

Acute Kidney Injury

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THE STORY IN ONE SENTENCE

The kidneys suddenly stop cleaning the blood fast enough—waste piles up, salt and water go out of balance, and every organ starts to feel it within hours to days.

“The body's filter clogs. Everything the filter should catch now floats in the blood.”

Normal anatomy — the body's job

Each kidney holds about one million tiny filters called nephrons. Blood flows into each nephron under high pressure. Water and small molecules—waste, sugar, salt—squeeze out of the blood into a collecting cup called Bowman's capsule. Then a long, twisting tube pulls back the useful stuff (water, sodium, glucose) and leaves behind the waste (creatinine, urea, extra potassium). What is left becomes urine. The kidneys also make two important hormones: erythropoietin (EPO), which tells your bone marrow to make red blood cells, and renin, which helps control blood pressure. They also turn on vitamin D so your bones can use calcium.

BODY'S JOB

Clean about 180 liters of blood every day (your entire blood volume gets filtered roughly 60 times). Balance salt, water, potassium, and acid in the blood. Dump waste and leftover drugs into urine. Make hormones that keep your red blood cells, blood pressure, and bones healthy.

1. Renal artery — Carries one-fifth of every heartbeat's blood—about one liter per minute—straight to the kidney.
2. Glomerulus (the filter ball) — High pressure squeezes water, waste, salt, and sugar out of the blood into Bowman's capsule. Think of it like a sieve catching everything small enough to fit through.
3. Proximal tubule (first twisty tube) — Grabs back almost all the sugar and most of the salt and water the body needs. Waste like creatinine stays in the tube.

4. Loop of Henle (deep U-shaped loop) — Pulls out even more salt and water, making urine more concentrated so you do not lose too much fluid.
5. Distal tubule and collecting duct (final tuning zone) — Hormones like aldosterone and ADH fine-tune how much salt and water go back into the blood. This is where your body decides how concentrated or dilute your urine will be.
6. Ureter, then bladder — Finished urine drains down a tube (ureter) into the bladder, where it waits until you pee.

What breaks

Something suddenly stops the kidney from doing its job. There are three big ways this happens. Pre-renal means not enough blood is reaching the kidney—picture a garden hose kinked so no water flows through. This happens with dehydration, severe bleeding, heart failure, or shock. Intrinsic-renal (also called intrinsic AKI) means the filter tissue itself gets damaged—like the filter screen gets torn or clogged. The most common cause is acute tubular tissue death (necrosis), or ATN, where the tubule cells die from lack of oxygen or are poisoned by drugs (aminoglycoside antibiotics, NSAIDs like ibuprofen, or IV contrast dye used in CT scans). Post-renal means the drain is blocked downstream—a kidney stone stuck in the ureter, an enlarged prostate squeezing the urethra, or a bladder tumor. Pressure backs up into the kidney and crushes the filters from the inside.

CLINICAL TERM

AKI is diagnosed by KDIGO 2012 criteria when any one of these happens: serum creatinine rises by 0.3 mg/dL or more within 48 hours, OR creatinine climbs to 1.5 times baseline (or higher) within seven days, OR urine output drops below 0.5 mL per kilogram per hour for six hours or more.

How the body compensates

When blood flow drops, the kidney tries to save itself by holding onto every drop of salt and water it can. It activates a hormone chain called the renin-angiotensin-aldosterone system (RAAS) that squeezes blood vessels and tells the tubules to pull back more sodium and water. Urine output falls. BUN (a waste product) climbs faster than creatinine, giving a BUN-to-creatinine ratio above 20—the hallmark of pre-renal AKI. If blood flow stays low too long, the tubule cells run out of oxygen and die—this is acute tubular tissue death (necrosis), or ATN. Once that happens, the kidney loses the power to concentrate urine at all. Urine may barely

trickle out, or it may flow but be dilute and useless (called isosthenuria, meaning the urine has the same concentration as blood plasma—the kidney is no longer doing any real filtering work).

