

Hyperkalemia

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THE STORY IN ONE SENTENCE

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“High potassium (Hyperkalemia) — the heart's electrical safety margin is vanishing.”

Normal anatomy — the body's job

Potassium is packed inside every cell at high concentration — about 140 units inside versus only 4 units outside in the blood. This steep difference creates a voltage gap across the cell membrane, like a charged battery. Heart muscle cells use this gap to fire and then reset after every beat. Your kidneys are the main exit door for potassium: they filter your blood and dump about 90 percent of the potassium you eat each day into your urine. The hormone aldosterone (made by the adrenal glands sitting on top of your kidneys) tells the kidney tubules to grab sodium back and kick potassium out. Blood flow through the kidneys and the amount of sodium delivered to the far end of the tubule also control how much potassium leaves. When this system works, blood potassium stays steady between 3.5 and 5.0 units, and the heart's voltage gap stays wide and safe.

BODY'S JOB

The wide voltage gap between inside and outside the cell is what lets the heart muscle fire cleanly and then recover fully before the next beat. Think of it like cocking a spring: the bigger the gap, the more reliable the snap. The kidneys act as the daily drain, matching potassium removal to potassium intake so the blood level stays flat. If you don't picture this balance first, the EKG changes and the crash make no sense.

1. You eat potassium in fruits, vegetables, beans, and salt substitutes every day.
2. Potassium is absorbed in your gut and enters your bloodstream.
3. About 90 percent is filtered by your kidneys and dumped into urine; the rest leaves in stool and sweat.

4. Aldosterone from your adrenal glands tells the kidney tubules to push potassium out and pull sodium back.
5. Blood potassium stays steady at 3.5–5.0 units, keeping the inside-outside gap wide.
6. Heart muscle cells use this gap to fire (depolarize) and then snap back (repolarize) in a clean, coordinated rhythm.

What breaks

High potassium (Hyperkalemia) happens when potassium piles up in the blood faster than the kidneys can remove it, or when cells burst open and dump their potassium load all at once. The kidneys are the bottleneck: if they fail or if medicines block aldosterone, potassium has nowhere to go. Cell breakdown floods the blood with potassium because cells hold 30 times more potassium than blood does. Either way, the rising blood potassium shrinks the voltage gap across the heart muscle membrane, and the heart's electrical system starts to misfire.

CLINICAL TERM

High potassium (Hyperkalemia) is serum potassium above 5.0 mEq/L; severe high potassium is above 6.5 mEq/L. The core mechanism is impaired renal potassium excretion, excessive potassium intake, or transcellular potassium shift from cells into blood. The rising extracellular potassium depolarizes the resting membrane potential (makes it less negative), narrowing the gap to the threshold potential and inactivating sodium channels, which slows conduction and can trigger fatal arrhythmias.

How the body compensates

The body has no direct way to bring potassium down fast on its own — there is no 'dump potassium now' reflex. Aldosterone release increases slightly, and the colon can excrete a bit more potassium in stool, but these responses are slow and weak. Meanwhile, vital signs (heart rate, blood pressure) can stay completely normal even as the EKG deteriorates, because blood pressure depends on vessel tone and pump volume, not membrane voltage. This silent period is the danger zone: the patient looks stable until the heart rhythm collapses in seconds. Clinicians must watch the EKG and the potassium trend, not just the vital signs, to catch the crash before it happens.

1. {'what': 'Aldosterone release increases slightly when potassium rises.', 'limit': 'This response is slow (hours to days) and only works if the kidneys and adrenal glands are healthy. It cannot match a sudden spike from cell lysis or missed dialysis.'}
2. {'what': 'The colon secretes a small amount of extra potassium into stool.', 'limit': 'Colonic excretion can rise from 10% to 30% of total potassium removal in severe kidney disease, but this takes days and cannot handle acute loads.'}

