

Rhabdomyolysis

Muscle cells have broken open and are dumping their insides — myoglobin (the oxygen-carrying protein in muscle), potassium, phosphate, and creatine kinase (CK, an enzyme) — into the bloodstream.

THE STORY IN ONE SENTENCE

Muscle cells have broken open and are dumping their insides — myoglobin (the oxygen-carrying protein in muscle), potassium, phosphate, and creatine kinase (CK, an enzyme) — into the bloodstream.

“Rhabdomyolysis means muscle is breaking down and spilling its guts into the blood; the kidneys are the organ most at risk of getting clogged and failing.”

Normal anatomy — the body's job

Muscle cells are wrapped in a thin, tough skin called the sarcolemma that keeps all the cell's contents — myoglobin (the oxygen-carrier), potassium, phosphate, calcium, and enzymes like creatine kinase (CK) — locked inside. The kidneys have tiny filters (glomeruli and tubules) that can handle small amounts of myoglobin and flush it out in urine. Keep that picture in your head, because the weird findings in rhabdomyolysis only make sense as a change from this normal state.

BODY'S JOB

The sarcolemma's job is to hold the muscle cell together and keep its high-energy contents inside so the muscle can contract and relax smoothly. The kidney's job is to filter waste and small proteins out of the blood and turn them into urine. When a tiny bit of myoglobin leaks from normal muscle turnover, the kidneys clear it without trouble. If you don't know this normal job first, the weird findings in rhabdomyolysis seem random and confusing.

1. Picture a muscle cell as a tiny sausage wrapped in plastic wrap (the sarcolemma).
2. Inside that cell, myoglobin holds onto oxygen so the muscle can burn fuel and move; potassium, phosphate, and calcium sit at carefully balanced levels; and enzymes like CK help the cell do its work.
3. The sarcolemma keeps all of this inside, like a sealed bag.

4. When you move, a few cells naturally wear out and release a small puff of myoglobin into the blood.
5. That myoglobin flows to the kidneys, passes through the glomerulus (the first filter, like a screen), and gets flushed out in the tubules (the tiny tubes that make urine).
6. Because the load is small and urine flow is good, nothing clogs, and the kidney stays healthy.

What breaks

The sarcolemma (the muscle cell's wrapper) tears open, and the cell's insides pour out into the blood and tissue spaces. Usually one upstream cause — trauma, crush injury, being stuck in one position too long (like a fall or coma), a seizure, extreme exercise, statin drugs, alcohol, street drugs (cocaine, heroin, amphetamines), severe heat (hyperthermia) or cold (hypothermia), an electrolyte imbalance (very low potassium or phosphate), or compartment syndrome (pressure building up and choking off blood flow inside a muscle compartment) — explains why the sarcolemma failed. That single broken thing upstream causes all the weird findings you see downstream.

CLINICAL TERM

Sarcolemmal disruption with release of intracellular muscle contents (myoglobin, CK, potassium, phosphate, purines) into the circulation, triggered by direct trauma, starved blood flow (ischemia), metabolic stress, or toxin-mediated injury.

How the body compensates

The body tries to keep vital signs steady and protect the heart and kidneys while muscle is breaking down. Vital signs can look okay for a while, even as dangerous electrolyte shifts and kidney injury are brewing underneath. The job is to see the crash point coming before the patient tips over the edge.

1. {'step': 'The body keeps blood pressure and heart rate steady at first', 'how': 'The kidneys hold onto sodium and water to keep blood volume up, and the heart pumps harder to deliver oxygen to the rest of the body, even though muscle is dying and toxins are building.'}
2. {'step': 'The kidneys try to flush myoglobin and waste', 'how': 'If the patient is drinking or getting IV fluids, the kidneys increase urine flow to wash myoglobin out before it clogs the tubules. This works only if fluids come fast and early.'}
3. {'step': 'The body buffers rising acid and potassium', 'how': 'Bones and lungs try to soak up extra hydrogen ions (acidosis), and cells try to pull potassium back inside to keep the blood level from spiking too high and stopping the heart.'}