1. Kidney senses low blood flow and activates RAAS—renin is released, angiotensin II tightens blood vessels, and aldosterone tells the tubules to hold onto sodium and water hard. Urine output drops.
2. BUN rises faster than creatinine because the tubules are reabsorbing urea along with water, giving a BUN:creatinine ratio greater than 20:1. This is the classic pre-renal picture.
3. Urine becomes very concentrated (specific gravity above 1.020, urine sodium below 20 mEq/L, fractional excretion of sodium—FENa—below 1%) because the kidney is squeezing every bit of water and salt back into the blood.
4. If blood flow does not improve, tubule cells starve for oxygen and die—this is acute tubular tissue death (necrosis) (ATN). The kidney loses the ability to concentrate urine. Urine may become dilute (isosthenuria, specific gravity near 1.010), urine sodium climbs above 40 mEq/L, and FENa goes above 2%.
5. Potassium cannot be dumped into urine, so it builds up in the blood (hyperkalemia). This is dangerous because high potassium stops the heart from beating normally.
6. Acid (hydrogen ions) cannot leave the body, so metabolic acidosis (acid building up in the blood) develops. Blood pH drops, breathing gets faster as the lungs try to blow off carbon dioxide to compensate.
7. Phosphate builds up because the kidney cannot clear it. Calcium drops because high phosphate binds calcium in the blood.
8. Fluid overload occurs—the body cannot get rid of salt and water, so weight climbs, lungs fill with fluid (pulmonary swelling (edema) causing shortness of breath), ankles swell (peripheral swelling), and blood pressure may rise.
9. Uremic toxins (urea, creatinine, and many others) pile up in the blood, causing nausea, itching, confusion, a metallic taste, and—if untreated—inflammation of the heart sac (pericarditis) and brain dysfunction (uremic encephalopathy).

Domino effect

1. Trigger hits the kidney: severe dehydration (vomiting, diarrhea, not drinking), sepsis (bloodstream infection that drops blood pressure), major bleeding, heart failure (heart cannot pump enough blood forward), IV contrast dye (used in CT scans, toxic to tubules especially in people with diabetes or existing kidney disease), NSAIDs (ibuprofen, naproxen—they block prostaglandins that keep kidney blood vessels open), ACE inhibitors or ARBs during dehydration (they relax the vessel that drains the filter, dropping pressure inside), aminoglycoside antibiotics (gentamicin, tobramycin—directly poison tubule cells), kidney stone stuck in the ureter, or enlarged prostate (BPH) blocking urine flow out of the bladder. — Any hit to blood flow (pre-renal), poison or injury to the filter tissue itself (intrinsic), or blockage stopping urine from draining (post-renal) can trigger AKI.
2. Glomerular filtration rate—GFR—drops. This is the speed at which blood gets filtered. Normal is around 90–120 mL per minute. In AKI it can fall to half that or lower within hours. — Less blood reaches the filters, or the filters themselves are damaged and cannot clean blood anymore.
3. Serum creatinine and BUN start climbing in the blood. Creatinine is a waste product made by muscles every day; BUN comes from breaking down protein. Both are normally filtered out and dumped into urine. — The kidney cannot clear waste fast enough, so it backs up into the bloodstream like a clogged drain.
4. Urine output drops. Low urine output (Oliguria) means less than 400 mL in 24 hours (less than about one and a half cups). No urine output (Anuria) means less than 100 mL a day (almost no urine at all). — Either not enough blood is reaching the filter to make urine, or the tubule cells are dead and cannot process fluid anymore.
5. Potassium builds up in the blood (hyperkalemia). Normal potassium is 3.5–5.0 mEq/L; in AKI it can climb to 6, 7, or higher. Acid also builds up (metabolic too much acid in the blood (acidosis)), dropping blood pH below 7.35. — The kidney normally dumps potassium and hydrogen ions into urine every day. When the kidney stops working, both pile up in the blood.
6. Fluid overloads the body. Lungs fill with water (pulmonary swelling (edema)), causing shortness of breath, cough, and low oxygen. Legs and ankles swell (pitting swelling—you press the skin and a dent stays). Weight climbs fast—sometimes several pounds in one day. Blood pressure may spike because extra fluid increases the volume in the blood vessels. — The kidney cannot get rid of salt and water anymore. Every glass of water you drink, every bit of salt in food, stays in the body and has nowhere to go.

