

Hypophosphatemia and Hyperphosphatemia

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THE STORY IN ONE SENTENCE

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“Low phosphorus starves every cell of energy and oxygen. High phosphorus pulls calcium out of blood and deposits it where it doesn't belong.”

Normal anatomy — the body's job

Phosphorus (as phosphate) is the backbone of ATP, the energy molecule every cell burns to do work. It also builds 2,3-DPG inside red blood cells, which is the chemical switch that lets oxygen drop off at tissues instead of staying stuck to hemoglobin. Most phosphorus in the body sits inside cells or locked in bone crystal. The kidneys keep blood phosphorus in a narrow safe range (2.5–4.5 mg/dL) by filtering it and either throwing it out in urine or pulling it back depending on what the body needs.

BODY'S JOB

Phosphate's job is to fuel every cell (as part of ATP), let red blood cells deliver oxygen to tissues (as 2,3-DPG), and give bones their hard structure (as calcium-phosphate crystal). Without it, cells cannot make energy, oxygen stays stuck in the blood instead of feeding tissues, and bones soften.

1. You eat phosphorus in protein foods (meat, dairy, beans); the small intestine absorbs it into the blood.
2. Blood carries phosphate to every cell, where it is built into ATP (the molecule that stores and releases energy when the cell needs to do work).
3. Inside red blood cells, phosphate forms 2,3-DPG, the molecule that loosens oxygen's grip on hemoglobin so tissues can pull oxygen off and use it.
4. Bone stores most of the body's phosphorus as calcium-phosphate crystal, which makes bone hard and acts as a reserve tank when blood levels drop.
5. The kidneys filter phosphate out of blood and either dump it in urine or pull it back into blood, depending on signals from parathyroid hormone (PTH) and vitamin D.
6. Normal blood phosphorus stays between 2.5 and 4.5 mg/dL; the kidneys tighten or loosen the reabsorption valve to keep it there.

What breaks

Low phosphorus (hypophosphatemia, under 2.5 mg/dL) happens when phosphate shifts from blood into cells all at once, the kidneys throw out too much, or the gut cannot absorb it. High phosphorus (hyperphosphatemia, over 4.5 mg/dL) happens when the kidneys cannot clear it (kidney failure) or a huge number of cells die and dump their inside contents into the blood (tumor lysis syndrome, rhabdomyolysis).

CLINICAL TERM

Hypophosphatemia results from transcellular shift (refeeding syndrome, treatment of diabetic ketoacidosis (the acid crisis of missing insulin), respiratory alkalosis (blowing off too much carbon dioxide)), renal wasting (primary hyperparathyroidism, Fanconi syndrome, diuretics), or gastrointestinal loss (chronic diarrhea, malabsorption, phosphate binders). Hyperphosphatemia results from decreased renal excretion (acute or chronic kidney disease, hypoparathyroidism) or increased phosphate load (tumor lysis syndrome, rhabdomyolysis, massive red blood cell destruction (hemolysis)).

How the body compensates

The body tries to fix low phosphorus by pulling it out of bone and shifting it between compartments, and it tries to fix high phosphorus by depositing calcium-phosphate crystals in soft tissues and raising PTH to push more out through the kidneys. These fixes work for a little while, but they cause secondary problems (weak bones, soft-tissue calcification, muscle spasms) and eventually fail when the underlying cause is not corrected. The danger is that vital signs and labs can look stable during compensation, so the team misses the patient sliding toward respiratory failure (low phosphorus) or cardiac arrest from low calcium (high phosphorus).

