

Tumor Lysis Syndrome

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THE STORY IN ONE SENTENCE

Cancer treatment kills millions of cancer cells so fast that the kidneys cannot clear all the stuff they dump into the blood.

“Tumor lysis syndrome happens when cancer cells die in huge numbers and spill their insides — potassium, phosphate, and uric acid — into the bloodstream faster than the kidneys can clean it up.”

Normal anatomy — the body's job

Every cell in the body holds potassium, phosphate, and DNA building blocks locked inside. When a cell dies naturally, it breaks open gently and releases tiny amounts of these chemicals into the blood. The kidneys filter them out and send them into the urine. The parathyroid glands (four tiny glands in the neck) and kidneys together keep calcium and phosphate balanced in the blood so muscles and nerves work smoothly.

BODY'S JOB

The kidneys act like the body's cleanup crew. They filter blood 24/7, pulling out waste chemicals — including uric acid (made from broken-down DNA), potassium (the main electrical signal inside cells), and phosphate (a mineral inside every cell). The kidneys send these chemicals into urine and keep the blood clean. At the same time, the parathyroid glands sense calcium levels and tell the kidneys to hold onto calcium or let it go, keeping the balance tight so the heart beats steadily, muscles relax after they squeeze, and nerves send signals without misfiring.

1. A cell dies and breaks open gently, releasing a small puff of potassium, phosphate, and purines (DNA pieces) into the bloodstream.
2. Blood flows through the kidneys, which have millions of tiny filters called nephrons (the kidney's working units).

3. Each nephron pulls potassium, phosphate, and uric acid (made from purines) out of the blood and sends them into a tube that drains into urine.
4. The parathyroid glands watch blood calcium levels; if phosphate rises even a little, they signal the kidneys to push it out faster and hold onto calcium.
5. Calcium stays steady in the blood, so the heart's electrical system fires on time and muscles relax smoothly after every contraction.
6. The whole system hums along quietly, cleaning the blood and keeping the heart, muscles, and brain safe.

What breaks

When chemotherapy, steroids, or targeted cancer drugs hit a bulky cancer — especially fast-growing blood cancers like Burkitt lymphoma, acute lymphoblastic leukemia, or acute myeloid leukemia — millions of cancer cells die within hours. Each cell bursts like a tiny water balloon, dumping all its potassium, phosphate, and DNA pieces into the blood at the same moment. The kidneys try to filter it all out, but the flood is too big and too fast. Potassium stacks up and stops the heart's rhythm. Phosphate grabs onto calcium and hides it, leaving the blood calcium dangerously low. The liver turns the DNA pieces into uric acid, which forms sharp crystals that clog the kidney tubes like sand blocking a drain, and the kidneys start to fail.

CLINICAL TERM

Massive tumor cell lysis (rupture) releases intracellular potassium, phosphate, and nucleic acids into the bloodstream faster than renal clearance can compensate.

How the body compensates

The body has no good compensation for tumor lysis syndrome because the flood of chemicals happens faster than any organ can adapt. The kidneys try to push out extra potassium and uric acid, but they are quickly overwhelmed. The parathyroid glands try to raise calcium, but phosphate keeps binding it faster than the glands can respond. Vital signs may look stable for a few hours while the kidneys strain to keep up, but once the kidneys fail, the whole system crashes — potassium stops the heart, low calcium triggers seizures, and the patient needs emergency dialysis. This is why prevention is everything: spotting high-risk patients before chemo and acting early stops the cascade before it starts.