3. {'what': 'Insulin release (triggered by rising glucose after a meal) drives potassium into cells temporarily.', 'limit': 'This is a meal-to-meal buffer, not a rescue. In diabetic patients without insulin, this protective shift is lost.'}
4. {'what': 'Heart rate and blood pressure stay normal during the silent phase.', 'limit': 'Blood pressure depends on cardiac output and vascular resistance; membrane voltage does not directly control these. A patient can be normotensive and alert with a potassium of 7.5 mEq/L — right up until a fatal arrhythmia.'}
5. {'what': 'The EKG changes are the only reliable early-warning signal.', 'limit': 'Peaked T waves appear around 6.0–6.5 mEq/L, then PR prolongation, P-wave flattening, and QRS widening follow as potassium climbs above 7.0 mEq/L. These changes can progress to sine wave and cardiac arrest within minutes.'}
6. {'what': 'The crash point is a wide QRS (>0.12 sec) or any sine-wave pattern.', 'limit': 'Once the QRS widens or merges into a sine wave, the heart is one or two beats away from asystole or ventricular fibrillation. Calcium must be given immediately to stabilize the membrane, even before confirmatory labs return.'}

Domino effect

1. Rising blood potassium shrinks the voltage gap across the heart muscle cell membrane. — Normal resting potential is about -90 millivolts inside; threshold to fire is -70 millivolts — a safe 20-millivolt gap. High blood potassium makes the inside less negative (depolarizes it toward -80 or -75 millivolts), cutting the safety margin in half.
2. The smaller gap makes the heart cell fire unpredictably and sluggishly. — With only a 5- or 10-millivolt gap, tiny drifts in voltage can trigger a beat early or skip a beat entirely. At the same time, some of the fast sodium channels that create the sharp upstroke become stuck in an inactive state because the membrane never repolarizes fully, so the heartbeat loses speed and strength.
3. The T wave becomes tall and peaked first. — The T wave represents ventricular repolarization (the heart muscle resetting). When the membrane is already partway depolarized, repolarization happens faster and the T wave gets narrow and tall — this is often the first EKG clue, appearing around 6.0–6.5 mEq/L.
4. The P wave flattens and the PR interval stretches. — As potassium climbs above 6.5 mEq/L, the atria (top chambers) depolarize weakly because their thin muscle has fewer sodium channels to spare. The P wave shrinks or disappears. Conduction through the AV node (the relay station between atria and ventricles) slows, so the PR interval — the time from atrial start to ventricular start — gets longer.

5. The QRS complex widens into a thick, slurred shape. — The ventricles (bottom chambers) rely on the His-Purkinje system (fast highways of conducting fibers) to spread the signal quickly. High potassium slows conduction in these fibers, so the QRS — normally a sharp spike under 0.10 seconds — stretches to 0.12 seconds or more and loses its crisp peaks.
6. The EKG turns into a sine wave — a smooth, rolling curve with no clear P, QRS, or T. — When potassium rises above 8–9 mEq/L, depolarization and repolarization blur together. The membrane voltage drifts slowly up and down instead of snapping on and off, so the EKG looks like a gentle wave instead of sharp beats. This is pre-arrest.
7. The heart stops completely (asystole) or quivers uselessly (ventricular fibrillation). — With almost no voltage gap left, the cells cannot coordinate. Some freeze in a depolarized state (asystole — flat line), others fire randomly in tiny chaotic loops (ventricular fibrillation). Either way, the heart pumps no blood.
8. This entire cascade can happen in minutes if potassium spikes suddenly. — The heart muscle membrane responds to blood potassium in real time — there is no buffering step. A sudden release from cell lysis (crush injury, tumor lysis) or a rapid IV potassium infusion can push the level from 5.0 to 8.0 mEq/L in under an hour, and EKG changes follow within minutes.

Symptoms · what the patient feels, and why

- Often no symptoms at all until the level is dangerously high: Potassium quietly does its electrical work inside cells, so when blood levels creep up slowly, the body often doesn't ring alarm bells until the heart's rhythm is in trouble.
- Muscle weakness (especially in the legs and arms): Too much potassium outside muscle cells changes the voltage balance, making it harder for muscles to squeeze and relax properly — like a light switch stuck between on and off.
- Tingling or numbness (paresthesias), especially around the mouth or in the hands and feet: High potassium disturbs the electrical signals nerves use to talk to each other, so you can feel pins and needles or strange buzzing sensations even though nothing is touching your skin.
- Heart palpitations (feeling your heart skip, race, or pound): The heart's rhythm depends on potassium flowing in and out of heart muscle cells in a smooth pattern. When potassium in the blood is too high, the heart's electrical system misfires and beats irregularly.
- Nausea or vomiting: High potassium can irritate the stomach and slow gut movement, triggering waves of nausea or the urge to throw up.

