

ESRD & Dialysis Complications

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THE STORY IN ONE SENTENCE

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“End-stage kidney disease means the kidneys have stopped working (below 15% function), so the person needs dialysis or a transplant to survive—watch for fluid overload, dangerous potassium spikes, access clots, and infections between treatments.”

Normal anatomy — the body's job

The kidneys are two fist-sized filters in your lower back that clean your blood 24/7, pull out waste and extra water, balance minerals and acid, and make hormones that control blood pressure, red blood cell production, and bone strength—every drop of your blood passes through them about 40 times a day.

BODY'S JOB

Each kidney has about a million tiny filters called nephrons. Blood flows into each nephron under pressure, water and small molecules (waste, sugar, salt, potassium) get pushed through a fine mesh (the glomerulus, a ball of capillaries), and then the tubule (a long twisty tube) decides what to keep (like sugar, the right amount of salt and water) and what to dump into urine (waste like urea and creatinine, extra potassium, extra acid). The kidneys also make three key hormones: erythropoietin (EPO, which tells your bone marrow to make red blood cells), renin (which raises blood pressure when it drops), and calcitriol (active vitamin D, which helps your gut absorb calcium and keeps bones strong). In 24 hours, your kidneys filter about 180 liters of fluid but only make about 1–2 liters of urine—the rest gets pulled back into your blood. This constant filtering keeps waste low, potassium in a safe range (3.5–5.0 mEq/L), acid-base balanced (pH 7.35–7.45), and fluid steady.

1. Blood enters the kidney through the renal artery, which branches into smaller and smaller vessels until it reaches the glomerulus (the tiny filter ball in each nephron).

2. The glomerulus lets water, waste (urea, creatinine), electrolytes (sodium, potassium, chloride), and small molecules pass through its walls into the tubule, but keeps large things like red blood cells and most proteins in the blood.
3. The tubule (proximal tubule, loop of Henle, distal tubule, collecting duct) fine-tunes what stays and what goes: it pulls back about 99% of the water, all the sugar, the sodium the body needs, and dumps waste, extra potassium, and acid into the urine.
4. Hormones are released: EPO is made in the kidney cortex when oxygen is low (to boost red blood cell production), renin is made when blood pressure drops (to raise it), and calcitriol is activated in the tubule cells (to help absorb calcium).
5. The final urine (1–2 liters a day) drains into the bladder; the cleaned blood flows out through the renal vein back to the heart.

What breaks

In end-stage renal disease, the nephrons (the tiny kidney filters) die off until fewer than 15% are left working—so the kidneys cannot clean waste, balance potassium or acid, pull off extra fluid, or make the hormones that control blood pressure, red blood cells, and bones.

CLINICAL TERM

ESRD is defined as glomerular filtration rate (GFR) below 15 mL/min/1.73 m² or the need for kidney replacement therapy (dialysis or transplant) to sustain life. The nephrons are destroyed by the underlying disease (most commonly diabetes, high blood pressure, glomerulonephritis, or polycystic kidney disease), and once GFR drops below 15%, the body cannot excrete nitrogenous waste (urea, creatinine), regulate potassium or acid-base balance, remove excess fluid, or produce adequate EPO, renin, or calcitriol. The result is severe waste buildup in the blood (uremia) (toxic buildup of waste), high potassium (hyperkalemia) (high potassium), metabolic acidosis (acid building up in the blood) (acid buildup), volume overload (fluid retention), anemia (low red blood cells), renal osteodystrophy (bone disease from high phosphorus and low calcium), and accelerated cardiovascular disease.

How the body compensates

The body tries to stabilize itself—breathing faster to blow off acid, bones releasing calcium to buffer acid, blood vessels constricting to raise pressure, thirst signaling to restore fluid—but

without working kidneys, every compensation backfires and makes the toxicity, fluid overload, and mineral imbalance worse.