1. {'step': 'The kidneys ramp up filtration', 'how': 'The nephrons (kidney filters) try to push extra potassium, phosphate, and uric acid into the urine as fast as possible.', 'limit': 'This only works if the patient is well-hydrated and the flood of chemicals is small. In tumor lysis syndrome, the flood is so large that the tubules get clogged with uric acid crystals and calcium-phosphate deposits, and filtration stops.'}

2. {'step': 'The parathyroid glands try to raise calcium', 'how': 'The parathyroid glands sense low blood calcium and release parathyroid hormone (PTH), which tells the kidneys to reabsorb calcium and tells bones to release calcium into the blood.', 'limit': 'Phosphate binds calcium faster than PTH can replace it, so calcium stays dangerously low despite the glands working overtime.'}
3. {'step': 'The heart tries to beat through rising potassium', 'how': 'The sinoatrial node (the heart's natural pacemaker) keeps firing, trying to maintain a steady rhythm.', 'limit': 'As potassium rises above 6.5–7 mEq/L, the heart muscle becomes electrically stunned and cannot repolarize (reset) between beats. The rhythm degenerates into ventricular fibrillation or flatline (asystole).'}- 4. {'step': 'Warning: Vital signs stay normal until the crash', 'how': 'Blood pressure, heart rate, and oxygen saturation may look fine while the kidneys are still filtering and the heart is still compensating.', 'danger': 'This stable window is deceptive. Labs (rising potassium, phosphate, uric acid, creatinine; dropping calcium) show the real story. Once the patient tips into kidney failure or arrhythmia, the crash is sudden and often irreversible without dialysis.'}
- 5. {'step': 'The crash point: cardiac arrest, seizure, or anuric kidney failure', 'reality': 'The worst outcomes are hyperkalemic cardiac arrest (heart stops because potassium is too high), tonic-clonic seizure from severe low calcium (hypocalcemia) (calcium too low), or anuric acute kidney injury requiring emergent dialysis.'}
- 6. {'step': 'Prevention is much easier than rescue', 'why': 'Once tumor lysis syndrome is full-blown, dialysis is often the only option. But if you identify high-risk patients (bulky Burkitt lymphoma, acute leukemia with white count >50,000, LDH >2× normal) before starting chemo, you can prevent the syndrome with aggressive IV hydration (150–200 mL/kg/day), allopurinol (blocks uric acid production), or rasburicase (breaks down existing uric acid), and close lab monitoring (potassium, phosphate, calcium, uric acid, creatinine every 4–6 hours for 24–48 hours after chemo).'}-

Domino effect

1. Chemotherapy or targeted therapy kills millions of cancer cells within hours — Drugs like cytarabine, cyclophosphamide, steroids (dexamethasone, prednisone), or rituximab trigger rapid cancer cell death (apoptosis or tissue death (necrosis)) in bulky, fast-growing tumors like Burkitt lymphoma, acute lymphoblastic leukemia, or acute myeloid leukemia. Each dying cell bursts open and spills its insides into the bloodstream.
2. Potassium floods the blood (hyperkalemia) — Every cell holds potassium inside at high concentration (about 140 mEq/L inside vs. 4 mEq/L outside). When millions of cells rupture at once, potassium pours into the blood, spiking serum potassium to 6, 7, or even 8 mEq/L within 12–24 hours.