1. In hypophosphatemia, the body first shifts phosphate out of bone and from inside cells to blood to keep the blood level from dropping further.
2. The kidneys reduce phosphate loss in urine by increasing reabsorption in the proximal tubule (helped by low PTH and active vitamin D).
3. This compensation holds blood phosphorus closer to normal for a few days, so labs may not show severe hypophosphatemia until the patient is already weak, confused, or struggling to breathe.
4. Once total body phosphorus is depleted, cells cannot make enough ATP or 2,3-DPG. Muscles (including the diaphragm) lose strength, red blood cells hemolyze, and the heart weakens. Vital signs may still look okay until the patient suddenly cannot breathe.
5. In hyperphosphatemia, the body tries to lower phosphorus by raising PTH (secondary hyperparathyroidism), which tells the kidneys to dump more phosphate in urine.
6. If the kidneys cannot respond (because of kidney failure), phosphorus binds calcium in the blood and forms calcium-phosphate crystals that deposit in blood vessels, heart valves, skin, and joints.
7. Blood calcium drops (hypocalcemia), which causes muscle cramps, spasms (tetany), and can trigger dangerous heart rhythms (prolonged QT interval).
8. The body also deposits extra phosphorus in bone (renal osteodystrophy), which weakens the skeleton over time.
9. Watch for the crash point: in hypophosphatemia, respiratory failure (diaphragm too weak to breathe) or rhabdomyolysis (muscle breakdown releases more toxins). In hyperphosphatemia, severe low calcium (hypocalcemia) causing seizures, tetany, or lethal arrhythmia.
10. A patient who looks stable (normal heart rate, normal blood pressure) but has a phosphorus of 1.2 mg/dL and is getting weaker each hour is not stable — they are on the edge of respiratory arrest.
11. Trend the labs and the exam together. If strength is fading or calcium is dropping despite treatment, the compensation is failing and the window to reverse the crash is closing.

Domino effect

1. Phosphate drops below 1.5 mg/dL (severe hypophosphatemia), so cells cannot make enough ATP (the energy molecule) or 2,3-DPG (the oxygen-release switch in red blood cells). — ATP is built from adenosine plus three phosphate groups; without phosphate, cells run out of fuel and cannot do work. 2,3-DPG needs phosphate to form; without it, hemoglobin holds onto oxygen too tightly and tissues starve even though blood oxygen saturation looks normal.
2. Muscles weaken because they cannot make ATP to power contraction. The diaphragm (main breathing muscle) fatigues and the patient develops shallow breathing, then respiratory failure. — Every muscle squeeze needs ATP. When phosphate is gone, muscle fibers cannot contract hard or long. The diaphragm works nonstop, so it is the first to fail — breathing becomes weak, carbon dioxide builds up, and the patient may need a ventilator.
3. Red blood cells hemolyze (break apart) because low ATP weakens the cell membrane and low 2,3-DPG disrupts the membrane skeleton. — Red blood cell membranes are held together by a phosphate-dependent protein scaffold. Without enough ATP and phosphate, the membrane becomes fragile and bursts, releasing hemoglobin into the blood (hemolysis), which can cause anemia and dark urine.
4. Rhabdomyolysis develops: skeletal muscle cells break down, releasing myoglobin, creatine kinase, potassium, and more phosphate into the blood. — Starved muscle cells cannot maintain their structure without ATP. They dissolve (rhabdomyolysis), which dumps toxic contents into the bloodstream. Myoglobin clogs the kidney tubules and can cause acute kidney injury.
5. Brain function declines (confusion, irritability, seizures, coma) because neurons need ATP to fire and maintain ion gradients. — The brain burns more ATP per gram than any other organ. Without phosphate, neurons cannot pump sodium and potassium across membranes, which disrupts electrical signals. Patients become confused, agitated, or unresponsive.
6. In hyperphosphatemia, phosphate binds calcium in the blood, forming calcium-phosphate precipitates. Blood calcium drops (hypocalcemia). — Phosphate and calcium stick together chemically. When phosphate is high, it pulls calcium out of solution. The calcium-phosphate crystals deposit in soft tissues (blood vessels, heart, skin, joints) and the free calcium left in blood falls dangerously low.
7. Low calcium causes tetany (muscle spasms and cramps), especially in the hands and face (Chvostek sign, Trousseau sign), numbness and tingling around the mouth and fingers, and prolonged QT interval on ECG (risk of lethal arrhythmia). — Calcium stabilizes nerve and muscle membranes. When it drops, nerves fire too easily (hyperexcitability), causing spasms, twitching, and abnormal heart rhythms. Severe low calcium (hypocalcemia) can trigger seizures or sudden cardiac arrest.

- 8.** Calcium-phosphate crystals deposit in soft tissues: blood vessels stiffen (vascular calcification), heart valves harden, skin itches and hardens (calcinosis cutis), and joints become painful. — High phosphorus drives calcium out of bones and blood into places it does not belong. Over time, these deposits narrow arteries (raising the risk of heart attack and stroke), damage heart valves, and cause severe itching and joint pain that do not respond to usual treatments.