- **Slow heart rate (bradycardia):** Excess potassium slows down the sinoatrial node (the heart's natural pacemaker in the right atrium) and blocks electrical signals from traveling smoothly through the atrioventricular node (the relay station between the top and bottom chambers), so the heart beats fewer times per minute — like a car engine sputtering because the spark plugs misfire.
- **Peaked (tall, narrow, tent-shaped) T waves on the EKG:** High potassium speeds up the repolarization phase (the reset stage when heart muscle cells recharge after each beat), making the T wave on the tracing taller and sharper — this is the earliest reliable warning sign the heart sees.
- **Widening of the QRS complex on the EKG (the heartbeat spike looks stretched out):** As potassium climbs even higher, the electrical signal takes longer to spread through the ventricles (the heart's main pumping chambers), stretching the QRS wave wider on the strip — a sign the heart is struggling to conduct electricity normally.
- **Flaccid paralysis (sudden limp weakness where muscles go completely soft):** When potassium rises very high, the voltage difference across muscle cell membranes collapses, and the muscles lose all ability to contract — like pulling the plug on a machine.
- **Shortness of breath or trouble breathing deeply:** If the diaphragm (the big breathing muscle below the lungs) and chest muscles weaken from high potassium, each breath becomes harder to pull in and push out.

Focused exam

- **Peaked T waves on EKG (tall, narrow, and pointy — like a tent):** This is the earliest sign that too much potassium outside heart cells is changing the voltage difference across the membrane. Normally, there is a big gap between the resting voltage and the firing threshold — like a safety buffer. When potassium climbs above 5.5 mEq/L, the outside-to-inside potassium ratio shrinks, the resting voltage drifts closer to the threshold, and the T wave spikes up tall and narrow. If you see peaked T waves with high potassium, the heart is already feeling the change and you need to act.
- **PR interval prolongation (the flat stretch before the QRS gets longer):** Signals from the atria (top chambers) to the ventricles (bottom chambers) travel through the AV node (the relay station). High potassium slows down the relay, stretching the PR interval beyond the normal 200 milliseconds. This means high potassium (hyperkalemia) is getting worse and the heart's wiring is struggling to pass the signal through.

- QRS widening (the tall spike that shows the ventricles squeezing gets fat and wide): When potassium rises high enough — usually above 7 mEq/L — the voltage gap shrinks so much that the ventricles depolarize (fire) slowly and unevenly. The normal QRS is under 120 milliseconds. A very wide QRS (greater than 120 ms) means the heart is close to stopping or flipping into ventricular fibrillation (chaotic quivering with no real pumping). This is a red-alert finding.
- P wave flattening or loss (the small bump before each QRS gets tiny or disappears): The P wave shows the atria firing. High potassium depolarizes atrial cells so much that they cannot generate a clean electrical wave anymore, so the P wave shrinks or vanishes. When the P wave is gone, potassium is usually above 7 mEq/L and the heart is in serious danger of stopping.
- Muscle weakness (patient says their legs feel heavy, arms feel floppy, or they cannot stand up easily): Skeletal muscle cells fire the same way heart cells do — by voltage difference across the membrane. High potassium makes muscle cells sit too close to their firing threshold, which paradoxically makes them tired and weak because they cannot reset properly between contractions. Weakness is less reliable than EKG changes but can be a clue, especially in chronic high potassium (hyperkalemia) or when potassium is above 6.5 mEq/L.
- Sine wave pattern on EKG (a smooth, wide, rolling wave with no clear P, QRS, or T): This is the final EKG pattern before the heart stops. The ventricles are depolarizing so slowly and chaotically that normal electrical structure is gone — everything blurs into one wide, rolling wave. Sine wave means potassium is critically high (often above 8 mEq/L) and cardiac arrest (asystole or ventricular fibrillation) is seconds to minutes away. This is a full code-blue emergency.