3. The heart's rhythm falls apart — The heart's electrical system depends on a precise potassium gradient across heart muscle cells. High potassium depolarizes (flattens) the cell membranes so they cannot repolarize (reset) after each beat. The EKG shows tall, peaked T waves first, then the QRS complex widens, the P wave disappears, and finally the rhythm degenerates into a sine wave, ventricular fibrillation, or asystole (flatline) — cardiac arrest.
4. Phosphate floods the blood (hyperphosphatemia) — Dying cells also release phosphate (another mineral stored inside cells). Blood phosphate shoots up to 6, 8, or 10 mg/dL or higher, far above the normal range of 2.5–4.5 mg/dL.
5. Phosphate binds calcium and drops blood calcium (hypocalcemia) — Phosphate and calcium are chemical opposites: when phosphate rises, it grabs onto calcium and forms solid calcium-phosphate crystals that deposit in tissues. Free calcium in the blood plummets, often below 7 mg/dL (normal is 8.5–10.5 mg/dL).
6. Muscles twitch and the brain seizes — Calcium is the off-switch for muscle contraction and the brake on nerve firing. Without enough free calcium, muscles cannot relax after they contract (causing tetany — rigid, painful muscle spasms, especially in the hands and feet) and neurons fire uncontrollably (causing tonic-clonic seizures).
7. Uric acid floods the blood and crystallizes in the kidneys (hyperuricemia and crystal nephropathy) — When DNA from dying cancer cells breaks down, the liver converts the purine bases (adenine and guanine) into uric acid. Uric acid levels spike to 10, 15, or 20 mg/dL (normal is under 7 mg/dL). Uric acid is not very soluble, especially in acidic urine, so it forms sharp, needle-shaped crystals in the kidney tubules, physically clogging them like sand blocking a drainpipe.
8. The kidneys fail (acute kidney injury) — Uric acid crystals and calcium-phosphate crystals obstruct the tubules and damage the nephrons (the kidney's working filters). Urine output drops (oliguria) or stops (anuria), creatinine and BUN rise, and the kidneys can no longer clear potassium, phosphate, or any other waste. This makes the high potassium (hyperkalemia) and hyperphosphatemia even worse, creating a vicious cycle.
9. The whole system crashes without dialysis — Once the kidneys fail, there is no way to clear the flood of potassium, phosphate, and uric acid except dialysis. Without it, the patient dies from cardiac arrest (hyperkalemia), seizure (hypocalcemia), or severe waste buildup in the blood (uremia) (waste buildup in the blood). Every step — heart arrhythmia, muscle spasms, kidney failure — traces back to one upstream event: too many cancer cells dying too fast.

Symptoms · what the patient feels, and why

- Fast or skipping heartbeat (arrhythmia): High potassium (from cancer cells breaking open) makes the heart's electrical signals misfire, so the heart beats out of rhythm or stops suddenly.

- Muscle stiffness, cramps, or twitching (tetany): Low calcium (pulled into crystals with the high phosphate released from dying tumor cells) makes nerves fire too easily, causing muscles to lock or jerk.
- Seizures: Low calcium or high phosphate throws off the brain's electrical balance, so neurons fire all at once, triggering a seizure.
- Peeing very little or not at all (acute kidney injury): Calcium and phosphate crystals clog the tiny filters (tubules) in the kidneys, and uric acid (from tumor DNA breaking down) plugs them further, so urine can't flow out.
- Nausea, vomiting, or diarrhea: High uric acid and high phosphate irritate the gut lining, and the kidneys can't clear waste, so toxins build up in the blood and upset the stomach.
- Feeling very tired and weak: High potassium, low calcium, and kidney failure together slow the heart and muscles and make cells struggle to do their jobs, draining energy.
- Confusion or feeling 'out of it': The brain is very sensitive to wrong levels of calcium, potassium, and waste products (like urea) that the kidneys can't clear, so thinking gets foggy.
- Flank pain (pain in the sides of the back, near the kidneys): Crystals and uric acid clog the kidney tubules so fast that the kidneys swell and stretch their capsule (outer covering), which hurts.

Focused exam

- Irregular heartbeat or fast heart rhythm, especially peaked T-waves on the EKG (heart tracing): When tumor lysis syndrome kills cancer cells in a flood, potassium spills out of those cells and builds up in the blood. High potassium (hyperkalemia) stops the heart from beating in a steady rhythm. The heart muscle gets too excited and can stop suddenly. This is the most dangerous sign.
- Muscle twitching, cramps, or shaking (tetany), especially in the hands, feet, and face: Dead cancer cells release phosphate. Phosphate latches onto calcium in the blood and pulls it out of circulation. Low calcium (hypocalcemia) makes nerve signals fire too easily, so muscles twitch and cramp. This tells you tumor lysis syndrome is dropping calcium fast.
- Seizure, confusion, or severe drowsiness: Low calcium from tumor lysis syndrome can make brain nerve cells fire uncontrollably and trigger a seizure. Very high potassium can also slow down brain signals and cause confusion or sleepiness. Either way, the brain is in danger and tumor lysis syndrome is the upstream cause.