7. Severe waste buildup in the blood (Uremia) develops. Waste products (urea, creatinine, guanidine compounds, and many others) reach toxic levels. Patients feel sick to their stomach, itch all over, taste metal, become confused or drowsy (uremic encephalopathy), and may develop pericarditis (chest pain from inflammation of the sac around the heart). If potassium climbs high enough, the heart rhythm becomes dangerously unstable (peaked T waves on EKG, then wide QRS, then ventricular fibrillation and cardiac arrest). — Waste and electrolytes that should leave the body every day are now poisoning the brain, heart, and every other organ.
8. Without treatment—fixing the cause, giving IV fluids (if pre-renal), stopping nephrotoxic drugs, relieving any obstruction (catheter for BPH, stent or surgery for stone), and sometimes urgent dialysis—AKI can become irreversible. Patients may need long-term dialysis or develop chronic kidney disease. In the most severe cases, multi-organ failure and death occur within days. — The longer the kidney goes without blood flow or stays poisoned, the more tubule cells die. Once enough nephrons are lost, the kidney cannot recover. Meanwhile, high potassium can stop the heart, fluid overload can drown the lungs, and uremic toxins can shut down the brain.

Symptoms · what the patient feels, and why

- Decreased urine output (oliguria < 400 mL/day or no urine output (anuria) < 100 mL/day): The kidney's filter speed (glomerular filtration rate, or GFR) has dropped, so less blood gets cleaned and less urine is made. Think of it like a water filter that's clogged—less water comes out the other side.
- Swelling in legs, ankles, hands, or face (peripheral swelling (edema)): The kidney can't dump extra salt and water into the urine, so fluid builds up in the blood and leaks into the tissues. It's like overfilling a water balloon—fluid seeps out where it shouldn't be.
- Shortness of breath, wet cough, or crackles on lung exam (pulmonary swelling (edema)): Extra fluid backs up from the bloodstream into the tiny air sacs of the lungs (alveoli), making it hard to breathe. It's like trying to breathe through a wet sponge.
- Nausea, vomiting, and loss of appetite: Waste products (urea, creatinine, and other toxins) pile up in the blood because the kidney isn't cleaning them out. These uremic toxins irritate the stomach lining and trigger vomit signals in the brain.
- Itching all over (uremic itching (pruritus)): Uremic waste in the blood irritates nerve endings in the skin, making you itch. The exact reason isn't fully known, but the build-up of toxins is the trigger.
- Confusion, drowsiness, or altered mental status (uremic encephalopathy): Uremic toxins cross from the blood into the brain and poison the nerve cells (neurons), slowing down brain signals. It's like fog rolling into your thoughts.

- **Fatigue and muscle weakness:** Two things happen: the blood becomes too acidic (metabolic too much acid in the blood (acidosis)) because the kidney can't dump acid, and red blood cells drop (anemia) because the kidney stops making erythropoietin, the hormone that tells bone marrow to make red cells. Both mean less oxygen reaches your muscles, like a phone battery running low.
- **Full, swollen bladder you can feel in the lower belly (suprapubic fullness):** A blockage below the kidney—like an enlarged prostate, a kidney stone in the ureter, or a tumor—stops urine from leaving the bladder. Urine backs up and the bladder swells like a water balloon.
- **Peaked (tall, pointy) T waves or wide QRS on EKG:** Dangerously high potassium in the blood (hyperkalemia, potassium > 6.0–6.5 mEq/L) changes the electrical signals in the heart muscle. This can lead to a heart that stops beating (cardiac arrest).
- **Dark or tea-colored urine:** Myoglobin (a muscle protein) or hemoglobin (from broken red blood cells) leaks into the urine and turns it dark. This happens in rhabdomyolysis (muscle breakdown) or red blood cell destruction (hemolysis) (red cell destruction), both of which can cause AKI by clogging kidney filters.

Focused exam

- **Orthostatic low blood pressure (hypotension)** (blood pressure drops when the patient stands up), dry mouth and tongue, skin that stays tented when you pinch it: These signs mean the patient does not have enough fluid in the blood vessels (volume depletion). When blood volume is low, the kidneys do not get enough blood flow to filter properly, causing pre-renal AKI (kidney injury from low blood delivery before the kidney itself is damaged).
- **Crackling or popping sounds at the bottom of the lungs when you listen with a stethoscope, neck veins that bulge when the patient sits up (elevated jugular venous pressure), swelling in the legs and ankles:** These signs mean too much fluid is backing up in the body. This happens when the heart cannot pump effectively (heart failure) or when the kidneys can no longer remove fluid. Fluid buildup in the lungs (pulmonary swelling (edema)) can make breathing hard and may require urgent treatment.
- **You can feel a full, swollen bladder low in the belly when you press gently; the lower belly looks swollen and tight:** This means urine is trapped and cannot flow out (post-renal obstruction). The backed-up urine puts pressure on the kidneys and damages them fast. You must drain the bladder or unblock the tubes right away to prevent permanent kidney damage.