Diagnostics · every test answers a question

- Serum potassium → Is blood potassium truly elevated, and how high is it?
- 12-lead EKG → Is high potassium (hyperkalemia) causing dangerous changes in the heart's electrical system?
- Basic metabolic panel (BMP) or comprehensive metabolic panel (CMP) → Are the kidneys failing, is there too much acid in the blood (acidosis), and what is the blood sugar?
- Arterial blood gas (ABG) or venous blood gas (VBG) → Is too much acid in the blood (acidosis) driving potassium out of cells?
- Urine potassium and transtubular potassium gradient (TTKG) if the cause is unclear → Are the kidneys excreting potassium appropriately, or are they holding onto it?
- Medication review → Is a drug blocking potassium excretion or releasing potassium from cells?

- Creatine kinase (CK) if rhabdomyolysis is suspected → Is muscle breakdown releasing potassium into the blood?
- Repeat potassium in 1-2 hours after emergency treatment → Did the treatment shift or remove potassium, and is it safe to slow down or stop?

Treatment logic

- Calcium gluconate 1-2 g IV (or calcium chloride 0.5-1 g IV) over 2-3 minutes → Calcium raises the threshold voltage that heart cells need to reach before they fire. Normally, the resting voltage is around -90 millivolts and the threshold is around -70 millivolts. High potassium brings the resting voltage up closer to threshold — dangerous. Calcium pushes the threshold higher, widening the safety gap again and stabilizing the heart membrane within 1-3 minutes. This protects the heart from arrhythmias (like ventricular fibrillation or asystole) while you work on lowering potassium.
- Regular insulin 10 units IV with dextrose 25 g (one ampule of D50) IV push → Insulin activates sodium-potassium pumps on muscle and fat cell membranes. These pumps pull potassium from blood into cells in exchange for sodium. This lowers blood potassium by 0.5-1.2 mEq/L within 15-30 minutes and lasts 4-6 hours. Dextrose (sugar) prevents low blood sugar (hypoglycemia) (low blood sugar, below 70 mg/dL) from the insulin — without it, the patient could pass out or seize.
- Albuterol 10-20 mg nebulized (or 0.5 mg IV if available) → Albuterol activates beta-2 receptors on muscle cell membranes, which turn on sodium-potassium pumps and pull potassium into cells. This lowers blood potassium by 0.5-1.0 mEq/L within 30 minutes. The effect adds to insulin — using both together is more powerful than either alone.
- Sodium bicarbonate 50-100 mEq IV over 5-10 minutes (only if metabolic acidosis (acid building up in the blood) is present, pH below 7.2) → Bicarbonate neutralizes acid in the blood. When blood pH rises, hydrogen ions (acid) move out of cells and potassium moves back in to keep the electrical charge balanced across the membrane. This lowers blood potassium by 0.5-1.0 mEq/L over 30-60 minutes. Works best when too much acid in the blood (acidosis) is the main driver of high potassium (hyperkalemia) — for example, in diabetic ketoacidosis (the acid crisis of missing insulin) or kidney failure with severe too much acid in the blood.
- Loop diuretic (furosemide 40-80 mg IV, or bumetanide 1-2 mg IV) if the kidneys are still making urine → Loop diuretics block the sodium-potassium-chloride transporter in the thick ascending limb of the loop of Henle (a part of the kidney tubule). This stops sodium from being reabsorbed, which increases urine flow and forces the distal tubule (the next segment) to excrete more potassium into urine. This removes potassium from the body and lowers blood potassium over 2-4 hours.

- Sodium polystyrene sulfonate (Kayexalate) 15–30 g oral or rectal (if the GI tract works and high potassium (hyperkalemia) is not immediately life-threatening) → This resin binds potassium in the gut, especially in the colon, and trades it for sodium. The potassium-resin complex then leaves the body in stool. This removes potassium from the body, lowering blood potassium by 0.5–1.0 mEq/L over 4–6 hours.
- Hemodialysis (if potassium is above 6.5 mEq/L with EKG changes, or refractory high potassium (hyperkalemia) that does not respond to medical treatment, or acute kidney injury / end-stage kidney disease with no urine output) → Dialysis physically removes potassium from blood by running it across a semipermeable membrane that lets potassium pass into dialysate fluid, which is then thrown away. This lowers potassium by 1–2 mEq/L per hour and can continue as long as needed. This is the only treatment that truly removes large amounts of potassium sustainably when the kidneys cannot do the job.