- Dark urine, cloudy urine with crystals, or very little urine coming out (oliguria, less than 0.5 mL/kg per hour): Tumor lysis syndrome dumps uric acid—a waste product from broken-down DNA—into the kidneys. Uric acid turns into sharp crystals that clog the tiny kidney tubes like gravel in a garden hose. The kidneys start to fail, so they make less urine and it looks darker or cloudy.
- Swelling in the legs, ankles, or face (edema); high blood pressure: When the kidneys fail from uric acid damage, they cannot clear salt and water from the body anymore. Fluid backs up into the tissues and blood vessels, causing swelling and pushing blood pressure up.
- Lab panel showing high potassium (> 6.0 mEq/L), high phosphate (> 4.5 mg/dL in adults, > 6.5 in children), high uric acid (> 8 mg/dL), low calcium (< 7 mg/dL), and rising creatinine (> 0.3 mg/dL increase or $> 1.5\times$ baseline): These lab numbers are the fingerprint of tumor lysis syndrome. They all point back to one upstream break: cancer cells dying in a massive wave and spilling their contents into the blood faster than the kidneys can clean them up. This confirms the diagnosis.

Diagnostics · every test answers a question

- Basic metabolic panel (BMP) with calcium and phosphate → Is the cell die-off flooding the blood with potassium, phosphate, and pulling calcium down? Are the kidneys still able to filter waste?
- Serum uric acid → Is uric acid—the waste from broken-down DNA—building up and threatening to clog the kidneys?
- Lactate dehydrogenase (LDH) → How much cell death is happening right now? LDH is a protein inside cells that spills out when cells burst.
- Urinalysis with microscopy → Are uric acid crystals forming in the urine and clogging the kidney tubes?
- Electrocardiogram (EKG) → Is high potassium already changing the heart's electrical rhythm in a dangerous way?
- Repeat BMP with calcium, phosphate, uric acid, and LDH every 4–6 hours during the first 24–48 hours after starting cancer therapy → Are the numbers getting better with treatment, or are they still climbing because cells are still dying in a flood?

Treatment logic

- Aggressive IV hydration with normal saline at 200–250 mL/m² per hour (about 3 L/m² per day) to keep urine output at least 80–100 mL/m² per hour → Flooding the bloodstream with IV fluid dilutes the burst-cell contents (potassium, phosphate, uric acid) and flushes them through the kidneys faster. Fast-flowing urine washes uric acid out before it can settle and turn into sharp crystals that clog the kidney tubes. This protects the kidneys from shutting down.
- Rasburicase (a recombinant enzyme) for high-risk patients, or allopurinol for low-to-intermediate risk, to lower uric acid → Rasburicase breaks down uric acid that already exists in the blood into allantoin, a harmless substance that dissolves easily and leaves through urine. This stops uric acid from crystallizing in the kidneys. Allopurinol blocks the enzyme (xanthine oxidase) that makes new uric acid, so less is produced going forward. Rasburicase works in hours; allopurinol takes days.
- Treat high potassium (hyperkalemia) with IV calcium gluconate (if EKG shows dangerous changes), insulin + dextrose (glucose), inhaled beta-agonists (albuterol), and oral or rectal potassium binders (sodium polystyrene sulfonate, patiomer) → Calcium gluconate protects the heart muscle from high potassium's dangerous electrical effects for 30–60 minutes but does not lower potassium itself. Insulin + dextrose and beta-agonists push potassium out of the blood and into cells temporarily (within minutes to hours). Potassium binders remove potassium from the body through stool over hours to days. Together, these treatments prevent deadly heart rhythms.
- Oral phosphate binders (sevelamer or lanthanum carbonate, NOT calcium-based if phosphate is very high) and avoid giving IV calcium until phosphate drops below 6 mg/dL → Phosphate binders stick to phosphate in the gut so the body does not absorb it from food. This lowers the amount of new phosphate entering the blood. Giving IV calcium too early when phosphate is sky-high causes calcium and phosphate to combine and form hard crystals that deposit in tissues (calcification), which damages organs. Lowering phosphate first makes it safe to replace calcium.
- Continuous heart (cardiac) monitoring on telemetry and urgent hemodialysis if potassium > 6.5 mEq/L despite treatment, phosphate > 10 mg/dL, severe symptoms (seizure, dangerous heart rhythm), or acute kidney injury with rising creatinine and low urine output → Continuous monitoring catches deadly heart rhythm changes the moment they start, so you can act in seconds. Hemodialysis (a machine that filters the blood) directly removes potassium, phosphate, and uric acid from the bloodstream in hours and takes over kidney function when the kidneys shut down. It is the only treatment that can drop all three electrolytes fast when medications are not enough.