4. {'step': 'This compensation hides how sick the patient really is', 'how': 'Vital signs stay stable, and the patient may feel weak but not collapsing — this is the dangerous middle, because it lets the team miss how close the patient is to a crash.'}
5. {'step': 'The crash point to watch for', 'how': 'The worst outcomes are hyperkalemic cardiac arrest (potassium gets so high the heart stops), severe acute kidney injury needing dialysis (myoglobin clogs the kidney filters), compartment syndrome (pressure builds up and kills more muscle), or DIC (disseminated intravascular coagulation, a blood clotting disaster) in very severe cases.'}
6. {'step': 'Death rate is high if acute kidney injury happens', 'how': 'Once the kidneys fail, toxins pile up, potassium spikes, and the heart and brain are in immediate danger. The window to reverse it closes fast.'}
7. {'step': 'Watch the trend, not just the snapshot', 'how': 'Look at whether CK, potassium, creatinine, and urine output are getting better or worse over hours, not just one moment in time. A stable patient on a downhill slope is not really stable.'}

Domino effect

1. The sarcolemma (muscle cell wrapper) tears open — A specific upstream cause — trauma, crush, prolonged pressure, seizure, extreme exercise, statin, alcohol, cocaine, heroin, severe heat or cold, electrolyte imbalance, or compartment syndrome — damages the sarcolemma so it can no longer hold the cell's insides in.
2. Muscle contents flood into the blood and tissues — Once the sarcolemma breaks, the muscle cell dumps myoglobin (oxygen-carrier), creatine kinase (CK, an enzyme), potassium, phosphate, calcium, and purines (waste) into the bloodstream and the space between cells, causing pain, swelling, and weakness at the site of injury.
3. Urine turns dark cola or tea color — Myoglobin floods the blood, filters through the kidneys, and pours into the urine, turning it dark brown. A urine dipstick shows positive for blood, but when you look under a microscope, there are no red blood cells — it is myoglobin, not bleeding.
4. Blood tests show sky-high CK, high potassium, high phosphate, low calcium, and rising acid — CK (creatinine kinase) spikes into the thousands or even hundreds of thousands because muscle cells are bursting open. Potassium floods out of dead muscle, raising blood potassium (hyperkalemia) and threatening the heart. Phosphate pours out, raising blood phosphate (hyperphosphatemia). Calcium gets trapped inside dying muscle, so blood calcium drops (hypocalcemia). Dying cells release acids and purines, raising blood acid (metabolic too much acid in the blood (acidosis)) and uric acid.

5. Myoglobin clogs the kidney tubules like sludge in a pipe — Myoglobin is a big, sticky protein. When it floods the kidney tubules, especially in acidic, low-flow urine, it clumps together and forms casts (solid plugs) that block the tiny filters. This triggers acute tubular tissue death (necrosis) (the kidney tubules die) and acute kidney injury.
6. The kidneys start to fail, and creatinine rises — With tubules clogged and dying, the kidneys cannot filter waste or make urine. Blood creatinine (a waste product) climbs, urine output drops, and toxins pile up. Potassium rises even higher, threatening cardiac arrest.
7. The heart is at risk of a fatal rhythm — High potassium (hyperkalemia) changes the heart's electrical signals. The ECG shows tall, peaked T waves, then a wide QRS, and finally the heart can stop in a fatal rhythm (sine wave, asystole, or ventricular fibrillation) if potassium is not brought down fast.
8. The body tries to survive, not hurt itself — The body is not attacking itself. High potassium (Hyperkalemia) is the cost of keeping dying cells from totally collapsing. Too much acid in the blood (Acidosis) is a signal that cells are screaming for oxygen. Low calcium (Hypocalcemia) is the body's attempt to keep calcium from flooding back into damaged muscle and causing more harm. Each finding is the body buying time, waiting for help.
9. Trace it all back to one broken thing, then fix it — When you see muscle pain, swelling, dark urine, high CK, high potassium, and rising creatinine, trace backward to the sarcolemma failure and its cause. Stop the upstream damage (remove the crush, stop the drug, cool the patient, release compartment pressure). Flood the kidneys with IV fluids (normal saline, target urine output $\geq 200\text{--}300$ mL/hour) to flush myoglobin before it clogs. Correct high potassium carefully (insulin + glucose, calcium to protect the heart, dialysis if severe). Support the kidneys (avoid nephrotoxins, monitor creatinine and urine output). Most patients recover if you catch it early and the kidneys do not fail.