Medications

Calcium gluconate · Cardiac membrane stabilizer

Calcium gluconate protects the heart RIGHT NOW. High potassium makes heart muscle cells jumpy and likely to fire at the wrong time, which can cause deadly rhythm problems. Calcium raises the trigger point — it makes each cell need MORE of a signal to fire, so the heart settles back into a safer zone. This does NOT lower potassium at all; it just buys time while you fix the real problem.

1. Calcium ions sit on the outside of heart muscle cells.
2. They change the electrical charge difference across the cell membrane.
3. This raises the voltage needed to make the cell fire (the threshold goes up).
4. Now the gap between resting voltage and firing voltage is wider again, so the heart beats steadily instead of chaotically.
5. Effect starts in 1–3 minutes and lasts 30–60 minutes.

SAFETY WATCH

Give slowly over 2–3 minutes to avoid heart rhythm glitches. · Watch the heart monitor during the infusion. · Use with caution in patients on digoxin (a heart drug) because calcium can make digoxin toxicity worse. · Repeat the dose if dangerous EKG changes (wide QRS, peaked T waves) do not go away after 5–10 minutes.

Regular insulin (with dextrose) · Cellular potassium-shift agent

Insulin pushes potassium FROM the blood INTO cells, where it is stored safely. Insulin turns on pumps in the cell membrane (called sodium-potassium ATPase pumps) that pull potassium

inside. We give dextrose (sugar) at the same time so the insulin does not drop blood sugar too low.

1. Insulin binds to receptors on the surface of muscle and other cells.
2. This activates sodium-potassium ATPase pumps in the cell membrane.
3. The pumps move potassium from blood into the cell interior.
4. Blood potassium drops by 0.5–1.2 mEq/L within 15–30 minutes.
5. Effect peaks around 60 minutes and lasts 4–6 hours.

SAFETY WATCH

Always give dextrose (usually 25 grams, one amp of D50) WITH the insulin to prevent low blood sugar (hypoglycemia). · Check blood sugar 30 minutes and 60 minutes after the dose. · Watch for low blood sugar symptoms: shaking, sweating, confusion, fast heartbeat. · Use a lower dextrose dose (or skip it) if blood sugar is already very high (over 250 mg/dL), but still monitor closely.

Albuterol (inhaled) · Beta-2 agonist (cellular potassium-shift agent)

Albuterol is a breathing medicine that also moves potassium into cells. It activates beta-2 receptors on muscle cells, which turn on the same sodium-potassium pumps that insulin uses. This gives you a second way to shift potassium quickly, and it works ALONGSIDE insulin (the effects add together).

1. Albuterol binds to beta-2 receptors on muscle cell surfaces.
2. This triggers a chemical messenger inside the cell (called cAMP) that activates sodium-potassium ATPase pumps.
3. The pumps pull potassium from blood into the cell.
4. Blood potassium drops by 0.5–1.0 mEq/L within 30 minutes.
5. Effect lasts 2–4 hours.

SAFETY WATCH

Can cause fast heart rate, shaking hands, and feeling jittery. · Use caution in patients with heart disease or abnormal heart rhythms. · Give a high dose by nebulizer (10–20 mg) — this is much more than the usual asthma dose. · Watch the heart monitor during treatment.

Sodium bicarbonate · Alkalinizing agent (cellular potassium-shift agent)

Sodium bicarbonate raises blood pH (makes the blood less acidic). When blood becomes less acidic, potassium moves from blood into cells in exchange for hydrogen ions. This works ONLY

if the patient has metabolic acidosis (acid building up in the blood) (low blood pH from too much acid, NOT from lung problems). If pH is normal, bicarbonate does not help much.