- Hold or reduce the dose of the next round of chemotherapy if tumor lysis syndrome is severe and not controlled → Slowing down the cancer cell die-off gives the kidneys and the rest of the body time to catch up and clear the flood of cell contents. This can prevent complete kidney failure and death.

Medications

Normal saline (IV fluids) · Isotonic crystalloid fluid

Floods the blood and kidneys with clean water and salt to dilute the dangerous chemicals that spill out when cancer cells die, then flushes them out through the urine before they can clog the kidney filters or cause heart problems.

1. Large volumes of saline (usually 200–300 mL per kilogram per day, which can be 3 liters per square meter of body surface per day) are infused directly into a vein.
2. The extra fluid dilutes the blood, lowering the concentration of potassium, phosphate, and uric acid that have leaked out of dying tumor cells.
3. The kidneys filter this larger volume of dilute blood and make more urine, carrying the dangerous waste chemicals out of the body before they can crystallize in the kidney tubules or trigger a dangerous heart rhythm.
4. Goal is to keep urine output high (at least 80–100 mL per square meter per hour) so nothing has time to settle and block the kidneys.

SAFETY WATCH

Watch for fluid overload – too much saline can fill the lungs (pulmonary swelling (edema)) and cause trouble breathing, especially in patients with heart or kidney disease. · Monitor weight, breathing rate, and listen to the lungs daily; stop or slow fluids if the patient becomes short of breath or gains weight rapidly. · Check electrolytes (potassium, calcium, phosphate) and kidney function (creatinine, urine output) every 4–6 hours in high-risk patients to catch problems early.

Allopurinol · Xanthine oxidase inhibitor

Blocks the enzyme (xanthine oxidase) that turns purines – the DNA building blocks released from dying cancer cells – into uric acid, so uric acid never forms in the first place.

1. When tumor cells die, their DNA breaks into purines (small molecules like hypoxanthine and xanthine).
2. Normally the enzyme xanthine oxidase adds oxygen to these purines and turns them into uric acid.

3. Allopurinol looks similar to a purine, so it tricks and binds to xanthine oxidase, jamming the enzyme so it cannot make uric acid.
4. The purines stay as xanthine and hypoxanthine, which dissolve in water better than uric acid and leave the body more easily through the kidneys.
5. Used in low- and intermediate-risk patients (smaller tumor burden, normal kidney function, lower white blood cell count) before chemotherapy starts, usually 100 mg per square meter every 8 hours (up to 300 mg total per dose).

SAFETY WATCH

Can cause a severe skin rash (Stevens-Johnson syndrome or DRESS syndrome) – stop the drug immediately if any new rash, mouth sores, or fever appear. · Adjust dose if kidneys are not working well; too much allopurinol can build up and cause side effects. · May raise levels of xanthine, which rarely can form crystals in the urine and kidneys – keep urine output high with IV fluids. · Do not use allopurinol and rasburicase together – allopurinol will block the breakdown rasburicase is trying to do.

Rasburicase · Recombinant urate oxidase enzyme

Directly breaks down uric acid that is already in the blood into allantoin, a harmless waste product that dissolves easily in water and leaves the body through urine within minutes.