Symptoms · what the patient feels, and why

- Muscle weakness or pain: Phosphate is the fuel inside every muscle cell that powers movement (it makes ATP, the cell's energy coin). When phosphate drops too low, muscles run out of energy and feel weak or achy. When phosphate climbs too high, extra calcium-phosphate crystals can form in muscle tissue and hurt.
- Confusion, irritability, or trouble thinking clearly: Brain cells need steady phosphate to make energy and keep their outer walls healthy. Low phosphate starves the brain and makes thinking foggy. High phosphate can raise calcium inside brain cells and throw off their electrical signals, causing confusion or crankiness.
- Numbness or tingling in hands and feet: When phosphate is very low, red blood cells lose a chemical called 2,3-DPG (a helper that nudges oxygen off hemoglobin). Without enough 2,3-DPG, oxygen sticks tight to hemoglobin and nerves do not get the oxygen they need, so they send tingly warning signals. High phosphate can also lower blood calcium, making nerves fire randomly and causing tingling.
- Bone pain or easy fractures: Bones are a living bank of calcium and phosphate. Long-term low phosphate (often from not enough vitamin D or kidney phosphate wasting) softens bones because new bone cannot harden properly. Long-term high phosphate (usually from failing kidneys) pulls calcium out of bone to keep blood levels balanced, weakening the skeleton over time.
- Heart rhythm changes or palpitations: The heart's electrical system depends on balanced phosphate, calcium, and potassium. Severe low phosphate weakens heart-muscle squeeze and can trigger irregular beats. Very high phosphate lowers blood calcium, which also disrupts the heart's rhythm and squeeze.
- Trouble breathing or needing the ventilator longer: The diaphragm (the big breathing muscle under your lungs) is a muscle like any other and needs phosphate for energy. Severe low phosphate makes the diaphragm too weak to pull air in well, so breathing becomes hard or shallow.
- Seizures: Very low phosphate starves brain cells of energy and oxygen, making them misfire all at once (a seizure). Very high phosphate can drop blood calcium so low that brain cells become jumpy and fire uncontrollably.

- Red blood cells breaking apart (hemolysis): Red blood cells need phosphate to keep their outer membrane flexible. When phosphate drops critically low, the membrane becomes stiff and cracks open, spilling hemoglobin into the blood (hemolytic anemia).

Focused exam

- Muscle weakness or floppiness, especially in the legs: Phosphate is the spark plug for muscle energy—it builds ATP (the fuel cells burn to move). When phosphate drops too low (hypophosphatemia), muscles run out of power and stop working. This is the hallmark sign you cannot miss.
- Shallow breathing or stopped breathing, especially if the person also looks tired or drowsy: The diaphragm (the big breathing muscle under your lungs) needs phosphate to contract. In severe hypophosphatemia, it weakens and may stop moving altogether, shutting down breathing. This can kill the patient quickly.
- Red blood cells bursting (hemolysis on blood smear, or rising bilirubin and falling hemoglobin): Red blood cells need phosphate to make 2,3-DPG (a chemical that helps them hold oxygen and keep their shape). Without phosphate, the cells lose their bend and pop open, spilling their insides.
- Confusion, seizures, or trouble staying awake: The brain runs on ATP, and neurons (brain cells) need phosphate to make it. Severe hypophosphatemia starves the brain of energy, breaking down thinking, memory, and even consciousness. This is a late, dangerous sign.
- Muscle pain and dark brown urine (signs of rhabdomyolysis—muscle breakdown): Muscles without phosphate cannot hold themselves together and fall apart. The broken muscle pieces spill myoglobin (a muscle protein) into the blood, turning urine brown and clogging the kidney filters. This is hypophosphatemia's signature muscle disaster.
- Stiff muscles, tingling around the mouth or fingers, and low calcium (in hyperphosphatemia): High phosphate grabs calcium out of the blood and locks it up in bone and soft tissue. The blood runs low on calcium (hypocalcemia), and muscles lock up or twitch (tetany). This is hyperphosphatemia's partner problem.
- White or chalky deposits in the skin, eyes (band keratopathy—white ring on the cornea), or around joints (in chronic hyperphosphatemia): When phosphate stays too high for weeks or months, calcium and phosphate form crystals and harden soft tissues everywhere—skin, blood vessels, heart valves, and eyes. This is metastatic calcification, the slow damage hyperphosphatemia leaves behind.