Symptoms · what the patient feels, and why

- Muscle pain and tenderness (myalgia): When muscle cells break apart (the main problem in rhabdomyolysis), the dying muscle tissue swells and releases chemicals that trigger pain nerves. Think of it like a bruise on the inside — the damage sends out alarm signals that hurt.
- Muscle weakness: Broken muscle fibers can't contract and pull like healthy ones. When enough fibers are destroyed — especially in big muscles like the thighs, shoulders, or calves — you lose strength because there aren't enough working pieces left to do the job.
- Dark tea-colored or cola-colored urine: Inside every muscle cell is a protein called myoglobin (it carries oxygen for the muscle). When the cells burst open, myoglobin floods into the bloodstream and the kidneys filter it out into the urine, turning it brown or dark red — like strong tea.
- Swollen, firm muscles

- Nausea and vomiting: The flood of muscle breakdown products (like creatine kinase, potassium, and myoglobin) into the blood makes the body feel poisoned. The brain's vomit center (the area postrema in the brainstem) picks up these toxic signals and triggers nausea.
- Confusion or agitation: When rhabdomyolysis is severe, potassium leaks out of dying muscle cells and piles up in the blood (hyperkalemia), and the kidneys may start to fail. High potassium and waste buildup (uremia) disturb the electrical signals in the brain, clouding thinking and mood.
- Decreased urine output or no urine at all: Myoglobin is toxic to the kidney filters (the tubules). It clogs them like mud in a drain, and it also triggers inflammation that shuts down filtration. When the kidneys stop working (acute kidney injury), urine production drops or stops.
- Fever: The massive tissue breakdown releases inflammatory chemicals (cytokines like interleukin-1 and tumor tissue death (necrosis) factor) that act on the hypothalamus (the brain's thermostat) and raise body temperature, just like an infection does.

Focused exam

- Muscle pain, swelling, and tenderness in the legs, arms, or back: In rhabdomyolysis, the outer wrapper of muscle cells (the sarcolemma) breaks open and the muscle tissue inside dies. You feel it as deep aching pain, and you can see and feel the swelling where muscle is damaged.
- Weakness in the same muscles that are swollen and hurting: Dead muscle cannot squeeze and contract anymore. The weakness is real—the muscle fibers are torn apart and leaking, not just tired or sore.
- Dark cola-colored or tea-colored urine: Myoglobin (the oxygen-carrying protein that lives inside muscle) has spilled into the bloodstream, and now the kidneys are trying to filter it out into the pee. Dark urine means myoglobin is passing through. This is the hallmark sign of rhabdomyolysis.
- Urine dipstick shows blood, but the microscope shows zero red blood cells: The dipstick reacts to both blood and myoglobin, so it turns positive. But when you look under the microscope and see no actual red blood cells, you know the dipstick is reacting to myoglobin instead. This pattern is classic for rhabdomyolysis and rules out regular bleeding in the urine.
- Very high creatine kinase (CK) level on the blood test—often over 1,000 and sometimes over 100,000: Creatine kinase is an enzyme that lives inside muscle cells and helps them make energy. When muscle cells die in rhabdomyolysis, CK pours into the blood. The higher the CK, the more muscle has been destroyed. A CK above 5 times normal (roughly over 1,000) is the main lab sign of rhabdomyolysis.

- High potassium (hyperkalemia) on the blood test: Potassium normally lives inside muscle cells. When the muscle cell wrapper breaks in rhabdomyolysis, potassium dumps into the bloodstream. Too much potassium in the blood can stop the heart or make it beat in a deadly rhythm, so you must check the heart tracing (EKG) and treat it fast.