1. Rasburicase is a man-made copy of an enzyme (urate oxidase) that humans do not naturally make but other animals do.
2. When injected into a vein, it immediately finds uric acid molecules in the blood and adds oxygen to them, breaking each uric acid into allantoin, carbon dioxide, and hydrogen peroxide.
3. Allantoin is ten times more soluble in water than uric acid, so it flows easily through the kidneys and out in the urine without forming crystals.
4. Works within 4 hours; usually one dose (0.2 mg per kilogram, up to 6–7.5 mg total) is enough, though a second dose can be given if uric acid stays high.
5. Used in high-risk patients (very high white blood cell count above 50,000–100,000, large bulky lymphoma especially Burkitt, high LDH above twice normal, or kidney problems before chemo starts) and in patients who already have laboratory or clinical tumor lysis syndrome.

SAFETY WATCH

NEVER give to patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency – rasburicase will trigger severe breakdown of red blood cells (hemolysis), causing life-threatening anemia; check G6PD before the first dose in high-risk populations (African, Mediterranean, Middle Eastern, Southeast Asian ancestry). · Can cause severe allergic reactions (anaphylaxis) – have epinephrine ready and watch the patient closely during and after the infusion. · The hydrogen peroxide made when rasburicase breaks down uric acid can cause methemoglobinemia (blood that cannot carry oxygen well) – stop drug if the patient turns blue or has trouble breathing despite oxygen. · Blood samples to measure uric acid must be put on ice immediately and tested within 4 hours, or the rasburicase enzyme still in the tube will falsely lower the result.

Phosphate binders (sevelamer, lanthanum, calcium acetate) · Intestinal phosphate binders

Stick to phosphate in the stomach and intestines so the phosphate leaves the body in stool instead of being absorbed into the blood, lowering blood phosphate levels when the kidneys cannot clear it fast enough.

1. Tumor lysis releases huge amounts of phosphate from dying cancer cells into the blood.
2. Normally the kidneys filter out extra phosphate, but in tumor lysis the flood is too large and the kidneys may already be damaged, so phosphate stays high.
3. Phosphate binders are taken by mouth with meals; they chemically bind to phosphate in food and phosphate secreted into the gut, forming a clump that cannot cross the intestinal wall.
4. The phosphate-binder clump travels through the intestines and leaves in the stool, lowering the total phosphate load in the body.
5. Sevelamer and lanthanum do not contain calcium; calcium acetate does, so sevelamer or lanthanum are preferred when phosphate is very high to avoid making calcium-phosphate crystals worse.

SAFETY WATCH

Avoid calcium-based binders (calcium acetate, calcium carbonate) when phosphate is very high – calcium plus phosphate can form crystals that deposit in blood vessels, heart, kidneys, and soft tissues, causing serious damage. · Can cause constipation or upset stomach; take with meals and drink plenty of water. · Monitor calcium and phosphate levels daily – goal is to bring phosphate down without driving calcium too low or too high.

Insulin with dextrose (glucose) · Hormone plus sugar solution (for high potassium (hyperkalemia))

Insulin opens doors on cell surfaces and shoves potassium from the blood back into cells temporarily, and the dextrose is given at the same time to prevent blood sugar from dropping dangerously low.

1. Tumor lysis dumps potassium into the blood when millions of cancer cells rupture and spill their contents.
2. High blood potassium (hyperkalemia) can stop the heart or cause a deadly irregular rhythm.
3. Insulin (usually 10 units given as an IV push) is injected into a vein; it binds to insulin receptors on cells and activates the sodium-potassium pump, which pulls potassium ions into the cells and pushes sodium out.
4. Potassium moves out of the blood and into cells within 15–30 minutes, lowering blood potassium by 0.5–1.5 mEq/L for 4–6 hours.
5. Dextrose (25–50 grams, often as one or two amps of D50) is given with the insulin to keep blood sugar from crashing, since insulin also drives sugar into cells.
6. This is a temporary fix – it shifts potassium but does not remove it from the body; dialysis or other treatments are needed to get rid of the extra potassium for good.

SAFETY WATCH

Check blood sugar before, during, and after – insulin can cause severe low blood sugar (hypoglycemia) with confusion, sweating, shaking, or seizures; keep sugar or juice nearby. · Recheck potassium every 1–2 hours; potassium will drift back up when insulin wears off. · Watch for signs of too much insulin – give extra dextrose if blood sugar drops below 70 mg/dL.

