edge. These crises can be fatal within hours.'}
8. {'step': 'Most CKD patients die of cardiovascular disease before reaching dialysis.', 'why': 'Chronic inflammation, high blood pressure, vascular calcification, and anemia all damage the heart and arteries faster than the kidneys fail.'}

Domino effect

1. Nephrons die from diabetes, high blood pressure, or another cause. — 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. — 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. — 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. — 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. — 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. — 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). — 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). — 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.

Symptoms · what the patient feels, 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.
- Fatigue and weakness that gets worse over time: Anemia (fewer oxygen-carrying red cells) and buildup of waste chemicals in the blood make muscles tired. The body is running on low fuel and dirty oil at the same time.
- Intense itching all over the skin: Waste products and imbalanced minerals (especially high phosphorus) in the blood irritate nerve endings in the skin, triggering constant itching — like having invisible dust under your skin that you can't scratch away.
- Loss of appetite, nausea, and sometimes vomiting: Waste toxins (uremia) build up in the blood and irritate the stomach and intestines, making food unappealing and triggering nausea — like feeling carsick all the time.

- Swelling in the ankles, feet, or around the eyes: Damaged kidneys can't remove extra salt and water, so fluid leaks out of blood vessels into nearby tissues. Gravity pulls it to the feet and ankles; lying down shifts it to the face and eyelids — like a sponge soaking up water.

Focused exam

- Blood pressure 140/90 mmHg or higher on repeated checks: High blood pressure damages the tiny filters in the kidneys and makes chronic kidney disease get worse faster. It also happens because damaged kidneys hold onto salt and water. Treating it slows the disease down.
- Pale skin, pale inside the lower eyelids, or pale nail beds: Healthy kidneys make a hormone called erythropoietin (say: ee-RITH-row-POY-ih-tin) that tells bone marrow to make red blood cells. When kidneys fail, they stop making enough of this hormone, so fewer red blood cells are made. Fewer red blood cells mean less oxygen reaches the body, causing tiredness and paleness.
- Pitting swelling (edema) (press your thumb into the ankle; a dent stays) in the legs, ankles, or around the eyes: Damaged kidneys let protein leak out into the urine and cannot get rid of extra salt and water. The fluid backs up into the tissues, causing swelling. This shows the filters are broken.
- Scratch marks on the arms, legs, or trunk: When kidneys fail, waste products build up in the blood. These wastes irritate the skin from the inside and cause severe itching. Scratch marks are a sign kidney function has dropped a lot.
- Uremic frost (white or yellow crystals on the skin) or a smell like urine on the breath: This happens only in very advanced kidney failure. Urea (a waste product) is so high in the blood that it comes out through the skin and lungs. This is a medical emergency; the person needs dialysis right away.
- Weak, irregular pulse or chest pain: Damaged kidneys cannot get rid of potassium. When potassium rises above 6.0 mEq/L, the heart's electrical system can stop suddenly. This needs emergency treatment.

Diagnostics · every test answers a question

- Serum creatinine and estimated glomerular filtration rate (eGFR) using the CKD-EPI equation → How well are the kidneys filtering waste out of the blood?
- Urine albumin-to-creatinine ratio (UACR) on a spot urine sample → Are the kidney filters letting protein leak into the urine?
- Complete blood count (CBC) with hemoglobin and hematocrit → Is the person anemic because the kidneys are not making enough erythropoietin?

- Serum potassium → Are the kidneys getting rid of potassium, or is it building up to dangerous levels?
- Serum phosphate, calcium, parathyroid hormone (PTH), and 25-hydroxyvitamin D → Are the kidneys controlling mineral balance, or is bone disease starting?
- Serum bicarbonate (part of a basic metabolic panel) → Are the kidneys getting rid of acid, or is acid building up in the blood?
- Renal ultrasound → Are the kidneys small and scarred (meaning chronic disease), or are they blocked or have a reversible problem?
- Hemoglobin A1c and fasting glucose → Is diabetes the cause of the kidney damage?

Treatment logic

- 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.
- 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.
- 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.
- 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.
- 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.

- Referral to a nephrologist for transplant evaluation and dialysis access planning when eGFR drops below 20 mL/min/1.73 m² → This prepares the person before an emergency happens. An arteriovenous fistula (a vein and artery joined surgically in the arm for dialysis) takes 2 to 4 months to mature and be ready to use. Transplant evaluation takes months of testing. Starting early gives the person choices and avoids emergency dialysis through a neck catheter.
- Dietary protein restriction to 0.8 g per kg body weight per day in stages 3 to 5 chronic kidney disease → Less protein means less nitrogen waste (urea) the kidneys have to filter. This reduces the workload on remaining nephrons and may slow decline. Too little protein, though, causes muscle loss.