1. {'what': 'Respiratory compensation for too much acid in the blood (acidosis)', 'how': 'When acid (hydrogen ions) builds up in the blood, chemoreceptors in the brainstem (the medulla) sense the low pH and signal the lungs to breathe faster and deeper (Kussmaul respirations) to blow off carbon dioxide (CO₂), which lowers acid—but this only partly offsets the metabolic acidosis (acid building up in the blood) and cannot fix the root problem.'}
2. {'what': 'Bone buffering of acid', 'how': 'To neutralize acid, the bones release calcium carbonate and calcium phosphate (which act as buffers)—this temporarily steadies the blood pH but weakens the skeleton over time, causing renal osteodystrophy (fragile bones, fractures, bone pain).'}}
3. {'what': 'Parathyroid gland activation (secondary hyperparathyroidism)', 'how': 'Low calcium (from poor gut absorption without calcitriol) and high phosphorus trigger the parathyroid glands to pour out parathyroid hormone (PTH), which pulls calcium from bone to raise serum calcium—but this makes bones even weaker and deposits calcium in blood vessels and soft tissues.'}
4. {'what': 'Renin-angiotensin-aldosterone system (RAAS) activation', 'how': 'Low blood flow to the kidneys (from fluid shifts or low pressure) triggers release of renin, which raises blood pressure and tells the adrenal glands to hold sodium and water (via aldosterone)—but in ESRD, this worsens fluid overload, raises blood pressure dangerously high, and strains the heart.'}
5. {'what': 'Thirst and fluid retention', 'how': 'Sensors in the hypothalamus detect concentrated blood and signal thirst—the person drinks, but the kidneys cannot dump the extra water, so fluid overload (edema, pulmonary edema (fluid flooding the lungs), high blood pressure (hypertension)) gets worse.'}
6. {'what': 'Sympathetic nervous system activation', 'how': 'To keep blood pressure and organ blood flow (perfusion) up, the sympathetic nerves fire more (releasing norepinephrine), causing vasoconstriction, faster heart rate, and more fluid retention—this strains the heart and accelerates cardiovascular disease (heart attacks, heart failure, arrhythmias).'}}

Domino effect

1. The nephrons die off (from diabetes, high blood pressure (hypertension), or other kidney disease) until fewer than 15% are working (GFR below 15 mL/min)—the kidneys cannot filter waste, balance minerals, control fluid, or make hormones. — The underlying disease (high blood sugar, high blood pressure, autoimmune attack, inherited cysts) damages the glomeruli (filter balls) and tubules (twisty tubes) over years; scar tissue replaces working nephrons, and once you lose 85% or more, the kidney can no longer sustain life on its own.

2. Waste (urea, creatinine) piles up in the blood (uremia), causing nausea, vomiting, loss of appetite, confusion, itching, a metallic taste, and eventually seizures or coma if untreated. — Urea and creatinine are breakdown products of protein and muscle; normally the kidneys dump them into urine, but when filtration fails, they accumulate in the blood and poison the brain, gut, and nerves—this is called uremic syndrome.
3. Potassium climbs (hyperkalemia) because the tubules cannot secrete it into urine—serum potassium above 6.0–6.5 mEq/L can cause dangerous heart rhythms (peaked T waves, wide QRS, sine wave, cardiac arrest). — Potassium controls the electrical charge across heart muscle cells; too much potassium makes the cells fire unpredictably, and the heart can stop suddenly—this is the most immediate life threat between dialysis sessions.
4. Acid builds up (metabolic too much acid in the blood (acidosis), low serum bicarbonate) because the kidneys cannot excrete hydrogen ions or make new bicarbonate—this causes fast breathing (Kussmaul), weakness, confusion, and breaks down bone and muscle. — The blood pH drops below 7.35; the body tries to compensate by breathing faster to blow off CO₂ and by pulling calcium carbonate from bone to buffer the acid, but this only buys time and weakens the skeleton.
5. Fluid overloads the body (volume overload) because the kidneys cannot pull off extra water—this causes swelling (edema in the legs, face), high blood pressure, and fluid in the lungs (pulmonary swelling (edema), shortness of breath, crackles on exam). — Every cup of water you drink or infuse stays in the bloodstream (no urine to dump it), so the heart has to pump against higher pressure, fluid leaks into the tissues and air sacs of the lungs, and the person feels like they are drowning.
6. Without erythropoietin (EPO) from the kidneys, the bone marrow does not make enough red blood cells—anemia (low hemoglobin) causes fatigue, pale skin, shortness of breath, dizziness, and worsens heart strain. — EPO is the hormone signal that tells the bone marrow to produce red blood cells; without it, hemoglobin drops (often below 10 g/dL), so oxygen delivery to tissues falls and the heart has to pump faster to compensate, which accelerates heart failure.
7. Without calcitriol (active vitamin D), calcium absorption drops, phosphorus rises (because kidneys cannot excrete it), and the parathyroid glands pour out PTH—this pulls calcium from bone (renal osteodystrophy), causing fractures, bone pain, and deposits calcium-phosphate in arteries and heart valves (vascular calcification). — Low calcium and high phosphorus signal the parathyroid glands to release parathyroid hormone (PTH); PTH leaches calcium from bone to raise serum calcium, but this weakens the skeleton and the high phosphorus binds calcium to form crystals that stiffen blood vessels—these calcified arteries raise the risk of heart attack and stroke.