Sodium polystyrene sulfonate (Kayexalate) or patiromer or sodium zirconium cyclosilicate · Potassium binders

Trade sodium (or other cations) for potassium in the intestines, trapping potassium so it leaves the body in stool instead of being reabsorbed into the blood.

1. Potassium binders are powders or suspensions taken by mouth or given as an enema.
2. In the intestines, the resin releases sodium (or calcium, hydrogen, depending on the binder) and grabs potassium ions in exchange.
3. The potassium-resin complex cannot be absorbed, so it travels through the colon and is eliminated in stool.

4. Sodium polystyrene sulfonate (Kayexalate) works in 2–6 hours if given by mouth with sorbitol (a laxative), faster if given as a retention enema; newer agents (patiromer, sodium zirconium cyclosilicate) work within 4–7 hours and are better tolerated.
5. Used for mild to moderate high potassium (hyperkalemia) or to keep potassium from creeping back up after insulin/dextrose.

SAFETY WATCH

Sodium polystyrene sulfonate with sorbitol can cause bowel tissue death (necrosis) (death of intestinal tissue), especially in patients after surgery or with slow gut motility – never use sorbitol in post-op patients or those with a stalled gut (ileus); watch for severe belly pain or blood in stool and stop immediately. · Can cause constipation (patiromer, sodium zirconium) or diarrhea (sodium polystyrene with sorbitol); keep the patient hydrated. · Raises sodium load – watch for fluid overload in patients with heart or kidney failure. · Recheck potassium daily to make sure it is coming down and not dropping too low.

Calcium gluconate (IV) · Membrane stabilizer (electrolyte replacement)

Does not lower potassium, but raises the threshold at which the heart's electrical cells fire, protecting the heart from dangerous rhythms caused by high potassium – like turning up the volume knob so a loud noise does not blow the speaker.

1. High potassium makes heart muscle cells too excitable and can cause the heart to stop or beat chaotically (ventricular fibrillation, asystole).
2. Calcium does not remove or move potassium – instead, it stabilizes the membrane of heart cells by shifting the threshold potential (the voltage needed for the cell to fire).
3. Calcium gluconate (1–2 grams, or 10–20 mL of 10% solution) is injected slowly into a vein over 2–5 minutes.
4. Within 1–3 minutes, the heart is protected; the effect lasts 30–60 minutes.
5. This buys time for other treatments (insulin, dialysis, potassium binders) to actually lower the potassium level.
6. Calcium gluconate is preferred over calcium chloride because it is less irritating to veins.

SAFETY WATCH

Do NOT give calcium if phosphate is very high (above 6–7 mg/dL) unless the patient is in immediate danger of death from high potassium (hyperkalemia) – calcium plus phosphate can form crystals that deposit in blood vessels, heart, lungs, and kidneys, causing strokes, heart attacks, and organ failure. · If calcium must be given despite high phosphate, use the smallest effective dose, give it slowly, and start treatments to lower phosphate immediately. · Monitor the ECG (heart tracing) during injection – too much or too fast can cause a slow heart rate (bradycardia) (slow heart rate) or arrhythmias. · Extravasation (leaking out of the vein into tissue) can cause tissue damage; make sure the IV is working well before injecting.

Hemodialysis or continuous renal replacement therapy (CRRT) ·**Extracorporeal blood purification (not a drug)**

Physically removes potassium, phosphate, and uric acid from the blood by pumping it outside the body through a filter (artificial kidney) that separates waste chemicals and extra fluid, then returns the cleaned blood – the fastest and most complete way to clear tumor lysis waste when kidneys fail or chemicals rise dangerously high.