- White powdery crystals on the skin (uremic frost), flapping hand tremor when the patient holds arms out (asterixis), scratchy rubbing sound over the heart when you listen (pericardial friction rub): These are signs of severe waste buildup in the blood (uremia), which means waste products are building up to toxic levels because the kidneys have stopped working. The patient needs dialysis (a machine to clean the blood) immediately to remove the toxins and prevent death.

Diagnostics · every test answers a question

- Serum creatinine and blood urea nitrogen (BUN), compared to the patient's usual baseline → How much kidney filtering function has been lost?
- Urinalysis with microscopy (looking at the urine under a microscope) → Is the problem low blood flow to the kidney, damage inside the kidney, or a blockage downstream?
- Urine sodium and fractional excretion of sodium (FENa) → Is the kidney able to hold onto sodium like it should?
- Renal ultrasound (sound-wave pictures of the kidneys) → Are the kidneys swollen from backed-up urine? Is there a blockage?
- Electrolyte panel (blood test for potassium, bicarbonate, phosphate, calcium) → Are there dangerous electrolyte imbalances that could stop the heart or cause other organ failure?
- Electrocardiogram (EKG, heart tracing) if potassium is high or the patient has chest pain, palpitations, or weakness → Is high potassium changing the heart rhythm in a dangerous way?
- Complete blood count (CBC) with differential → Is there anemia from kidney failure, or signs of infection or bleeding?
- Creatine kinase (CK) if rhabdomyolysis (muscle breakdown) is suspected (history of trauma, seizure, prolonged immobility, or drug use) → Is muscle damage releasing toxins that are injuring the kidneys?

Treatment logic

- Stop all nephrotoxic drugs immediately: NSAIDs (ibuprofen, naproxen, ketorolac), aminoglycoside antibiotics (gentamicin, tobramycin), hold ACE inhibitors or ARBs, hold metformin → Removes the chemical insult that is damaging the kidneys. Stopping metformin prevents lactic acidosis (acid from oxygen-starved tissues) (dangerous buildup of lactic acid when the kidneys cannot clear it). Holding ACE inhibitors and ARBs allows kidney blood vessels to autoregulate (adjust blood flow) better during injury.

- Give intravenous isotonic crystalloid fluids (normal saline or lactated Ringer's solution) ONLY if the patient is volume-depleted (dry mucous membranes, low blood pressure, high heart rate) → Restores blood flow to the kidneys so they can filter again. Reverses pre-renal AKI by correcting low blood flow (perfusion) (low blood delivery). Creatinine should start dropping within 24 to 48 hours if this was the main problem.
- Give loop diuretics (furosemide, bumetanide) if the patient is fluid-overloaded with crackles in the lungs or swelling—do NOT use in pre-renal AKI → Removes excess fluid from the body by making the kidney excrete more urine. Relieves symptoms like shortness of breath from pulmonary edema (fluid flooding the lungs).
- Treat high potassium (hyperkalemia) in the following sequence: (1) intravenous calcium gluconate 1 gram over 2 to 3 minutes, (2) insulin 10 units IV with dextrose 25 grams IV, (3) inhaled albuterol 10 to 20 mg by nebulizer, (4) oral or rectal potassium binders (sodium polystyrene sulfonate, patiomer, sodium zirconium cyclosilicate), (5) urgent dialysis if refractory → Calcium gluconate stabilizes the heart cell membrane in 1 to 3 minutes to prevent arrhythmia (does not lower potassium). Insulin and albuterol shift potassium from blood into cells within 15 to 30 minutes, lowering serum potassium by 0.5 to 1 mEq/L temporarily (lasts 4 to 6 hours). Binders and dialysis physically remove potassium from the body (permanent removal).
- Relieve urinary obstruction urgently: place a Foley catheter if the bladder is blocked (urinary retention), place a nephrostomy tube (tube through the skin into the kidney) or ureteral stent (tube inside the ureter) if the ureters or kidney outlet are blocked → Restores urine drainage immediately. If obstruction was the only cause of AKI, creatinine often starts dropping within hours once the blockage is relieved. Prevents permanent scarring of the kidney from prolonged high pressure.
- Start renal replacement therapy (hemodialysis or continuous renal replacement therapy, CRRT) using the mnemonic AEIOU: Too much acid in the blood (Acidosis) (pH less than 7.1 that does not respond to bicarbonate), Electrolytes (hyperkalemia refractory to medical treatment), Ingestions (toxic alcohols like ethylene glycol or methanol, lithium), fluid Overload (pulmonary swelling (edema) not responding to diuretics), Severe waste buildup in the blood (Uremia) (encephalopathy, pericarditis, or bleeding from platelet dysfunction) → Mechanically does the kidney's job: removes waste products (urea, creatinine), corrects acid-base balance, removes excess fluid, and normalizes electrolytes. Buys time for the native kidneys to heal, which can take days to weeks.