Diagnostics · every test answers a question

- Creatine kinase (CK) level → Is muscle breaking down, and how much?
- Serum potassium → Is potassium spilling out of dying muscle and threatening the heart?
- Serum creatinine and blood urea nitrogen (BUN) → Are the kidneys starting to fail from myoglobin clogging the filters?
- Urinalysis with microscopy → Is there myoglobin in the urine, and is it true rhabdomyolysis or just blood in the pee?
- Serum calcium → Is calcium being trapped in the dead muscle tissue?
- Serum phosphate → Is phosphate spilling out of broken muscle cells?
- Electrocardiogram (EKG) → Is the high potassium changing the heart's electrical pattern and putting the patient at risk for a deadly rhythm?
- Urine output monitoring → Are the kidneys still making enough urine to flush out myoglobin, or are they shutting down?

Treatment logic

- Aggressive IV fluid resuscitation with normal saline, starting at 1 to 1.5 liters per hour and adjusting to keep urine output at 200 to 300 mL/hour → High-volume IV fluids dilute the myoglobin in the kidney tubules and flush it out before it can clump together and clog the filters. Fluids also keep blood pressure high enough to push fluid through the kidney's filtering units (glomeruli) and prevent the kidney from shutting down.
- Treat high potassium (hyperkalemia) with insulin plus dextrose (to shift potassium into cells), calcium gluconate (to protect the heart if the EKG shows changes), sodium bicarbonate (to shift potassium), or potassium binders like sodium polystyrene sulfonate or patiomer → These treatments either push potassium back into cells or remove it from the body, lowering the blood level and stopping the heart from firing in a dangerous rhythm. Calcium does not lower potassium but steadies the heart muscle so it does not respond to the high potassium.

- Start dialysis (hemodialysis or continuous renal replacement therapy) if the kidneys fail, potassium stays dangerously high despite treatment, the patient is overloaded with fluid and at risk for lung swelling, or severe acid or phosphate buildup happens → Dialysis removes potassium, phosphate, acid, and extra water from the blood when the kidneys cannot do it anymore. It prevents deadly heart rhythms from high potassium and keeps fluid from backing up into the lungs.
- Find and treat the underlying cause—stop the statin or other muscle-toxic drug, cool down the overheated patient, stop the seizure, release the crushed limb, manage the drug overdose or withdrawal, or treat the infection → Stopping the cause halts ongoing muscle injury at the source, so new myoglobin, potassium, and cell contents stop pouring into the blood. This is the only way to truly stop rhabdomyolysis from getting worse.
- Surgical fasciotomy (cutting open the tight tissue wrapping around a muscle compartment) if compartment pressure stays above 30 mmHg despite fluids and threatens to cut off blood flow and kill more muscle → Cutting the tight fascia lets swollen muscle expand outward so pressure drops and blood can flow back into the muscle. This prevents a second wave of muscle death from pressure cutting off the blood supply.
- Give calcium gluconate for low calcium (hypocalcemia) only if the patient has symptoms like muscle cramps, tingling around the mouth, or seizures; avoid giving calcium routinely → Raises blood calcium just enough to stop nerves and muscles from firing on their own (tetany). Also avoids pushing more calcium into dead muscle, where it can harden and make inflammation worse.
- Consider alkalinizing the urine with sodium bicarbonate (targeting urine pH above 6.5) in select cases, though evidence is mixed and it is no longer routine in all guidelines → Raising urine pH may help keep myoglobin dissolved and prevent it from crystallizing in the tubules. Bicarbonate also treats the acid buildup (metabolic too much acid in the blood (acidosis)) that often comes with severe rhabdomyolysis.

Medications

Normal saline or lactated Ringer's solution (IV fluids) · Crystalloid intravenous fluids

Water with salt (or water with salt and a tiny bit of lactate) pumped directly into a vein to flood the kidneys with extra liquid so they can flush out the muscle protein (myoglobin) before it clogs the kidney filters and causes kidney failure.

1. Start at 1 to 1.5 liters per hour through an IV to quickly boost blood volume.
2. Extra fluid floods the kidneys, making the filters work faster and produce more urine.