Medications

Lisinopril or Enalapril (ACE inhibitor) · ACE inhibitor (angiotensin-converting enzyme inhibitor)

Your kidney has tiny filters called glomeruli. When you have CKD, pressure builds up inside these filters and protein leaks through. This drug blocks a chemical in your blood called angiotensin II that squeezes the blood vessels going OUT of the filter. When you block that squeeze, the pressure inside the filter drops and less protein leaks out. It also lowers your overall blood pressure, which protects your heart and slows down kidney damage.

1. Your body makes a hormone called angiotensin I.
2. An enzyme called ACE turns angiotensin I into angiotensin II.
3. Angiotensin II tightens the blood vessel leaving the filter (efferent arteriole) more than the one coming in, so pressure inside the filter rises.
4. This drug blocks ACE, so less angiotensin II is made.
5. With less angiotensin II, the outgoing vessel relaxes, pressure inside the filter drops, and less protein leaks into the urine.
6. Your blood pressure also goes down because blood vessels throughout your body relax.

SAFETY WATCH

Can raise potassium in your blood (your doctor will check blood tests regularly). · Can cause a dry cough in some people (switching to an ARB usually fixes this). · Rarely causes swelling of the lips, tongue, or throat (angioedema) — if this happens, stop the drug and get emergency help right away. · Should NOT be used during pregnancy because it can harm the baby. · Your kidney function may dip slightly when you start — this is expected and usually okay, but your doctor will watch your blood tests closely.

Losartan or Valsartan (ARB) · ARB (angiotensin receptor blocker)

This drug does the same job as an ACE inhibitor but by a different route. Instead of stopping your body from making angiotensin II, it blocks the spots (receptors) where angiotensin II would normally grab on. So even though angiotensin II is still in your blood, it can't squeeze the blood vessels. The result is the same: pressure inside the kidney filter drops, less protein leaks out, and your blood pressure goes down.

1. Your body still makes angiotensin II.
2. Angiotensin II floats through your blood looking for its receptor (AT1) to land on.
3. This drug sits on the AT1 receptor and blocks angiotensin II from docking there.
4. Without angiotensin II docking, the blood vessel leaving the kidney filter stays relaxed.
5. Pressure inside the filter drops, less protein leaks into urine, and overall blood pressure falls.

SAFETY WATCH

Can raise potassium (regular blood tests needed). · Usually does NOT cause a cough, so it's the usual switch if ACE inhibitors cause cough. · Same risk of angioedema as ACE inhibitors, though much rarer. · Should NOT be used during pregnancy. · Small dip in kidney function when starting is expected; your doctor will monitor.

Empagliflozin or Dapagliflozin (SGLT2 inhibitor) · SGLT2 inhibitor (sodium-glucose cotransporter-2 inhibitor)

Your kidney normally filters sugar out of your blood, then sucks most of it back in so you don't lose it in urine. A protein called SGLT2 does that sucking-back job in the first part of the kidney tube. This drug blocks SGLT2, so sugar (and a little salt and water) spills into your urine instead. Losing that sugar lowers blood sugar if you have diabetes. But even if you don't have diabetes, this drug protects your kidney by lowering the workload on the filters, reducing inflammation, and helping your heart pump better. Studies show it slows CKD in both diabetic and non-diabetic patients.

1. Blood flows through the glomerulus and gets filtered; sugar enters the tubule.
2. Normally, SGLT2 proteins in the tubule wall grab sugar and salt and pull them back into the blood.
3. This drug blocks SGLT2, so sugar and salt stay in the tubule and leave in your urine.
4. You pee out extra sugar, which lowers blood sugar if you have diabetes.
5. You also pee out a little extra salt and water, which lowers blood pressure slightly and reduces strain on the heart.

6. The kidney itself uses less oxygen and has less inflammation, which slows scarring and protects the filters over time.

SAFETY WATCH

You will urinate more often, especially in the first few weeks. · More sugar in urine raises the risk of yeast infections (especially in women) and urinary tract infections — good hygiene helps. · Rarely can cause a serious condition called euglycemic DKA (diabetic ketoacidosis even when blood sugar isn't very high) — if you feel very sick, nauseous, or have trouble breathing, get help right away. · Drink enough water; dehydration can happen if you're also taking diuretics or losing fluids from illness. · Not recommended if eGFR is under 20 mL/min.