8. Dialysis (hemodialysis 3 times a week) removes some waste, potassium, acid, and fluid—but only for a few hours, so between sessions everything creeps back up, and the patient lives in a cycle of buildup and cleanup. — Dialysis runs blood through an external filter (the dialyzer) that pulls out urea, creatinine, potassium, and water by diffusion and ultrafiltration—but it cannot fully replace the kidney's 24/7 work, cannot make hormones, and leaves gaps between sessions where toxins and fluid accumulate again.
9. During dialysis, rapid fluid removal (ultrafiltration) and electrolyte shifts cause low blood pressure (hypotension, dizziness, cramping), and sometimes the dialysate chemistry causes nausea, headache, or confusion (disequilibrium syndrome from rapid urea shifts). — Pulling off 2–4 liters of fluid in 4 hours drops blood volume faster than the body can refill it from the tissues, so blood pressure falls; rapid changes in blood osmolality (from dropping urea quickly) cause water to shift into brain cells, causing transient swelling and symptoms.
10. The vascular access (arteriovenous fistula or graft, or dialysis catheter) is the lifeline for dialysis—but it can clot (thrombosis, no thrill or a whooshing sound over a vessel (bruit) on exam), get infected (redness, pus, fever, bacteremia), or steal blood from the hand (ischemic steal syndrome, cold, painful fingers). — The fistula is a surgically created connection between an artery and vein, creating high flow that can handle repeated needle sticks—but high flow can clot if it slows, bacteria can enter through needle sites, and sometimes too much blood is shunted away from the hand, starving it of oxygen.
11. Even with dialysis, cardiovascular disease (heart attack, heart failure, arrhythmias, stroke) is the leading cause of death in ESRD patients—because chronic inflammation, vascular calcification, anemia, fluid overload, and high blood pressure (hypertension) damage the heart and arteries relentlessly. — Dialysis cleans waste but does not stop the cascade: uremic toxins inflame blood vessel walls, high phosphorus calcifies arteries, anemia strains the heart, fluid overload raises afterload, and chronic high blood pressure (hypertension) thickens the heart muscle (left ventricular hypertrophy)—all of these multiply the risk of fatal cardiovascular events.
12. Only a kidney transplant can restore near-normal kidney function and break the dialysis cycle—but it requires a donor organ, lifelong immunosuppression, and carries risks of rejection and infection; without transplant, dialysis is lifelong or the patient chooses palliative care. — A transplanted kidney can filter waste 24/7, make hormones, and balance minerals like a natural kidney—but the immune system sees the new kidney as foreign and will attack it unless you take anti-rejection drugs (immunosuppressants) every day, which raise infection risk; still, transplant offers better survival and quality of life than dialysis alone.