- Adjust all medication doses for reduced kidney function using the calculated glomerular filtration rate (GFR or eGFR); avoid contrast dye unless absolutely necessary, and if needed, give intravenous isotonic saline before and after contrast to reduce risk → Prevents drug toxicity from medications that the kidney normally clears (antibiotics, antivirals, anticoagulants). Reduces the risk of contrast-induced AKI (contrast dye damages kidney tubules). Periprocedural hydration with normal saline is the only proven strategy to lower contrast-AKI risk.

Medications

Calcium gluconate (10%) · Membrane stabilizer for high potassium (hyperkalemia) (dangerously high potassium)

Does not lower potassium—instead it makes the heart's cell membranes less excitable (less likely to misfire) so a deadly heart rhythm does not start while other treatments move potassium into cells or remove it from the body.

1. High potassium makes the heart muscle cell's resting voltage too positive (depolarized), which brings it dangerously close to the firing threshold—like a hair-trigger gun.
2. Intravenous calcium raises the threshold potential (the voltage needed to trigger a heartbeat), moving it farther from that resting voltage.
3. The gap between resting and threshold widens.
4. The heart is now less likely to fire an abnormal, life-threatening beat while potassium is being lowered.

SAFETY WATCH

Never mix with bicarbonate in the same IV line—it will form a white chalky precipitate (solid particles that clog the line). · Give as slow IV push over 5–10 minutes. · Watch for extravasation (leaking into tissue around the vein)—calcium can cause tissue death (necrosis) (tissue death) if it escapes the vein. · Use with caution in patients on digoxin (a heart drug)—calcium can worsen digoxin toxicity and trigger dangerous rhythms.

Regular insulin + IV dextrose · Potassium-shifting therapy

Insulin drives potassium out of the blood and into cells within 15–30 minutes. Dextrose (sugar) is given at the same time to prevent low blood sugar (hypoglycemia) (dangerously low blood sugar).

1. Insulin activates sodium-potassium ATPase pumps on cell membranes—think of these as tiny doors that let potassium into cells and push sodium out.
2. Potassium is pulled into cells.

3. Blood potassium drops by 0.5–1.2 mEq/L within 30–60 minutes.
4. Effect lasts 4–6 hours.

SAFETY WATCH

Check blood glucose at 30 minutes, 1 hour, 2 hours, and 4–6 hours after giving the insulin.

· Older adults and people with kidney disease can develop delayed low blood sugar (hypoglycemia)—consider a continuous dextrose drip for 4–6 hours after the insulin dose. ·

Use 10 units regular insulin IV plus 25 grams dextrose (one amp of D50) if baseline glucose is above 250 mg/dL; otherwise use 50 grams dextrose (two amps D50) to prevent blood sugar from dropping too low. · If the patient is already hypoglycemic, skip the insulin or give dextrose first and recheck glucose before dosing insulin.

Sodium polystyrene sulfonate (Kayexalate) · Potassium binder (cation-exchange resin)

A powder or liquid given by mouth or rectally that trades sodium for potassium in the gut. As digested food moves through the intestines, the resin grabs potassium and releases sodium. The potassium is then removed from the body in stool.