Atorvastatin or Rosuvastatin (statin) · Statin (HMG-CoA reductase inhibitor)

CKD raises your risk of heart attack and stroke because it increases inflammation and causes cholesterol to build up in artery walls. Your liver makes cholesterol using an enzyme called HMG-CoA reductase. This drug blocks that enzyme, so your liver makes less cholesterol. Your liver then pulls more cholesterol out of your blood to make up for it, which lowers the cholesterol floating around that can clog arteries. Statins also calm down inflammation in blood vessel walls.

1. Your liver cells use the enzyme HMG-CoA reductase to build cholesterol.
2. This drug blocks that enzyme.
3. Your liver makes less cholesterol inside its cells.
4. To get the cholesterol it needs, the liver puts more LDL receptors on its surface and pulls LDL cholesterol out of your blood.
5. LDL cholesterol in your blood drops.
6. Less cholesterol is available to stick in artery walls, and inflammation goes down, which protects your heart and brain.

SAFETY WATCH

Can rarely cause muscle pain or weakness — tell your doctor if this happens. · Very rarely can damage muscle tissue (rhabdomyolysis), especially at high doses or when combined with certain other drugs — your doctor will check for this if you have severe muscle pain, dark urine, or feel very weak. · Can raise liver enzymes slightly; your doctor may check blood tests. · Avoid grapefruit juice with some statins (it raises drug levels in your blood). · Safe for most people with CKD, even advanced CKD; dose may be adjusted.

Calcium carbonate or Sevelamer (phosphate binder) · Phosphate binder

As CKD gets worse, your kidney can't get rid of phosphorus (a mineral from food) as well. Phosphorus builds up in your blood and pulls calcium out of your bones, making them weak. High phosphorus also causes calcium and phosphorus to form crystals that deposit in your blood vessels and heart, making them stiff. Phosphate binders are pills you take with meals. They grab onto phosphorus in your stomach so it sticks to the pill and leaves in your poop instead of entering your blood.

1. You eat food that contains phosphorus.
2. Phosphorus enters your stomach and intestines.
3. You take a binder pill with your meal.
4. The binder grabs phosphorus and holds it tightly.
5. The phosphorus-binder clump moves through your intestines and leaves in your stool.
6. Less phosphorus enters your blood, so blood phosphorus levels stay closer to normal.

SAFETY WATCH

Calcium-based binders (like calcium carbonate) can raise calcium in your blood if you take too much; your doctor will monitor levels. · Can cause constipation or upset stomach. · Must be taken WITH meals to work — if you take them between meals, they won't bind phosphorus. · Some binders (sevelamer) do not contain calcium and are used if your calcium is already high.

Cinacalcet (calcimimetic) · Calcimimetic

When phosphorus goes up and calcium goes down because of CKD, your parathyroid glands (four tiny glands in your neck) sense the problem and pump out parathyroid hormone (PTH). PTH pulls calcium out of your bones to raise blood calcium, but this weakens your bones and makes blood vessel calcification worse. This drug tricks your parathyroid glands into thinking calcium is higher than it really is, so they make less PTH. Lower PTH means less calcium pulled from bone and lower phosphorus and calcium in your blood.

1. Your parathyroid glands have receptors that sense calcium in your blood.
2. When calcium is low (or phosphorus is high), the glands release PTH.
3. This drug sticks to the calcium-sensing receptor and makes it more sensitive.
4. The receptor now thinks there's more calcium than there really is.
5. The parathyroid glands release less PTH.
6. With less PTH, less calcium is pulled from bone, and phosphorus and calcium levels in blood stay more balanced.

SAFETY WATCH

Can lower calcium in your blood too much — your doctor will check levels regularly. · Can cause nausea or vomiting, especially when starting. · Usually used in stage 4–5 CKD or dialysis, not in earlier stages.

Calcitriol or Paricalcitol (active vitamin D) · Active vitamin D analogue

Your kidney normally turns regular vitamin D into its active form (calcitriol). Active vitamin D helps your intestines absorb calcium from food and tells your parathyroid glands to calm down and make less PTH. When your kidneys fail, they can't make enough active vitamin D, so calcium drops and PTH goes up, weakening bones. This drug is already-active vitamin D, so it can do the job even though your kidneys can't activate it anymore.