Symptoms · what the patient feels, and why

- Swelling in the legs, ankles, or face: The kidneys can no longer remove extra water from the blood, so fluid builds up in the body's tissues (like a sponge soaking up water it cannot wring out).
- Shortness of breath: Extra fluid backs up into the lungs (pulmonary swelling (edema)), making it harder for air sacs to fill with oxygen; or waste products in the blood (uremia) make the lungs inflamed and stiff.
- Feeling very tired and weak all the time (fatigue): The kidneys no longer make enough erythropoietin (the hormone that tells bone marrow to build red blood cells), so the blood cannot carry enough oxygen to muscles and the brain, causing anemia.
- Itching all over (uremic itching (pruritus)): Waste products like urea and phosphate pile up in the blood and irritate nerve endings in the skin; also, extra phosphate can form tiny crystals in the skin that trigger itching.
- Nausea, vomiting, or loss of appetite: High levels of urea and other toxins (uremia) irritate the stomach lining and trigger the brain's vomiting center (the area postrema in the brainstem), making food unappealing and causing sickness.
- Trouble thinking clearly or concentrating (brain fog): Toxins like urea cross into the brain and interfere with neurotransmitters (the brain's chemical messengers), slowing down signals between nerve cells; low red blood cells also mean less oxygen reaches the brain.
- Muscle cramps, especially during or after dialysis: Dialysis removes fluid quickly, which can drop blood volume and reduce blood flow to muscles; also, shifts in sodium and potassium (electrolytes) disrupt the electrical signals that tell muscles when to relax.
- Chest pain or irregular heartbeat (arrhythmia): High potassium (hyperkalemia) or sudden changes in calcium and magnesium during dialysis disrupt the heart's electrical rhythm; fluid overload can also strain the heart muscle (like overfilling a water balloon).
- Fever, redness, warmth, or pus at the dialysis access site (catheter or fistula): Bacteria enter through the access site (the tube or blood-vessel connection used for dialysis) and multiply in the bloodstream or surrounding tissue, triggering the immune system to send white blood cells and causing inflammation.
- Very high blood pressure that does not come down with medicine: The kidneys can no longer remove salt and water, so blood volume stays too high (like overfilling a garden hose); also, damaged kidneys release extra renin (a hormone that signals blood vessels to squeeze tight), raising pressure even more.

Focused exam

- Vascular access — feel for thrill (a buzzing vibration under your fingers) or listen with a stethoscope for a whooshing sound over a vessel (bruit) (a whooshing sound) in the arteriovenous fistula (AVF), graft, or catheter site: Dialysis cannot happen without working access. No thrill or a whooshing sound over a vessel (bruit) means the access is clotted shut, and the patient cannot get dialyzed — toxins and fluid build up fast and can kill within days.
- Lungs — listen with a stethoscope for crackles (wet popping sounds) at the bottom of the lungs; ask if they are short of breath: ESRD kidneys cannot remove extra fluid, so water backs up into the lungs like a flooded basement. This is pulmonary edema (fluid flooding the lungs) (fluid in the air sacs), and it stops oxygen from getting into the blood.
- Heart rhythm — check the pulse, look at the monitor for peaked T-waves (tall and narrow) or wide QRS (the main heartbeat spike stretched out) on the EKG: High potassium in ESRD (hyperkalemia) stops the heart's electrical system between dialysis sessions. Potassium above 6.5 mEq/L can cause sudden cardiac arrest — this is the kill-you-now danger.
- Fluid status — check weight gain since last dialysis, listen to lungs for crackles, check for pitting swelling (edema) (pressing leaves a dent) in the legs and over the tailbone (sacrum): ESRD patients cannot urinate or remove fluid on their own. Fluid builds up between dialysis sessions. Too much fluid causes high blood pressure, heart strain, and pulmonary edema (fluid flooding the lungs).
- Blood pressure — measure before and after dialysis, sitting and standing: Dialysis removes fluid fast, so blood pressure can crash during treatment (intradialytic low blood pressure (hypotension)). Between sessions, fluid overload drives pressure up and damages the heart and blood vessels.
- Access site — look for warmth, redness, drainage, or swelling around the AVF, graft, or catheter: Access infection spreads bacteria straight into the bloodstream (bacteremia) and is a top killer in ESRD. Fever plus a red, warm access site means draw blood cultures immediately before starting antibiotics.
- Mental status and energy — confusion, restlessness, or severe fatigue: Severe waste buildup in the blood (Uremia) (buildup of kidney waste poisons like urea and creatinine), anemia (low red blood cell count), and high potassium all fog the brain. This tells you whether dialysis is removing enough toxins (adequacy).

Diagnostics · every test answers a question

- Basic metabolic panel (BMP): sodium, potassium, chloride, bicarbonate, BUN, creatinine, glucose → Is potassium dangerously high? Is the patient acidotic (low bicarbonate)? How much uremic waste has piled up since last dialysis?