3. Myoglobin (the reddish-brown muscle protein that leaked into the blood when muscle cells broke open) gets diluted and washed out in urine before it can crystallize and clog the tiny kidney tubes.
4. Adjust the drip rate to keep urine output around 200 to 300 milliliters per hour (about one to one-and-a-half cups every hour) – this steady flush protects the kidneys.
5. Continue IV fluids until the urine clears (no longer tea-colored) and creatine kinase (CK, the blood marker of muscle breakdown) starts dropping.

SAFETY WATCH

Watch for fluid overload – too much IV fluid can back up into the lungs (pulmonary swelling (edema)) and cause trouble breathing, especially in people with heart failure or kidney disease. · Monitor urine output closely – if urine drops despite fluids, the kidneys may already be failing and dialysis may be needed. · Check potassium and calcium levels frequently – aggressive fluids alone do not fix dangerously high potassium (hyperkalemia) or low calcium (hypocalcemia).

Insulin (regular) plus glucose (dextrose) · Hormone plus sugar (emergency potassium-lowering therapy)

Insulin (the hormone that opens the door for sugar to enter cells) is given along with sugar (glucose) to push potassium from the blood back inside muscle and other cells, temporarily lowering dangerously high blood potassium that can stop the heart.

1. Give 10 units of regular insulin by IV along with 25 grams of dextrose (sugar water) to prevent low blood sugar.
2. Insulin turns on the sodium-potassium pump in cell membranes, which trades sodium (coming out) for potassium (going in).
3. Potassium shifts from the blood into muscle cells, liver cells, and fat cells.
4. Blood potassium drops by about 0.5 to 1.2 mEq/L within 15 to 60 minutes.
5. Effect lasts 4 to 6 hours – this buys time while fluids, dialysis, or other treatments address the root cause.

SAFETY WATCH

Check blood sugar before and after – insulin can cause dangerously low blood sugar (hypoglycemia), so always give glucose with it and recheck sugar in 30 minutes. · Recheck potassium in 1 hour – the shift is temporary, and potassium can climb again as insulin wears off. · Use with caution in kidney failure – if the kidneys cannot remove potassium, the blood level will rise again once the insulin effect fades.

Calcium gluconate or calcium chloride (IV) · Electrolyte replacement (cardiac membrane stabilizer)

Calcium (a mineral that helps the heart's electrical system fire correctly) is given by IV to immediately protect the heart muscle from the dangerous effects of high potassium, which can stop the heartbeat – calcium does NOT lower potassium, it just shields the heart while other treatments bring potassium down.

1. Give 1 gram of calcium gluconate (or 0.5 to 1 gram of calcium chloride) by slow IV push over 2 to 5 minutes.
2. Calcium floods the space around heart muscle cells and counteracts the effect of high potassium on the cell membrane.
3. The heart's electrical threshold is stabilized – dangerous rhythms (peaked T waves, wide QRS, sine wave) are less likely to progress to cardiac arrest.
4. Protection begins in 1 to 3 minutes and lasts 30 to 60 minutes.
5. Repeat the dose if ECG changes persist or worsen.

SAFETY WATCH

Give slowly – fast IV calcium can cause a dangerous drop in heart rate or even cardiac arrest. · Do NOT give calcium through the same IV line as bicarbonate – they react and form a chalky precipitate that can clog the line or blood vessels. · Use calcium chloride (not gluconate) if immediate effect is critical or if the person is in cardiac arrest – chloride form delivers more usable calcium faster. · Avoid giving calcium if the person has symptomatic low calcium (hypocalcemia) from rhabdomyolysis unless they are having seizures or severe symptoms – extra calcium can deposit back into damaged muscle and worsen injury during the recovery phase.

Sodium bicarbonate (IV) · Alkalinizing agent (urine pH modifier)

Baking soda given by IV to make the blood and urine more alkaline (less acidic), which in theory keeps myoglobin (the muscle protein clogging the kidneys) dissolved and prevents it from crystallizing in the kidney tubes – however, recent evidence shows NO clear benefit, and many hospitals no longer use it routinely for rhabdomyolysis.