1. Normally, your kidney converts inactive vitamin D to calcitriol.
2. In CKD, the kidney can't do this well.
3. You take calcitriol or a similar drug (already active).
4. It helps your intestines absorb more calcium from food.
5. It signals the parathyroid glands to make less PTH.
6. Calcium goes up slightly, PTH goes down, and bones stay stronger.

SAFETY WATCH

Can raise calcium and phosphorus in your blood — your doctor will check levels often. · High calcium and phosphorus together can cause calcium deposits in blood vessels and soft tissues. · Used carefully, usually in stage 3–5 CKD when PTH is rising.

Ferrous sulfate or Iron sucrose (iron supplement) · Iron supplement (oral or intravenous)

Your bone marrow needs iron to make hemoglobin, the part of red blood cells that carries oxygen. In CKD, you often don't absorb enough iron from food, and inflammation locks iron inside storage cells so your marrow can't use it. Taking iron (by mouth or through an IV) gives your marrow the building blocks it needs to make more red blood cells and raise your hemoglobin.

1. You take an iron pill or receive iron through an IV.
2. Iron enters your blood.
3. Your bone marrow pulls iron from the blood.
4. Marrow cells use iron to build hemoglobin.
5. New red blood cells with hemoglobin are released into your blood.

6. Hemoglobin carries oxygen to your tissues, so you feel less tired and weak.

SAFETY WATCH

Oral iron (pills) can cause constipation, dark stools, nausea, or stomach upset. · IV iron works faster and avoids stomach problems but must be given in a clinic or hospital. · Your doctor will check iron levels (ferritin and transferrin saturation) to make sure you're getting enough but not too much. · Too much iron can be harmful, so don't take extra without asking your doctor.

Epoetin alfa or Darbepoetin alfa (ESA) · ESA (erythropoiesis-stimulating agent)

Your kidney makes a hormone called erythropoietin (EPO) that travels to your bone marrow and tells it to make red blood cells. In CKD, your damaged kidney makes less EPO, so your marrow slows down and you become anemic (low red blood cell count). This drug is man-made EPO. It gives your marrow the signal it's missing, so it speeds up and makes more red cells. Your hemoglobin (oxygen-carrying protein) goes up, and you feel less tired and short of breath.

1. You receive an ESA injection (under the skin or into a vein).
2. The ESA travels through your blood to your bone marrow.
3. It docks on EPO receptors on marrow cells.
4. The marrow cells get the signal to make more red blood cells.
5. New red cells enter your bloodstream over the next few weeks.
6. Hemoglobin rises, and your tissues get more oxygen.

SAFETY WATCH

Target hemoglobin is 10 to 11.5 g/dL — higher levels raise the risk of stroke, heart attack, and blood clots. · Your doctor will check hemoglobin regularly and adjust the dose to stay in the safe range. · Can raise blood pressure — monitor BP closely. · Rarely can cause serious allergic reactions or pure red cell aplasia (marrow stops making red cells entirely) — very rare but serious. · Only used when hemoglobin is low (usually under 10 g/dL) and iron stores are okay.

Sodium bicarbonate (baking soda tablets) · Alkalinizing agent

Your kidney normally removes acid that your body makes every day from breaking down protein. In CKD, the kidney can't get rid of acid as well, so acid builds up in your blood (metabolic too much acid in the blood (acidosis)). This makes you feel tired, weakens your bones, and makes kidney disease progress faster. Sodium bicarbonate is a base (the opposite of

acid). When you take it, it neutralizes the extra acid in your blood, bringing your blood pH back toward normal.

1. Your body makes acid from metabolism.
2. Your damaged kidney can't remove it all.
3. Acid builds up in your blood, lowering blood pH (making it more acidic).
4. You take sodium bicarbonate tablets.
5. Bicarbonate (a base) mixes with acid in your blood and neutralizes it.
6. Blood pH rises back toward normal, and your body's chemistry works better.

SAFETY WATCH

Contains sodium (salt), so it can raise blood pressure or cause fluid retention — your doctor will monitor. · Can cause bloating or gas. · Your doctor will check blood bicarbonate levels (usually aiming for 22–26 mEq/L). · Usually used when serum bicarbonate is under 22 mEq/L.

Diuretics (furosemide, bumetanide, or metolazone) · Loop diuretic or thiazide-type diuretic

When your kidney can't remove enough salt and water, fluid builds up in your body — you see swelling in your legs, ankles, or lungs (shortness of breath). Diuretics are 'water pills' that tell your kidney to dump more salt and water into your urine. This shrinks the fluid overload, lowers blood pressure, and makes breathing easier. Loop diuretics (like furosemide) work in the loop of Henle (a deeper part of the kidney tube) and are stronger. Thiazide-type diuretics (like metolazone) work in a different part of the tube and are sometimes added if loop diuretics alone aren't enough.