- Hemoglobin and hematocrit → Is the patient anemic? Are they producing enough red blood cells even with erythropoiesis-stimulating agent (ESA) treatment?
- Calcium, phosphate, and intact parathyroid hormone (PTH) → Is the bone disease of ESRD (renal osteodystrophy) under control? Is the parathyroid gland working too hard to keep calcium normal?
- Albumin → Is the patient malnourished or losing protein through dialysis?
- Ferritin and transferrin saturation (TSAT) → Does the patient have enough iron stores to make red blood cells, even with ESA treatment?
- Electrocardiogram (EKG) → Is high potassium damaging the heart's electrical system right now?
- Chest X-ray → Is there fluid overload in the lungs (pulmonary swelling (edema)) or heart enlargement (cardiomegaly) from chronic volume overload?
- Ultrasound of vascular access (Doppler flow study) → Is blood flowing fast enough through the access for effective dialysis? Is there a clot or narrowing (stenosis)?

Treatment logic

- Hemodialysis (typically 3 times per week, 4 hours per session) or peritoneal dialysis (daily exchanges at home) → The dialysis machine does what broken kidneys cannot: it pulls out extra water, potassium, urea, creatinine, and acid through a filter (artificial kidney). This reverses the toxic buildup (uremia) and fluid overload that happen between sessions.
- Create and maintain vascular access (arteriovenous fistula preferred, then arteriovenous graft, then tunneled dialysis catheter as last resort) → A good access (preferably an AVF, where a surgeon connects an artery directly to a vein in the arm) lets blood flow fast enough through the dialysis machine — at least 300–400 mL per minute — so toxins and fluid actually leave the body efficiently in 4 hours.
- Phosphate binders (calcium carbonate, sevelamer, lanthanum) taken with meals, plus calcitriol (active vitamin D) or calcimimetics (cinacalcet) to control parathyroid hormone → Broken kidneys cannot filter out phosphate or activate vitamin D, so phosphate rises, calcium drops, and the parathyroid gland works overtime (secondary hyperparathyroidism). This leaches calcium from bones (renal osteodystrophy) and deposits it in blood vessels and heart valves (vascular calcification). Phosphate binders trap phosphate in the gut so it leaves in stool instead of blood. Calcitriol replaces the missing active vitamin D. Calcimimetics tell the parathyroid gland to calm down.

- Erythropoiesis-stimulating agent (ESA: epoetin alfa or darbepoetin alfa injected weekly or every 2 weeks) plus intravenous iron supplementation → Broken kidneys stop making erythropoietin (the hormone that signals bone marrow to produce red blood cells), so red blood cell production drops and anemia develops. ESA replaces the missing erythropoietin. Iron (given IV because gut absorption is poor in ESRD) supplies the raw material to build hemoglobin inside red blood cells.
- Dietary restriction: potassium < 2–3 grams/day, sodium < 2 grams/day, fluid intake limited to urine output plus 1 liter per day (most ESRD patients make little to no urine) → Restricting what enters the body between dialysis sessions reduces the gap between toxic buildup and the next cleaning. This prevents dangerous potassium spikes (hyperkalemia), pulmonary edema (fluid flooding the lungs) from fluid overload, and high blood pressure from sodium and volume.
- Blood pressure control: optimize dry weight (target weight after dialysis removes all extra fluid), ACE inhibitor or ARB if residual kidney function remains, and other antihypertensives (beta-blockers, calcium channel blockers) as needed → High blood pressure in ESRD comes from fluid overload and damaged kidney blood pressure regulation. Lowering BP protects any remaining kidney tissue, slows heart failure and left ventricular hypertrophy (thickened heart muscle), and reduces stroke risk. Reaching dry weight (the weight where fluid is balanced) is the most important step.
- Kidney transplant evaluation and listing (for appropriate candidates) → A transplanted kidney restores hormone production (erythropoietin, calcitriol, renin), filters toxins continuously (not just 12 hours per week like dialysis), and dramatically improves survival and quality of life compared to staying on dialysis.

Medications

Phosphate binders (sevelamer, calcium acetate, lanthanum) · Phosphate binder

When your kidneys fail, they can't clear phosphorus from food, so it builds up in your blood and pulls calcium out of your bones and into your arteries, making them stiff like rock. Phosphate binders stick to phosphorus in your stomach and gut so it leaves in your stool instead of entering your blood.