1. Bicarbonate neutralizes excess hydrogen ions (acid) in the blood.
2. Blood pH rises (becomes less acidic).
3. Cells release hydrogen ions into the blood and take in potassium in exchange.
4. Blood potassium drops by 0.4–0.6 mEq/L over 1–2 hours IF too much acid in the blood (acidosis) was present.
5. If pH is already normal, the effect is minimal.

SAFETY WATCH

Can cause fluid overload (too much water in the body) because it contains a lot of sodium. · Can cause metabolic alkalosis (the blood turning too alkaline) (blood becomes TOO alkaline) if you give too much. · Use cautiously in patients with heart failure or kidney failure who cannot handle extra fluid. · Check blood pH and electrolytes after giving it.

Furosemide (Lasix) · Loop diuretic (potassium eliminator)

Furosemide makes the kidneys dump extra potassium into the urine and flush it out of the body. It blocks a transporter in the kidney tubules (the tiny pipes that make urine) so salt, water, and potassium all leave the body together. This **ONLY** works if the kidneys still function — it does NOT help if kidneys are badly damaged.

1. Furosemide blocks the sodium-potassium-chloride cotransporter (NKCC2) in the thick ascending limb of the loop of Henle.
2. Sodium, potassium, and chloride stay in the urine instead of being pulled back into the blood.
3. More water follows the salt out, so urine volume increases.
4. Potassium is lost in the urine, and blood potassium drops over 4–6 hours.
5. Effect depends on kidney function — works best when GFR is above 30 mL/min.

SAFETY WATCH

Can cause dehydration and low blood pressure. · Can cause low levels of sodium, magnesium, and calcium. · Check kidney function and electrolytes regularly. · Not helpful in anuric patients (those making little or no urine) — use dialysis instead.

Patiromer (Veltassa) · Oral potassium binder (potassium eliminator)

Patiromer is a powder you mix with water and swallow. It sits in the intestines and trades calcium for potassium — it grabs potassium from the gut (including potassium that the body

dumps into the gut from the blood) and holds onto it, so the potassium leaves the body in stool instead of being absorbed. This is a SLOW fix for chronic (long-term) high potassium, not for emergencies.

1. Patiromer particles release calcium ions.
2. They bind to potassium ions in the intestine in exchange.
3. Potassium sticks to the patiromer and cannot be absorbed into the blood.
4. The patiromer-potassium complex leaves the body in stool.
5. Blood potassium drops by 0.5–1.0 mEq/L over 7 hours to several days.

SAFETY WATCH

Can cause constipation or diarrhea, stomach pain, nausea. · Can cause low magnesium — check magnesium levels monthly. · Do NOT take within 3 hours of other oral medicines because it can bind them and stop them from working. · Mix only with water or apple juice, never with orange juice or cranberry juice (those are high in potassium).

Sodium zirconium cyclosilicate (Lokelma) · Oral potassium binder (potassium eliminator)

Sodium zirconium cyclosilicate is another powder you mix and swallow. It traps potassium in the intestines by pulling it into tiny pores in its structure, like a super-fine sponge. The potassium stays locked inside and leaves the body in stool. It works faster than patiromer (starts in 1 hour) but is still not fast enough for true emergencies.

1. Sodium zirconium cyclosilicate particles have tiny pores that selectively capture potassium ions.
2. Potassium in the intestine (from food and from body secretions) gets trapped in the pores.
3. The drug-potassium complex is NOT absorbed and leaves the body in stool.
4. Blood potassium begins to drop within 1 hour and continues to fall for up to 48 hours.

SAFETY WATCH

Can cause swelling (edema) because it contains sodium. · Use cautiously in patients with heart failure. · Can cause constipation, diarrhea, or nausea. · Do NOT heat it or add it to heated foods or liquids (heat destroys the drug).

Hemodialysis · Renal replacement therapy (potassium eliminator)

Hemodialysis is a machine treatment that cleans the blood when kidneys cannot. Blood is pumped out of the body through a tube, passed alongside a special cleaning fluid (dialysate) across a filter membrane, and returned to the body. Potassium crosses from the blood (high

concentration) into the dialysate (low or zero potassium), and the machine throws the dirty fluid away. This is the MOST powerful and reliable way to remove potassium.