Diagnostics · every test answers a question

- Serum phosphate (phosphorus) level → Is phosphate too low or too high, and how severe is it?

- Serum calcium (total and ionized if available) → Is calcium low because high phosphate is pulling it out of the blood, or is calcium high because phosphate is low?
- Serum magnesium → Is magnesium also low, blocking phosphate from staying in cells?
- Basic metabolic panel (BMP) with creatinine and eGFR → Are the kidneys working well enough to balance phosphate, or is kidney failure letting phosphate pile up?
- Creatine kinase (CK) → Are muscles breaking down from phosphate deficiency?
- 24-hour urine phosphate or fractional excretion of phosphate (FEphos) → Is the kidney throwing out too much phosphate (renal wasting), or is the kidney trying to hold onto it (normal response to low phosphate)?
- Parathyroid hormone (PTH) level → Is the parathyroid gland too active, telling the kidneys to dump phosphate into the urine?
- Lactate dehydrogenase (LDH) and uric acid (if tumor lysis syndrome is suspected) → Are cancer cells dying all at once and dumping their phosphate into the blood?

Treatment logic

- IV sodium phosphate or potassium phosphate 0.16–0.32 mmol/kg (roughly 15–30 mmol total) over 4–6 hours for severe hypophosphatemia (phosphate < 1.0 mg/dL) → Replaces the missing phosphate so cells can make ATP again, muscles get the fuel they need to contract, and red blood cells stop bursting. This lifts phosphate back into the safe zone (≥ 2.5 mg/dL) within hours.
- Stop or slow refeeding; restart nutrition very slowly with small amounts (start 10–20 kcal/kg/day, advance slowly over 4–7 days) and give thiamine, phosphate, magnesium, and potassium from day one → When you feed a starving body too fast, cells suck phosphate (and potassium and magnesium) out of the blood to restart their engines. Slowing down and pre-loading electrolytes lets phosphate stay in the blood long enough for the body to balance it safely. This prevents refeeding syndrome.
- Oral phosphate supplements (Neutra-Phos or K-Phos) 1–2 grams per day in divided doses for mild to moderate hypophosphatemia (phosphate 1.0–2.5 mg/dL) if the person can swallow → Refills phosphate stores slowly through the gut without the risks of IV phosphate (which can drop calcium suddenly or deposit in the wrong places). Works well when the person is stable and phosphate is not life-threateningly low.
- For hyperphosphatemia: give phosphate binders (sevelamer 800–1600 mg, calcium acetate 667–1334 mg, or lanthanum 500–1000 mg) by mouth with every meal → These medicines stick to phosphate in food and carry it out in the stool so your body cannot absorb it. This lowers blood phosphate without the kidneys having to do all the work. Sevelamer does not add calcium; calcium acetate does (so it helps if calcium is also low, but risks making calcium too high).

- For tumor lysis syndrome: give rasburicase 0.2 mg/kg IV once, aggressive IV fluids (150–200 mL/kg/day), and avoid allopurinol if uric acid is very high; consider urgent hemodialysis if phosphate > 10 mg/dL or kidneys are failing → Rasburicase breaks down uric acid so the kidneys do not get clogged by uric acid crystals, and IV fluids flush phosphate and other cell debris out through urine. This stops the sudden phosphate spike when cancer cells die all at once and dump their insides. Dialysis directly pulls phosphate out of the blood when kidneys cannot handle the load.
- Treat chronic kidney disease (CKD) with dietary phosphate restriction (≤ 800 – 1000 mg/day), phosphate binders, and dialysis if needed (stage 5 CKD, $\text{eGFR} < 15$ mL/min/1.73m²) → Healthy kidneys throw out extra phosphate when you have too much and hold onto phosphate when you have too little. When kidneys fail, phosphate piles up. Limiting phosphate in food, blocking absorption with binders, and clearing it with dialysis keeps phosphate from climbing into the danger zone (> 5.5 mg/dL in CKD).
- Give IV calcium gluconate 1–2 grams (10–20 mL of 10% solution) over 10 minutes if calcium is very low (< 7.5 mg/dL) and muscles are twitching or locking up (tetany) because of high phosphate → Calcium stops the muscle twitching and locking right away by calming the nerves and muscle cells. This buys time while you lower the phosphate with binders or dialysis.