1. The resin particles travel through the gut and reach the colon.
2. Each resin particle holds sodium; when it touches potassium in the gut, it swaps—releases sodium and traps potassium.
3. The trapped potassium leaves the body in stool.
4. Blood potassium drops by 0.5–1.0 mEq/L over 4–6 hours.

SAFETY WATCH

Never give by mouth to a patient who cannot have bowel movements or who has a bowel obstruction—risk of bowel tissue death (necrosis) (tissue death in the intestine). · Do NOT use sorbitol (a laxative often mixed with this drug) in older adults or post-surgical patients—it has caused fatal bowel tissue death (necrosis). · Watch for constipation—give a gentle laxative if needed. · Monitor for sodium overload and fluid retention—the resin adds sodium to the body. · Check magnesium and calcium—this drug can also bind and lower them.

Patiromer (Veltassa) · Potassium binder (newer oral polymer)

A powder mixed with water or soft food that binds potassium in the colon and removes it in stool. Works more gently and predictably than older binders. Exchanges calcium for potassium instead of sodium.

1. Patiromer particles swell in the colon and create binding sites for potassium.

2. Potassium in the gut fluid sticks to the polymer.
3. The polymer-potassium complex leaves the body in stool.
4. Blood potassium drops by 0.5–1.0 mEq/L over 7 hours; maximum effect at 48 hours with daily dosing.

SAFETY WATCH

Do NOT give with other oral medicines—patiomer can bind them and stop them from working. Give other drugs at least 3 hours before patiomer. · Do NOT heat or add to hot food—destroys the drug. · Monitor magnesium—patiomer can lower it. · Use with caution if the patient has a history of bowel obstruction or severe constipation. · Approved for chronic high potassium (hyperkalemia), not acute emergencies.

Sodium bicarbonate (IV) · Alkalinizing agent and potassium-shifting therapy

Raises blood pH (makes blood less acidic). When blood becomes less acidic, potassium moves from blood into cells in exchange for hydrogen ions. Most effective when the patient has metabolic acidosis (acid building up in the blood) (too much acid in the blood) from acute kidney injury.

1. Bicarbonate is given IV and buffers (neutralizes) excess hydrogen ions in the blood.
2. Blood pH rises (becomes less acidic).
3. Cells release hydrogen ions and take up potassium in exchange.
4. Blood potassium drops by 0.2–0.4 mEq/L within 30–60 minutes.

SAFETY WATCH

Do NOT mix with calcium in the same IV line—will precipitate. · Can cause volume overload (too much fluid in the blood) and worsening heart failure—use cautiously in patients with fluid overload. · Can cause paradoxical intracellular too much acid in the blood (acidosis) (cells become MORE acidic even though blood becomes less acidic) if given too fast. · Monitor for tetany (muscle spasms) if calcium is low—raising pH lowers ionized (active) calcium. · Check arterial blood gas before and after to confirm too much acid in the blood (acidosis) and guide dosing.

Albuterol (nebulized) · Beta-2 agonist bronchodilator used off-label for potassium shifting

Activates beta-2 receptors on cell membranes, which turn on sodium-potassium ATPase pumps. These pumps pull potassium into cells. Usually used to open airways in asthma, but high doses can lower potassium.

1. Albuterol binds to beta-2 receptors on muscle cell membranes.

2. This activates adenylyl cyclase inside the cell, raising cyclic AMP (a messenger molecule).
3. Cyclic AMP activates sodium-potassium ATPase pumps.
4. Potassium is pulled into cells from the blood.
5. Blood potassium drops by 0.5–1.0 mEq/L within 30–60 minutes.

SAFETY WATCH

Use high dose: 10–20 mg nebulized over 10 minutes (much higher than asthma dose). · Watch for a fast heart rate (tachycardia) (fast heart rate) and tremor—common side effects. · Use with caution in patients with heart disease or arrhythmias. · Less effective in patients on beta-blockers (drugs that block the same receptors albuterol activates). · Monitor heart rate and rhythm during and after treatment.

Furosemide (Lasix) · Loop diuretic (water pill)

Blocks sodium and chloride reabsorption in the loop of Henle (a part of the kidney tubule). This causes the kidney to dump sodium, chloride, water, and potassium into the urine. Only works if the kidneys still make urine.