1. You take the binder pill with each meal or snack
2. The binder molecule grabs onto phosphorus in the food you just ate
3. The bound phosphorus cannot cross into your blood
4. It leaves your body in your stool
5. Blood phosphorus drops toward a safer range (goal under 5.5 mg/dL)

SAFETY WATCH

Calcium-based binders (calcium acetate) can raise blood calcium too high if you also take vitamin D; your team will watch your calcium level. · Sevelamer can cause nausea, bloating, or constipation. · Lanthanum can rarely build up in bone over many years; long-term safety is still being studied. · Take exactly with meals — taking between meals wastes the dose.

Active vitamin D (calcitriol, paricalcitol) · Active vitamin D analog

Healthy kidneys turn regular vitamin D into its active form, which tells your gut to absorb calcium and tells your parathyroid glands (four tiny glands in your neck that control calcium and bone) to calm down. In ESRD your kidneys can't do that conversion, so your parathyroid glands overproduce parathyroid hormone (PTH), which eats away at your bones. Active vitamin D replaces what your kidneys can no longer make.

1. You take active vitamin D by mouth or through your dialysis line
2. It binds to vitamin D receptors on your parathyroid glands
3. The glands sense 'enough vitamin D' and make less PTH
4. Lower PTH means less calcium pulled from bone
5. Active vitamin D also tells your gut to absorb more calcium from food

SAFETY WATCH

Can raise blood calcium and phosphorus too high, leading to calcium deposits in blood vessels and heart valves. · Your team will check calcium, phosphorus, and PTH every month and adjust the dose. · Tell your doctor if you feel very tired, have muscle aches, or become confused (signs of high calcium).

Calcimimetics (cinacalcet, etelcalcetide) · Calcimimetic (calcium-sensing receptor activator)

Your parathyroid glands have a sensor that detects how much calcium is in your blood. In ESRD, low active vitamin D and high phosphorus make the glands think calcium is too low, so they pour out PTH, which pulls calcium from your bones. Calcimimetics trick the sensor into thinking blood calcium is higher than it really is, so the glands make less PTH.

1. You take cinacalcet by mouth or get etelcalcetide through your dialysis line
2. The drug binds to the calcium-sensing receptor on parathyroid cells
3. The receptor becomes more sensitive, as if more calcium is present
4. The glands release less PTH
5. Bone breakdown slows and blood calcium may drop slightly

SAFETY WATCH

Can drop blood calcium too low (hypocalcemia), causing muscle cramps, tingling, or in rare cases seizures. · Cinacalcet often causes nausea or vomiting; taking it with food helps. · Your team will check PTH and calcium regularly and adjust the dose. · Not used if your calcium is already low.

Erythropoiesis-stimulating agents (epoetin alfa, darbepoetin alfa) · ESA (erythropoietin analog)

Healthy kidneys make erythropoietin (EPO), a hormone that tells your bone marrow to build red blood cells. In ESRD your kidneys make almost none, so your marrow slows down and you become anemic (not enough red cells to carry oxygen). ESAs are lab-made versions of EPO that restart red-cell production.

1. You get an ESA injection under the skin or into your dialysis line, usually once a week or every two weeks
2. The ESA travels to your bone marrow and binds to EPO receptors on early red-cell precursors
3. Those precursor cells multiply and mature into red blood cells
4. More red cells enter your bloodstream
5. Your hemoglobin (the oxygen-carrying protein) rises toward the target (usually 10–11.5 g/dL)

SAFETY WATCH

Raising hemoglobin too high (above 11.5 g/dL) increases the risk of stroke, heart attack, and blood clots; guidelines recommend the lowest dose that avoids transfusion. · Can raise blood pressure. · Rarely, antibodies form against the ESA and destroy your own red cells (pure red-cell aplasia); stop the drug immediately if hemoglobin suddenly drops. · Does not work without enough iron; your team will monitor iron stores (ferritin and transferrin saturation).