1. Blood flows through a dialysis catheter or fistula into the dialysis machine.
2. Inside the machine, blood runs along one side of a semipermeable membrane.
3. Dialysate (cleaning fluid with little or no potassium) flows along the other side.
4. Potassium diffuses from blood into dialysate down its concentration gradient.
5. The machine discards the potassium-rich dialysate, and cleaned blood returns to the patient.
6. Blood potassium can drop 1-2 mEq/L per hour during dialysis.

SAFETY WATCH

Requires vascular access (a large IV catheter, fistula, or graft). · Can cause low blood pressure, muscle cramps, or infection at the catheter site. · Removes other electrolytes and medications along with potassium — monitor closely. · Reserve for patients with kidney failure, very high potassium (over 6.5–7.0 mEq/L), or potassium that does not respond to medicines.

Red flags

- EKG changes (peaked T waves, wide QRS, flat P waves, or any new rhythm problem) — even if the patient feels fine
- Muscle weakness so severe the patient cannot stand or move arms and legs normally
- Heart rate below 50 or above 120, or any irregular, skipped beats
- Potassium level 6.5 mEq/L or higher (immediate treatment needed)
- Sudden chest pain, shortness of breath, or feeling faint (may signal a dangerous heart rhythm about to start)

Nursing priorities

1. Place the patient on continuous cardiac monitoring and obtain a 12-lead EKG immediately. — High potassium (Hyperkalemia) stops the heart before it causes other symptoms; the EKG shows peaked T waves, widened QRS complexes (the big spike on the rhythm strip), or sine waves (a pattern right before the heart stops) so we know how urgent treatment is.

2. Establish large-bore IV access and prepare calcium gluconate 1 to 2 grams for immediate IV push if EKG changes are present. — Calcium stabilizes the heart muscle cell membrane (outer layer of the cell) within minutes and prevents fatal arrhythmias like ventricular fibrillation (chaotic deadly heart rhythm); it does not lower potassium but buys time for other treatments to work.
3. Administer insulin 10 units IV with dextrose 25 grams IV (one ampule of D50) as ordered. — Insulin pushes potassium from the blood back into cells by activating sodium-potassium pumps (tiny gates in cell membranes); dextrose prevents low blood sugar (hypoglycemia) from the insulin; potassium drops within 15 to 30 minutes.
4. Give albuterol 10 to 20 milligrams by nebulizer (breathing treatment) as ordered. — Albuterol is a beta-2 agonist (medicine that activates cell receptors) that also drives potassium into cells; it works in 30 minutes and adds extra potassium-lowering power alongside insulin.
5. Check blood glucose before and 30 minutes after giving insulin and dextrose; treat low blood sugar (hypoglycemia) immediately if glucose drops below 70. — Insulin can drop blood sugar dangerously low; low blood sugar (hypoglycemia) causes confusion, seizures, or loss of consciousness, which can look like worsening high potassium (hyperkalemia); catching it early prevents brain injury.
6. Hold all potassium supplements, potassium-sparing diuretics, ACE inhibitors, and ARBs (angiotensin receptor blockers) until the provider reviews them. — Continuing these medicines keeps adding potassium or blocking the kidneys from removing it; stopping them prevents the level from climbing higher while we treat.
7. Monitor urine output every hour and notify the provider immediately if output is less than 30 milliliters per hour or the patient is anuric (making no urine). — Potassium leaves the body mainly through urine; if the kidneys are not making urine, medicines like furosemide will not work and the patient needs urgent hemodialysis to remove potassium.
8. Prepare for emergent hemodialysis if potassium is above 6.5 with EKG changes, the patient has acute kidney injury or end-stage renal disease, or medical treatments are not lowering the level. — Hemodialysis is the only treatment that actually removes potassium from the body when kidneys cannot; it is life-saving when the heart is in danger and works faster than waiting for kidneys to recover.

Sources

- KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease
- AHA/ACC 2020 Guideline for the Management of Patients With Valvular Heart Disease
- American Heart Association ACLS Provider Manual

- UpToDate Clinical Guidelines

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