Medications

Intravenous (IV) sodium phosphate or potassium phosphate · Phosphate replacement (intravenous)

Delivers phosphate straight into the bloodstream to quickly raise the blood phosphate level back toward normal. Phosphate is a mineral the body needs to make energy inside every cell, keep bones strong, and help nerves and muscles work. When phosphate drops too low (hypophosphatemia), cells can't make enough ATP (the molecule cells burn for energy), so muscles get weak, the heart can struggle, and in severe cases the person can become confused or have trouble breathing. IV phosphate skips the gut and refills the blood supply directly, so cells can grab what they need right away.

1. The nurse hangs a bag of sodium phosphate or potassium phosphate and infuses it slowly through an IV line, usually over 4 to 6 hours.
2. Phosphate ions enter the bloodstream and spread through the circulation.
3. Cells pull phosphate from the blood and use it to make ATP, the energy molecule that powers almost every job inside the cell.
4. Red blood cells use phosphate to make 2,3-DPG (a molecule that helps hemoglobin release oxygen to tissues), so oxygen delivery improves.

5. Muscles – including the diaphragm (the breathing muscle) and the heart – regain strength as their energy supply is restored.
6. The kidneys adjust how much phosphate they keep or let go, depending on the body's new balance.

SAFETY WATCH

Infuse slowly (usually 15 to 30 millimoles over 4 to 6 hours) – pushing it too fast can cause a sudden spike in blood phosphate, which can drop calcium dangerously low and trigger muscle spasms, seizures, or an irregular heartbeat. · Check calcium level before and during treatment – phosphate binds to calcium in the blood, so giving phosphate can lower calcium (hypocalcemia). If calcium drops too much, the patient may need calcium replacement. · Watch potassium if using potassium phosphate – patients with kidney problems or those already on the higher side of potassium can develop dangerous high potassium (hyperkalemia) (too much potassium), which can stop the heart. · Monitor kidney function – if the kidneys are not working well, they may not be able to clear excess phosphate, raising the risk of giving too much. · Avoid giving phosphate into a vein that also has calcium running – calcium and phosphate can combine and form crystals that clog the IV line or, worse, deposit in blood vessels and soft tissues. · Check magnesium – low magnesium often goes hand-in-hand with low phosphate and makes it harder for cells to hold onto phosphate. Replacing magnesium at the same time helps the phosphate replacement work better.

Oral phosphate supplements (sodium phosphate or potassium phosphate tablets or liquid) · Phosphate replacement (oral)

Raises blood phosphate by giving phosphate through the mouth, which the gut absorbs into the bloodstream. Used when phosphate is low but not dangerously low (mild to moderate hypophosphatemia) and the person can swallow and absorb food normally. Oral replacement is slower and gentler than IV but works well when there is time and the gut is healthy.

1. The patient swallows phosphate tablets or liquid, usually two to three times a day.
2. The phosphate moves through the stomach into the small intestine.
3. Cells lining the small intestine absorb phosphate into the bloodstream.
4. Blood phosphate level gradually rises over hours to days.
5. Cells pull phosphate from the blood to make ATP, restore energy, and support normal muscle, nerve, and bone function.
6. The kidneys sense the rising phosphate level and adjust how much they hold onto or release in the urine.

SAFETY WATCH

Common side effect is diarrhea – phosphate pulls water into the gut, so loose stools are frequent. Taking smaller doses more often, or switching formulations, can help. · Nausea and stomach cramps can occur – taking the supplement with food may ease stomach upset. · Watch kidney function – if the kidneys are weak, oral phosphate can build up and cause hyperphosphatemia (too much phosphate), which can lead to calcium deposits in soft tissues and blood vessels. · Check calcium level – taking phosphate can lower calcium in the blood; if the patient develops tingling around the mouth, muscle cramps, or twitching, check calcium. · Monitor potassium if using potassium phosphate – too much potassium can be dangerous, especially in people with kidney problems or those taking certain blood pressure medicines (ACE inhibitors, ARBs, potassium-sparing diuretics). · Follow the prescribed dose – taking too much phosphate at once increases the risk of diarrhea, nausea, and dangerous shifts in calcium and potassium.