1. Give 50 to 100 milliequivalents (mEq) of sodium bicarbonate mixed in IV fluids (or as a separate infusion).
2. Bicarbonate buffers acid in the blood and is filtered into the urine, raising urine pH.
3. In theory, alkaline urine keeps myoglobin from forming solid crystals that block kidney tubes.
4. Goal is urine pH above 6.5.

5. Recheck blood pH and electrolytes every few hours – bicarbonate can cause too little acid in the blood (alkalosis) (too much base) and worsen low calcium.

SAFETY WATCH

Can worsen low calcium (hypocalcemia) by shifting calcium into bones and tissues – do NOT give if calcium is already dangerously low unless seizures or severe symptoms are present. · Can cause metabolic alkalosis (the blood turning too alkaline) (blood too alkaline), which triggers muscle cramps, confusion, and irregular heartbeats. · Provides a large sodium load – risky in heart failure or fluid overload. · Many major guidelines (including Kidney Disease: Improving Global Outcomes, KDIGO) now recommend AGAINST routine bicarbonate for rhabdomyolysis because high-quality evidence of benefit is lacking.

Mannitol (IV) · Osmotic diuretic (kidney-flushing agent)

A sugar-alcohol that pulls extra water from body tissues into the bloodstream and then forces the kidneys to make more urine, flushing out myoglobin and reducing swelling inside muscle compartments – used in some centers but NOT universally recommended because evidence of benefit is weak.

1. Give 12.5 to 25 grams of mannitol by IV (often as a 20% solution).
2. Mannitol creates osmotic pressure in the blood and kidney tubules, pulling water from tissues into the bloodstream and then into the urine.
3. Urine output increases, flushing myoglobin and other muscle debris out of the kidneys.
4. May reduce swelling in tight muscle compartments (compartment syndrome).
5. Effect starts within 30 to 60 minutes and lasts 3 to 6 hours.

SAFETY WATCH

Can cause sudden fluid overload and pulmonary edema (fluid flooding the lungs) (water in the lungs) if given too fast or if the kidneys are already failing and cannot make urine. · Do NOT use if the person is not making urine (anuric) – mannitol will accumulate and worsen fluid overload. · Can cause rebound swelling and worsening electrolyte imbalances. · Many centers have STOPPED using mannitol for rhabdomyolysis because large studies show no clear benefit and potential harm.

Hemodialysis (renal replacement therapy) · Extracorporeal blood filtration (not a drug – a procedure)

A machine that takes blood out of the body, runs it past a filter (dialyzer) that removes waste, extra potassium, acid, and fluid, and then returns the cleaned blood – used when the kidneys

have failed and cannot clear the flood of muscle breakdown products, or when potassium or fluid overload is life-threatening.

1. Place a large IV catheter (dialysis catheter) into a central vein (neck, chest, or groin).
2. Blood is pumped out of the body into the dialysis machine.
3. The dialyzer (artificial filter) uses diffusion and ultrafiltration to pull out waste, extra potassium, acid, and fluid.
4. Cleaned blood is returned to the body.
5. Sessions typically run 3 to 4 hours and are repeated daily or every other day until kidney function recovers.

SAFETY WATCH

Requires central venous catheter – risk of infection, bleeding, or blood clot. · Can cause low blood pressure during the session, especially if too much fluid is removed too quickly. · Can cause electrolyte shifts – low potassium (hypokalemia), low phosphate (hypophosphatemia), or dialysis disequilibrium syndrome (brain swelling from rapid waste removal). · Does NOT remove myoglobin efficiently – myoglobin is a large protein that does not pass easily through the dialysis membrane, so dialysis mainly treats the complications (high potassium, fluid overload, acid buildup, kidney failure) rather than the myoglobin itself.

Albuterol (nebulized) · Beta-2 adrenergic agonist (bronchodilator, used off-label for high potassium (hyperkalemia))

A rescue inhaler medicine (usually used for asthma) that activates beta-2 receptors on cell membranes, turning on pumps that push potassium from the blood back into cells – used as a temporary emergency measure to lower dangerously high potassium.