1. A large catheter (tube) is placed in a neck, chest, or groin vein; blood is pumped out of the body into the dialysis machine.
2. The blood flows on one side of a thin membrane; on the other side flows dialysate, a specially balanced fluid.
3. Potassium, phosphate, uric acid, and other waste molecules move across the membrane from blood into dialysate (down their concentration gradient); water is pulled off if the patient is fluid-overloaded.
4. The cleaned blood is pumped back into the body.
5. Hemodialysis is done in 3–4 hour sessions, usually daily or even twice daily in severe tumor lysis; CRRT runs continuously 24 hours a day and is gentler but requires ICU care.
6. Indications: severe high potassium (hyperkalemia) (potassium above 6.5–7.0 mEq/L) not responding to medical treatment, severe hyperphosphatemia, kidney failure (rising creatinine, low urine output), fluid overload, or uric acid above 10 mg/dL with kidney injury.

SAFETY WATCH

Watch blood pressure closely during and after dialysis – rapid fluid removal can cause dangerous drops in blood pressure. · Monitor for bleeding – patients receive heparin or citrate to keep blood from clotting in the machine. · Catheter site can become infected – keep dressing clean and dry, check for redness, pus, or fever. · Recheck potassium, phosphate, calcium, and uric acid every 4–6 hours and after each dialysis session – levels can rebound when the next wave of tumor cells dies. · Dialysis removes some chemotherapy drugs; coordinate timing with oncology team.

Red flags

- Heart rhythm suddenly becomes very slow, very fast, or stops (from dangerously high potassium)
- Urine output drops to almost nothing within hours of starting cancer treatment
- Seizure or severe muscle spasms (tetany) that won't stop

Nursing priorities

1. Place patient on continuous cardiac monitoring and check potassium level immediately if any arrhythmia appears — High potassium (Hyperkalemia) (high potassium) from dying tumor cells causes peaked T waves then wide QRS complexes on EKG and can lead to ventricular fibrillation and cardiac arrest within minutes
2. Measure intake and output every hour and notify the provider if urine output drops below 100 mL per hour or 2 mL per kilogram per hour in children — The kidneys must flush out huge amounts of uric acid, potassium, and phosphate; decreased urine output means uric acid crystals are blocking kidney tubules and acute kidney failure is starting
3. Run IV normal saline at the ordered rate of 3 liters per square meter per day (usually 200 to 250 mL per hour in adults) without stopping for bathroom breaks — High-volume hydration dilutes uric acid and other toxins in the blood and kidney tubules so crystals cannot form; also increases urine flow to wash out the metabolic waste before it damages kidneys
4. Check basic metabolic panel (electrolytes, creatinine, calcium, phosphate) and uric acid level every 4 to 6 hours during the first 48 hours after chemotherapy — Potassium, phosphate, and uric acid rise within hours as billions of tumor cells lyse open; trending labs every few hours catches dangerous electrolyte shifts before they cause arrhythmia or seizures

5. Hold all potassium-containing IV fluids and medications and remove high-potassium foods from the meal tray — Dying tumor cells release massive amounts of intracellular potassium into the bloodstream so adding any extra potassium can push levels into the lethal range above 6.5 mEq per liter
6. Administer rasburicase (or allopurinol) exactly as ordered and do not delay doses — Rasburicase is an enzyme that breaks down uric acid into allantoin which dissolves easily in urine and prevents uric acid nephropathy (kidney damage from crystals); allopurinol stops new uric acid from forming but works slower
7. Do not give calcium replacement unless the patient has tetany or seizures and only after notifying provider, because phosphate level must be lowered first — Giving calcium when phosphate is high causes calcium-phosphate crystals to deposit in kidneys, heart, lungs, and blood vessels leading to tissue death; calcium only binds more phosphate and makes organ damage worse
8. Prepare for emergency hemodialysis by checking if a temporary dialysis catheter is in place and notifying nephrology if potassium is above 6 mEq per liter despite treatment — When kidneys fail or electrolyte levels rise too fast for medications to control, hemodialysis is the only way to quickly remove potassium, phosphate, and uric acid before they cause cardiac arrest or multi-organ failure

Sources

- British Society for Haematology (BSH) 2015 TLS guideline
- Cairo-Bishop TLS classification (Journal of Clinical Oncology 2004, updated consensus)
- National Cancer Institute (NCI) PDQ Tumor Lysis Syndrome
- American Society of Clinical Oncology (ASCO) supportive care guidelines

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