Iron supplementation (oral ferrous sulfate or IV iron sucrose, ferric gluconate, iron dextran) · Iron supplement

Red blood cells need iron to make hemoglobin, the protein that carries oxygen. Dialysis patients lose iron through blood left in the dialysis tubing, frequent blood tests, and poor gut

absorption. Without enough iron, even ESAs can't build red cells effectively. Iron supplements refill your body's iron stores.

1. Oral iron is absorbed in the small intestine; IV iron is given directly into your bloodstream through your dialysis line
2. Iron binds to transferrin, a transport protein in your blood
3. Transferrin carries iron to your bone marrow
4. Marrow cells use the iron to build hemoglobin inside new red blood cells
5. Your iron stores (measured as ferritin) and the percentage of transferrin carrying iron (TSAT) both rise

SAFETY WATCH

Oral iron often causes constipation, black stools, nausea, or stomach upset; taking with food helps but may reduce absorption. · IV iron can rarely cause allergic reactions (flushing, itching, chest tightness); test doses are sometimes given first. · Too much iron (iron overload) can damage the heart and liver; your team monitors ferritin and TSAT and stops iron when stores are full (ferritin >500 ng/mL or TSAT >50%). · IV iron dextran has a higher allergy risk than newer forms (iron sucrose, ferric gluconate).

Antihypertensives (ACE inhibitors, ARBs, beta-blockers, calcium-channel blockers, diuretics in early ESRD) · Multiple classes targeting blood pressure

ESRD patients often have high blood pressure because damaged kidneys can't shed salt and water, and they release too much renin (a hormone that tightens blood vessels). High BP over time damages the heart and speeds up dialysis-access clotting. Blood-pressure medicines work in different ways—some relax vessels, some slow the heart, some block the kidney's renin system, and some (early on) help remove extra fluid.

1. ACE inhibitors and ARBs block the renin-angiotensin system, preventing blood vessels from tightening
2. Beta-blockers slow your heart rate and reduce how hard your heart squeezes
3. Calcium-channel blockers relax the muscle in vessel walls so they widen
4. Loop diuretics (like furosemide) can still help remove fluid if you have residual urine output (common early in dialysis)
5. Lower BP reduces strain on your heart, arteries, and dialysis access

SAFETY WATCH

ACE inhibitors and ARBs can raise potassium; your team will monitor levels closely. · Beta-blockers can mask low blood sugar if you have diabetes. · Calcium-channel blockers can cause ankle swelling and constipation. · Too-low BP (hypotension) during or after dialysis can cause dizziness, falls, or fainting; report symptoms immediately.

Potassium binders (sodium polystyrene sulfonate, patiomer, sodium zirconium cyclosilicate) · Potassium binder

Healthy kidneys clear potassium from your blood into urine. In ESRD, potassium builds up between dialysis sessions, and high levels (hyperkalemia) can stop your heart. Potassium binders work in your gut: they grab potassium from food and digestive juices and trap it so it leaves in your stool instead of entering your blood.

1. You take the binder by mouth (powder mixed with water or a suspension)
2. In your colon, the binder trades sodium or other ions for potassium
3. Potassium sticks to the binder
4. The bound potassium leaves your body in stool
5. Blood potassium drops toward a safer range (goal under 5.5 mEq/L)

SAFETY WATCH

Sodium polystyrene sulfonate (Kayexalate) can cause constipation or, rarely, serious bowel damage (necrosis) if it gets stuck; newer binders (patiomer, zirconium cyclosilicate) are safer. · Patiomer and zirconium cyclosilicate can cause mild GI upset (nausea, constipation, or diarrhea). · All binders can interfere with absorption of other medicines; take other drugs at least 3 hours before or after the binder. · Not a substitute for a low-potassium diet.

Immunizations (inactivated influenza, pneumococcal [PCV13 + PPSV23], hepatitis B, COVID-19, Tdap) · Vaccines

ESRD and dialysis weaken your immune system (dialysis filters and severe waste buildup in the blood (uremia) both blunt white-cell function), making infections more dangerous. Vaccines train your immune system by showing it a harmless piece or weakened form of a germ, so your body builds antibodies and memory cells that recognize and fight the real infection quickly if you encounter it. Dialysis patients often need higher or extra doses because their immune response is weaker.