Sevelamer · Phosphate binder (non-calcium-based)

Binds to phosphate in the food you eat so your gut cannot absorb it, and the bound phosphate leaves the body in stool instead. Used in hyperphosphatemia (high blood phosphate), most commonly in people with chronic kidney disease whose kidneys cannot filter out enough phosphate. By blocking phosphate absorption, sevelamer keeps blood phosphate from climbing even higher, which protects blood vessels and soft tissues from dangerous calcium-phosphate deposits.

1. The patient swallows sevelamer tablets or powder with every meal and snack that contains phosphate (meat, dairy, beans, nuts, soda, processed foods).
2. Sevelamer travels through the stomach into the small intestine.
3. The drug's polymer chains grab onto phosphate ions released from food during digestion.
4. The sevelamer-phosphate clump is too big to be absorbed, so it moves through the intestine and exits in the stool.
5. Less phosphate enters the bloodstream, so blood phosphate level drops or stays stable.
6. Over weeks to months, lower blood phosphate reduces the risk of calcium-phosphate crystals forming in blood vessels, heart valves, skin, and other soft tissues.

SAFETY WATCH

Must be taken WITH meals or snacks – if you take it on an empty stomach or hours after eating, it will not bind phosphate and will not work. · Common side effects: nausea, vomiting, diarrhea, constipation, and stomach pain – starting with a lower dose and slowly increasing can help the gut adjust. · Can lower levels of fat-soluble vitamins (A, D, E, K) and folic acid over time – patients on sevelamer long-term may need vitamin supplements, especially if they are also on dialysis. · Does NOT contain calcium (unlike older binders like calcium acetate), so it does NOT raise calcium levels – this makes it safer for patients who already have high calcium or vascular calcification. · Can interfere with absorption of some other medications (like ciprofloxacin, levothyroxine) – take those medicines at least one hour before or three hours after sevelamer. · Expensive compared to older phosphate binders – insurance coverage varies, so cost can be a barrier for some patients.

Calcium acetate · Phosphate binder (calcium-based)

Binds to phosphate in food inside the stomach and intestine so the body cannot absorb it, and the bound phosphate is eliminated in stool. Used in hyperphosphatemia, especially in chronic kidney disease. At the same time, the calcium part of the pill is absorbed, which can help if the patient is also low in calcium – but it can be a problem if calcium is already normal or high because too much calcium in the blood can deposit in arteries and heart valves.

1. The patient takes calcium acetate tablets with every meal and phosphate-containing snack.
2. In the stomach, the pill dissolves and releases calcium ions.
3. Calcium ions bind tightly to phosphate ions from food, creating calcium phosphate crystals.
4. These crystals are too large to pass through the intestinal wall, so they travel through the gut and exit in the stool.
5. Less phosphate enters the blood, lowering or stabilizing the blood phosphate level.
6. A portion of the calcium is absorbed, which can be helpful if blood calcium is low but risky if it is normal or high.

SAFETY WATCH

Must be taken WITH meals – timing is critical; if you take it hours before or after eating, it will not bind dietary phosphate. · Can raise blood calcium (hypercalcemia) – symptoms include nausea, vomiting, confusion, extreme thirst, and bone pain. High calcium plus high phosphate together greatly increase the risk of calcium deposits forming in arteries, heart, lungs, and skin. · Not safe if calcium or calcium-phosphate product (calcium × phosphate) is already high – in those cases, a non-calcium binder like sevelamer is preferred. · Common side effects: constipation, nausea, upset stomach – taking with food and drinking plenty of water can help. · Do NOT take calcium acetate at the same time as iron supplements, certain antibiotics (tetracyclines, fluoroquinolones), or levothyroxine – calcium interferes with their absorption. Space them at least two hours apart. · Frequent monitoring of calcium and phosphate levels is needed – the dose often has to be adjusted to keep both in a safe range.

Rasburicase · Recombinant urate oxidase enzyme

Breaks down uric acid (a waste product) into a more soluble compound that the kidneys can easily clear. Used to prevent or treat tumor lysis syndrome (TLS) – a dangerous condition that happens when cancer treatment causes a huge number of tumor cells to burst open all at once, dumping potassium, phosphate, and uric acid into the blood faster than the kidneys can handle. The flood of phosphate can cause severe hyperphosphatemia, kidney failure, and dangerous heart rhythms. Rasburicase lowers uric acid quickly, which helps protect the kidneys so they can better clear the phosphate and other wastes.