1. Fluid filters into the kidney tube.
2. Normally, most salt and water are pulled back into the blood so you don't lose too much.
3. This drug blocks the transporters that pull salt back in.
4. Salt stays in the tube, and water follows salt (water goes where salt goes).
5. You pee out more salt and water.
6. Fluid overload goes down, swelling shrinks, and blood pressure drops.

SAFETY WATCH

Can lower potassium, sodium, or magnesium — your doctor will check blood tests regularly. · Can make you feel dizzy or lightheaded when you stand up (low blood pressure). · You will urinate much more, especially in the first few hours after taking the pill — plan bathroom access. · Can make kidney function look worse on blood tests (creatinine may rise slightly), but this is usually okay if you were overloaded with fluid. · Hearing loss (rare) with very high doses of loop diuretics, especially IV.

Patiromer or Sodium zirconium cyclosilicate (potassium binder) · Potassium binder

In CKD, your kidney can't remove potassium well, and some CKD medications (ACE inhibitors, ARBs, spironolactone) raise potassium even more. High potassium (hyperkalemia) is dangerous — it can stop your heart. These drugs are powders you mix with water and drink. They travel to your colon (large intestine), grab onto potassium there, and carry it out in your stool. This lowers the potassium in your blood so you can safely keep taking the protective kidney medicines.

1. Potassium from food and from your body enters your colon.
2. You drink the binder powder.
3. The binder particles stick to potassium in the colon.
4. The potassium-binder clump leaves in your stool.
5. Less potassium gets reabsorbed into your blood.
6. Blood potassium drops.

SAFETY WATCH

Can cause constipation, diarrhea, nausea, or stomach discomfort. · Patiromer can lower magnesium — your doctor may check levels. · Must be taken several hours apart from other medicines because it can interfere with absorption. · Your doctor will check potassium levels regularly to make sure the dose is right.

Red flags

- 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

Nursing priorities

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. — 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)). — 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. — 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.
4. Check blood pressure every 4 hours and ensure ACE inhibitor (such as lisinopril) or ARB (such as losartan) is given daily, aiming to keep BP below 130/80 mmHg. — High BP damages the glomeruli (tiny kidney filters) by causing scarring and protein leakage; ACE inhibitors and ARBs lower pressure inside the glomeruli, reduce proteinuria (protein in urine), and slow progression to kidney failure by 30 to 50 percent.
5. Review the latest hemoglobin, and if below 10 g/dL, check iron studies (ferritin and transferrin saturation) before giving erythropoiesis-stimulating agent (ESA) or iron supplementation. — Healthy kidneys make erythropoietin (a hormone that tells bone marrow to make red blood cells); failing kidneys produce less, causing anemia; ESA only works if iron stores are adequate, so we must replace iron first to reach a safe hemoglobin target of 10 to 11.5 g/dL and prevent fatigue and heart strain.

6. Check serum phosphate, calcium, and parathyroid hormone (PTH) levels, and give phosphate binders (such as calcium acetate or sevelamer) with meals if phosphate is above 5.5 mg/dL. — Failing kidneys cannot remove phosphate, so it builds up in the blood and binds with calcium, forming deposits in blood vessels and heart valves (vascular calcification) and triggering secondary hyperparathyroidism; binders trap phosphate in the gut so it leaves in stool instead of damaging the body.
7. Teach the patient to limit dietary potassium (avoid bananas, oranges, tomatoes, potatoes, beans) and phosphorus (avoid dairy, nuts, cola drinks, processed meats) and refer to a renal dietitian. — When kidneys fail, potassium and phosphorus from food accumulate in the blood; high potassium stops the heart, and high phosphorus weakens bones and hardens arteries, so diet control reduces these risks and can delay the need for dialysis.
8. Assess the patient's understanding of CKD progression and discuss advance care planning, including options for hemodialysis, peritoneal dialysis, kidney transplant, or conservative management without dialysis. — CKD is irreversible and progressive; early education allows patients to make informed choices about dialysis access (AV fistula placement takes months to mature), transplant evaluation (waitlist time is years), or palliative care, reducing emergency dialysis starts and improving quality of life and outcomes.

Sources

- KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease
- National Kidney Foundation (NKF)
- American Society of Nephrology (ASN)
- CDC Chronic Kidney Disease Initiative

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