

Rhabdomyolysis

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.

THE ONE-LINE TRUTH

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 chain — how it unfolds

1. The sarcolemma (muscle cell wrapper) tears open — why: 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 — why: 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 — why: 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 — why: 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 — why: 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 — why: 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 — why: 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 — why: 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.

Spot it — what you see, 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.

Red flags — act now

- 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)

Treat it — in this order

1. 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.
2. 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.
3. 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.
4. 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.

5. 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.

Your top priorities at the bedside

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. — why: 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. — why: 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. — why: 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.