1. The patient (usually someone with a high-burden blood cancer like leukemia or lymphoma about to start chemotherapy) receives a single dose of rasburicase by slow IV infusion before or right after the first chemo dose.
2. Rasburicase enters the bloodstream and begins breaking down uric acid into allantoin.
3. Uric acid levels drop within hours – often dramatically – because allantoin is five to ten times more soluble than uric acid and washes out in the urine.
4. With uric acid out of the way, the kidneys are less likely to become blocked by uric acid crystals, so they can keep filtering and removing the massive phosphate load released from tumor cells.
5. Blood phosphate, potassium, and other electrolytes are monitored closely; IV fluids, phosphate binders, and sometimes dialysis are added as needed to manage the remaining imbalances.

SAFETY WATCH

Absolutely contraindicated in patients with G6PD deficiency (glucose-6-phosphate dehydrogenase deficiency, a genetic condition common in people of African, Mediterranean, or Asian descent) – rasburicase causes severe breakdown of red blood cells (hemolysis) in these patients, which can be life-threatening. Screen for G6PD before giving rasburicase. · Can trigger severe allergic reactions (anaphylaxis) – watch for rash, difficulty breathing, chest tightness, or swelling of the face or throat, especially in patients receiving more than one dose. · Blood samples for uric acid must be placed immediately on ice and processed quickly – rasburicase continues to break down uric acid in the test tube, so a room-temperature sample will show a falsely low uric acid level. · Methemoglobinemia (a condition where red blood cells cannot carry oxygen properly) is a rare but serious side effect – watch for bluish skin, shortness of breath, and confusion. · Not safe in pregnancy – rasburicase is classified as pregnancy category C; use only if absolutely necessary. · Usually given as a single dose – repeated doses increase the risk of antibody formation and allergic reactions.

Red flags

- Seizures or sudden confusion
- Severe muscle weakness or trouble breathing
- Heart rhythm changes or chest pain
- Red or brown urine (sign that red blood cells are breaking apart)

Nursing priorities

1. Watch breathing rate, depth, and oxygen saturation every 1 to 2 hours during treatment for severe low phosphorus below 1.0 mg/dL — Low phosphorus weakens the diaphragm (main breathing muscle) and can cause respiratory failure (stopped breathing) requiring a ventilator
2. Check the IV phosphate infusion rate every 30 minutes and never give faster than 7.5 mmol per hour — Too-fast IV phosphate drops calcium suddenly causing tetany (muscle spasms), seizures, or cardiac arrest (stopped heart); phosphate must go in slowly over 4 to 6 hours
3. Monitor heart rhythm on telemetry (heart monitor) continuously during phosphate replacement — Low phosphorus causes arrhythmias (irregular heartbeats) and giving phosphate too fast drops calcium which also causes dangerous arrhythmias and QT prolongation (electrical delay that triggers sudden death)

4. Measure phosphorus, calcium, magnesium, and potassium levels every 6 hours during active treatment — Phosphorus and calcium move in opposite directions; replacing phosphorus can drop calcium dangerously low, and low magnesium or potassium also need fixing or phosphorus will not stay normal
5. For high phosphorus from tumor lysis syndrome, measure strict intake and output hourly and keep urine output above 100 mL per hour with IV fluids — High phosphorus binds with calcium and forms crystals that clog kidney tubules (tiny filters) causing permanent kidney failure; high urine flow flushes the crystals out
6. Give phosphate binders like sevelamer or calcium acetate with every meal and snack for chronic high phosphorus — Phosphate binders stick to phosphorus in food inside the stomach and intestines so the body does not absorb it; they only work when taken with food
7. Restart nutrition very slowly at 10 calories per kilogram per day for malnourished or starved patients, and add thiamine 100 mg IV before any feeding — Starting nutrition too fast in starved patients triggers refeeding syndrome with severe low phosphorus, low potassium, and low magnesium; thiamine (vitamin B1) prevents Wernicke encephalopathy (brain damage from thiamine deficiency during refeeding)
8. Check for muscle pain, dark urine, or rising creatine kinase (muscle enzyme) levels that signal rhabdomyolysis — Severe low phosphorus causes rhabdomyolysis (muscle breakdown) releasing myoglobin (muscle protein) that clogs kidneys and causes permanent kidney failure requiring dialysis

Sources

- American Society of Nephrology
- National Kidney Foundation KDOQI guidelines
- UpToDate clinical reference
- New England Journal of Medicine

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