1. Furosemide blocks the sodium-potassium-chloride cotransporter (NKCC2) on the luminal (urine-facing) side of tubule cells.
2. Sodium, potassium, and chloride stay in the urine instead of being pulled back into the blood.
3. Water follows sodium into the urine by osmosis (water follows salt).
4. Urine output increases; blood potassium drops if kidney function is preserved.

SAFETY WATCH

Monitor urine output—if no urine comes out after a dose, the drug is not working and higher doses or dialysis are needed. · Can cause volume depletion (too little fluid in the blood) and low blood pressure. · Can cause low potassium (hypokalemia) (low potassium) if used repeatedly after kidney function improves. · Monitor electrolytes (sodium, potassium, magnesium, calcium) closely. · Can cause ototoxicity (hearing damage) at very high IV doses. · Less effective in patients on NSAIDs (anti-inflammatory drugs like ibuprofen) because NSAIDs block prostaglandins that help furosemide work.

Hemodialysis · Renal replacement therapy (not a drug, but the definitive treatment)

Blood is pumped out of the body through a machine that filters waste, excess fluid, and potassium through a semi-permeable membrane (a filter with tiny holes). Clean blood is

returned to the body. The fastest and most effective way to remove potassium when kidneys have failed.

1. Blood flows from the patient through a catheter or fistula into the dialysis machine.
2. Blood passes on one side of a membrane; dialysate (a cleaning fluid) flows on the other side.
3. Potassium moves from blood (high concentration) to dialysate (low concentration) by diffusion (spreading out).
4. Excess fluid is pulled out by ultrafiltration (pressure difference).
5. Cleaned blood returns to the patient.
6. Potassium drops by 1.0–1.5 mEq/L per hour of dialysis.

SAFETY WATCH

Requires vascular access (a large IV catheter in the neck, chest, or groin, or a surgically created fistula in the arm). · Risk of bleeding during and after—heparin (a blood thinner) is used to keep blood from clotting in the machine. · Risk of low blood pressure (hypotension) (low blood pressure) during treatment—especially if too much fluid is removed too fast. · Risk of dialysis disequilibrium syndrome (brain swelling and confusion) if waste is removed too quickly in a patient who has never had dialysis before—start slowly. · Risk of infection at the catheter site. · Risk of air a traveling clot lodging in a vessel (embolism) (air bubble in the blood) if machine is not set up correctly. · Monitor potassium, calcium, phosphate, and bicarbonate closely during and after dialysis.

Red flags

- Potassium > 6.5 mEq/L or EKG changes (peaked T waves, wide QRS, sine wave)—heart can stop at any moment (cardiac arrest)
- Pulmonary edema (fluid flooding the lungs) (wet lungs, crackles, shortness of breath) not getting better with diuretics (water pills like furosemide)—dialysis needed urgently to pull fluid off
- Uremic encephalopathy (confusion, lethargy), pericarditis (chest pain with friction rub), or uremic bleeding (nose bleeds, bruising)—severe waste buildup in the blood (uremia) requiring urgent dialysis
- No urine output (Anuria) (no urine at all) plus a palpable bladder or hydronephrosis (swollen kidneys) on ultrasound—obstruction blocking urine flow; needs urgent catheter or nephrostomy tube
- Toxic ingestion (methanol, ethylene glycol, lithium, aspirin overdose)—dialyzable poison requiring emergent hemodialysis to remove it before it kills the patient

Nursing priorities

1. Strict I/O and daily weight — Best trend for volume status.
2. Recognize high potassium (hyperkalemia) signs early (EKG changes, weakness) — $K > 6.5$ is a code waiting to happen.
3. Hold all nephrotoxic drugs; check every medicine on the list — Ongoing insult delays recovery.
4. Assess for retention with bladder scan when low urine output (oliguria) is new — Post-renal cause is fastest to reverse.
5. Renal-dose all meds and reconcile with pharmacy — Toxic accumulation of drugs is common in AKI.

Sources

- KDIGO Clinical Practice Guideline for Acute Kidney Injury 2012
- American Society of Nephrology (ASN)
- UpToDate
- National Kidney Foundation (NKF)

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