1. Give 10 to 20 milligrams of albuterol by nebulizer (breathing treatment) over 10 minutes.
2. Albuterol binds to beta-2 receptors on muscle and other cells.
3. The receptors signal the sodium-potassium pump to shift potassium from blood into cells.
4. Blood potassium drops by 0.5 to 1.0 mEq/L within 30 to 90 minutes.
5. Effect lasts 2 to 4 hours – repeat doses may be needed.

SAFETY WATCH

Can cause rapid heart rate (tachycardia), tremor, and anxiety – use cautiously in people with heart disease. · Effect is temporary – potassium will rise again as the drug wears off, so combine with other treatments (insulin, dialysis). · Does NOT work in everyone – about 30% of people do not respond well to nebulized albuterol for potassium lowering.

Sodium polystyrene sulfonate (Kayexalate) or patiromer (Veltassa) or sodium zirconium cyclosilicate (Lokelma) · Potassium binders (gut exchange resins)

Powders or liquids given by mouth or rectum that bind potassium in the intestines and carry it out in the stool, slowly lowering blood potassium over hours to days – NOT used for emergency potassium lowering (too slow), but sometimes used after the crisis to prevent potassium from climbing again.

1. Give 15 to 30 grams of sodium polystyrene sulfonate by mouth (mixed with water or sorbitol) or as a retention enema.
2. The resin travels through the intestines and exchanges its sodium for potassium in the gut.
3. Potassium binds to the resin and is carried out in the stool.
4. Blood potassium begins to drop in 2 to 6 hours and continues to fall over 24 to 48 hours.
5. Newer agents (patiromer, sodium zirconium cyclosilicate) work faster (4 to 24 hours) and have fewer side effects.

SAFETY WATCH

Sodium polystyrene sulfonate can cause severe constipation, gut blockage (impaction), or – rarely – intestinal damage (colonic tissue death (necrosis)), especially when given with sorbitol. · Can cause sodium overload and fluid retention (dangerous in heart failure). · Do NOT use in people with bowel obstruction, severe constipation, or recent abdominal surgery. · Newer agents (patiromer, sodium zirconium cyclosilicate) are safer and preferred if available, but are expensive and not always stocked in hospitals.

Pain control (acetaminophen, opioids as needed) · Analgesics (pain relievers)

Medicines to reduce severe muscle pain from rhabdomyolysis – acetaminophen (Tylenol) blocks pain signals in the brain, and opioids (morphine, fentanyl) bind to opioid receptors in the brain and spinal cord to dull pain perception – important for comfort but do NOT treat the muscle breakdown itself.

1. Give acetaminophen 500 to 1,000 milligrams by mouth or IV every 6 hours (max 4 grams per day).
2. Acetaminophen inhibits cyclooxygenase (COX) enzymes in the brain, reducing pain and fever.
3. If pain is severe, add an opioid (morphine 2 to 10 mg IV, or fentanyl 25 to 100 micrograms IV) as needed.
4. Opioids bind to mu-opioid receptors in the brain and spinal cord, blocking pain signals and producing pain relief within 5 to 15 minutes.
5. Use the lowest effective dose and taper as muscle pain improves.

SAFETY WATCH

Acetaminophen – do NOT exceed 4 grams per day (risk of liver damage, especially in people with liver disease or alcohol use). · Avoid NSAIDs (ibuprofen, naproxen, ketorolac) – they reduce blood flow to the kidneys and can worsen acute kidney injury in rhabdomyolysis. · Opioids – watch for slowed breathing (respiratory depression), low blood pressure, confusion, and constipation; use cautiously in kidney failure (opioids and their breakdown products accumulate and can cause overdose).