1. The vaccine is injected into your muscle (usually your shoulder)

2. Immune cells called dendritic cells pick up the vaccine antigen and carry it to nearby lymph nodes
3. In the lymph nodes, B cells learn to make antibodies against that germ, and T cells learn to kill infected cells
4. Memory B and T cells stay in your body for months to years
5. If the real germ enters, memory cells wake up and attack it before you get very sick

SAFETY WATCH

Common side effects: sore arm, mild fever, fatigue for 1–2 days. · Hepatitis B vaccine requires a higher-dose series (40 µg instead of the usual 20 µg) and a blood test afterward to confirm you made enough antibodies; many dialysis patients need a booster. · Flu shot should be given every fall; use the inactivated (killed) shot, not the nasal spray. · Pneumococcal: you need both PCV13 and PPSV23 in sequence, then a PPSV23 booster years later. · COVID-19: dialysis patients should stay current with boosters per CDC guidance. · Tell your team if you have a history of severe allergic reaction to any vaccine.

Red flags

- Sudden severe shortness of breath or chest pain — may mean fluid in the lungs or a heart problem and needs emergency care right away.
- Fever with chills, especially with redness, warmth, or drainage at the dialysis catheter or fistula site — may be a blood infection (sepsis) and needs antibiotics and urgent evaluation.
- New confusion, severe headache, seizure, or trouble staying awake — may mean toxins are affecting the brain (uremic encephalopathy) or there is bleeding in the brain and needs immediate attention.
- Irregular heartbeat, palpitations, or feeling like the heart is racing or skipping beats — may be dangerously high potassium and needs an ECG and emergency treatment to protect the heart.

Nursing priorities

1. Assess dialysis access site for thrill, a whooshing sound over a vessel (bruit) (whooshing sound with stethoscope), redness, swelling, warmth, and drainage every 4 hours — The access is the patient's lifeline for dialysis; a clot stops treatment and needs emergency intervention, while infection can cause sepsis and death if not caught early

2. Weigh patient on same scale at same time daily and compare to dry weight (goal weight after dialysis removes extra fluid) — Each kilogram of weight gain equals one liter of retained fluid; tracking trends prevents pulmonary edema (fluid flooding the lungs) (fluid in lungs) and guides how much fluid to remove during next dialysis
3. Monitor vital signs and assess for fluid overload: listen to lung sounds for crackles, check jugular venous distension (bulging neck veins), and examine ankles for pitting swelling (edema) (indents when pressed) — Kidneys cannot remove excess fluid, so it backs up into lungs and tissues; early detection allows urgent dialysis before respiratory failure occurs
4. Protect the dialysis access arm: no blood pressure cuffs, no needle sticks, no tight clothing or jewelry, and post signs at bedside — Compression or injury damages the fragile blood vessels in the fistula or graft; once clotted or scarred, it cannot be used and requires surgery to create a new access
5. Monitor potassium levels and assess for high potassium (hyperkalemia) signs: muscle weakness, irregular heartbeat, or ECG changes (tall peaked T waves, wide QRS) — Kidneys normally remove potassium; when they fail, levels rise and can stop the heart suddenly (cardiac arrest); potassium above 6.5 mEq/L needs emergency dialysis
6. Administer phosphate binders with meals and monitor calcium and phosphorus lab values — High phosphorus binds with calcium and forms deposits in blood vessels and heart valves (vascular calcification), causing heart attacks and strokes; binders work only when taken with food
7. Teach and reinforce fluid restriction based on urine output plus 500 to 750 milliliters per day, and provide strategies like ice chips or frozen lemon wedges for thirst — Patients make little or no urine so cannot excrete extra fluid; drinking too much causes life-threatening pulmonary edema (fluid flooding the lungs) and forces more aggressive fluid removal during dialysis which drops blood pressure dangerously
8. Assess for anemia symptoms (fatigue, shortness of breath, pale skin) and ensure ESA and iron supplementation are given as prescribed — Kidneys make erythropoietin (hormone that signals bone marrow to produce red blood cells); without it, severe anemia develops, reducing oxygen delivery to all organs and worsening heart failure

Sources

- KDIGO Clinical Practice Guideline for CKD and Dialysis 2012 (updated 2020)
- National Kidney Foundation KDOQI Guidelines
- American Journal of Kidney Diseases (AJKD)
- UpToDate Nephrology

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