Red flags

- Dark or tea-colored urine (sign that myoglobin is spilling into the urine and may be damaging the kidneys)
- Little or no urine output (sign the kidneys are failing)
- Severe muscle pain, swelling, or weakness that keeps getting worse (risk of compartment syndrome — pressure building up so high it cuts off blood flow)
- Confusion, irregular heartbeat, or chest pain (sign that potassium or calcium is dangerously out of balance and threatening the heart)
- History of a major trigger: crush injury, prolonged immobilization, seizures, extreme exercise, heat stroke, or certain drugs (statins, cocaine, amphetamines)

Nursing priorities

1. Start two large-bore IV lines and infuse normal saline or lactated Ringer solution at 1.5 liters per hour, then adjust the rate to keep urine output between 200 and 300 milliliters every hour. — Aggressive IV fluids dilute and flush myoglobin out of the kidney tubules before it can precipitate (form crystals) and cause acute kidney injury; high urine flow physically washes the toxin through and protects kidney function.
2. Place a Foley catheter and measure urine output every hour; observe and document the color of the urine. — Hourly urine measurement allows you to titrate (adjust) IV fluid rates precisely to the goal of 200 to 300 milliliters per hour; dark tea or cola-colored urine confirms myoglobin is being excreted and tells you the kidneys are still filtering.
3. Draw blood immediately for CK (creatine kinase, a muscle enzyme), potassium, creatinine (kidney function marker), calcium, phosphate, and check a baseline EKG for peaked T waves or wide QRS complexes. — CK levels above 5000 confirm severe muscle breakdown; potassium above 5.5 milliequivalents per liter can cause fatal arrhythmias (irregular heartbeats), and the EKG shows high potassium (hyperkalemia) changes before the heart stops; creatinine rising means the kidneys are failing.

4. Monitor continuous telemetry and watch for peaked T waves, widened QRS, or arrhythmias; have calcium gluconate 1 gram IV and insulin with dextrose at the bedside ready to give immediately. — High potassium (Hyperkalemia) destabilizes the heart muscle membrane and can cause ventricular fibrillation (a fatal rhythm) within minutes; calcium gluconate protects the heart membrane while insulin plus dextrose shifts potassium back into cells and buys time for definitive treatment.
5. Perform neurovascular checks every 2 hours on all limbs: assess pulses, capillary refill, sensation, movement, and measure compartment firmness; notify the physician immediately if pain is out of proportion to exam or if pulses weaken. — Compartment syndrome occurs when swelling inside a closed muscle space compresses blood vessels and nerves; the 6 Ps (pain, pressure, tingling or numbness (paresthesia) (numbness), pale skin (pallor) (pale skin), pulselessness, paralysis) indicate urgent surgical fasciotomy is needed to restore blood flow and save the limb.
6. Track daily weights, strict intake and output totals, and watch for signs of fluid overload such as crackles in the lungs, shortness of breath, or jugular vein distension. — Liters of IV fluid are needed to protect the kidneys but can cause pulmonary edema (fluid flooding the lungs) (fluid in the lungs) and heart failure if the kidneys stop working; daily weights and lung sounds catch overload early so you can slow fluids or start dialysis.
7. Hold all nephrotoxic drugs (NSAIDs like ibuprofen, ACE inhibitors, aminoglycoside antibiotics) and statins; review the medication list with the provider and confirm nothing is making the rhabdomyolysis worse. — NSAIDs and other nephrotoxic medications reduce kidney blood flow and worsen acute kidney injury; statins continue to break down muscle; stopping these drugs prevents additional harm while the body heals.
8. Prepare for urgent hemodialysis or continuous renal replacement therapy if potassium stays above 6.5 milliequivalents per liter despite treatment, creatinine keeps rising, urine output drops below 0.5 milliliters per kilogram per hour for 6 hours, or the patient becomes volume overloaded. — Dialysis physically removes potassium, myoglobin, and excess fluid when the kidneys fail; it is life-saving when medical management of high potassium (hyperkalemia) fails or when fluid overload threatens the lungs and heart.

Sources

- UpToDate Clinical Guidelines on Rhabdomyolysis
- American College of Physicians (ACP) Clinical Practice
- National Kidney Foundation (NKF) Acute Kidney Injury Guidelines
- Cochrane Reviews on Muscle Injury and Acute Renal Failure

Last reviewed: 2026-07-07