

Mg (magnesium) disorders

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THE STORY IN ONE SENTENCE

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“Magnesium disorders — low Mg drives unfixable potassium and calcium problems, high Mg paralyzes everything.”

Normal anatomy — the body's job

Magnesium sits inside every cell and steadies the electrical gates on cell walls, helps hundreds of enzyme workers run the body's chemistry, and keeps potassium and calcium in balance. Picture it as the quiet foreman making sure nerves fire cleanly, muscles contract smoothly, and the heart beats steadily.

BODY'S JOB

Magnesium does three main jobs. First, it steadies the tiny gates (ion channels) in cell walls that let sodium, potassium, and calcium flow in and out—this keeps the electrical signals in nerves and muscles smooth and predictable. Second, it is a helper (cofactor) for over 300 enzymes, so nearly every chemical reaction in the body—from making energy (ATP) to copying DNA—needs magnesium to run. Third, it works hand-in-hand with potassium and calcium: magnesium helps the kidney hold onto potassium and keeps calcium from flooding into cells and causing them to lock up. Without enough magnesium, the electrical system misfires, the enzymes slow down, and potassium and calcium slip out of control.

1. Magnesium enters the body through food (green leafy vegetables, nuts, whole grains, beans) and gets absorbed mainly in the small intestine.

2. About 99% of the body's magnesium hides inside cells and bone; only 1% floats in the blood, so blood tests can miss early shortages.
3. Inside the cell, magnesium sits on the inner side of the cell wall and blocks calcium from rushing in—this keeps muscles relaxed until a nerve signal says to contract.
4. Magnesium also plugs into enzymes like a key in a lock, letting them build energy molecules (ATP), copy genes (DNA polymerase), and run hundreds of other reactions.
5. In the kidney tubule (the tiny pipe that makes urine), magnesium helps the ROMK channel (a potassium gate) stay open so potassium flows back into the blood instead of leaking into urine.
6. The kidney normally saves magnesium in the thick ascending limb of the loop of Henle (a hairpin turn in the kidney tubule) and the distal convoluted tubule (the next winding section), keeping blood magnesium between 1.7 and 2.2 mg/dL.
7. When magnesium is balanced, nerves fire on cue, muscles contract and relax smoothly, the heart beats in steady rhythm, and potassium and calcium stay in their lanes.

What breaks

Magnesium runs low when the gut can't absorb it (long diarrhea, inflammatory bowel disease, stomach surgery) or the kidney wastes it (diuretics like furosemide, alcohol, proton-pump inhibitors like omeprazole, cisplatin chemo, or a rare genetic tubule defect called Gitelman syndrome). Magnesium climbs too high when someone gets large IV or enema doses (usually magnesium sulfate for eclampsia) and the kidneys can't clear it because of kidney failure.

CLINICAL TERM

Low magnesium (Hypomagnesemia) (low Mg, <1.7 mg/dL) from GI loss (diarrhea, malabsorption, pancreatitis) or renal wasting (loop or thiazide diuretics, alcohol, PPI, aminoglycosides, cisplatin, Gitelman or Bartter syndrome). Hypermagnesemia (high Mg, >2.2 mg/dL) from exogenous load (IV MgSO₄, magnesium-containing laxatives or antacids) plus impaired renal excretion (acute or chronic kidney disease, GFR <30 mL/min).

How the body compensates

The body has almost no good way to compensate for broken magnesium balance. When magnesium is low, the parathyroid glands (four tiny glands behind the thyroid) try to raise calcium by releasing PTH (parathyroid hormone), but low magnesium actually blocks PTH release and makes cells ignore PTH, so calcium drops too—this makes the problem worse, not better. When magnesium is high, there is no counter-hormone to push it down; the body simply

waits for the kidneys to clear it. Vital signs can look stable for a while even as magnesium drifts dangerously low or high, then suddenly the heart rhythm breaks or breathing stops.

1. When magnesium drops, the parathyroid glands sense low calcium (because magnesium and calcium track together) and try to release PTH to pull calcium from bone—but low magnesium blocks the calcium-sensing receptor on the parathyroid, so PTH release stalls.
2. At the same time, low magnesium makes bone and kidney cells resistant to whatever PTH does get out, so calcium stays low (hypocalcemia) even though the body is trying to raise it.
3. Low magnesium also opens the ROMK potassium channel in the kidney tubule too wide, so potassium leaks into urine (renal potassium wasting) and blood potassium drops (hypokalemia)—giving oral or IV potassium replaces it temporarily, but it keeps leaking out until magnesium is fixed first; this is called refractory low potassium (hypokalemia).
4. The heart tries to keep a steady rhythm, but without enough magnesium to stabilize potassium channels in heart muscle, the repolarization phase (when the heart resets electrically) drags out, lengthening the QT interval on the EKG and setting the stage for torsades de pointes (a spinning, chaotic rhythm that can stop the heart).
5. When magnesium climbs too high, it floods into the synapse (the gap between nerve and muscle) and blocks the release of acetylcholine (the go signal for muscle contraction), so muscles go limp, reflexes disappear, and the diaphragm weakens—breathing slows and can stop altogether if magnesium reaches 12–15 mg/dL.
6. Blood pressure drops because high magnesium relaxes smooth muscle in blood vessel walls (vasodilation), and the heart rate can slow (bradycardia) because magnesium blocks calcium entry into the heart's pacemaker cells.
7. The danger zone is the silent middle: a patient with magnesium of 1.0 mg/dL might feel only mild cramps and look stable, but is one stress (infection, more diuretic) away from a lethal arrhythmia; a patient with magnesium of 6 mg/dL might seem sleepy but is approaching respiratory arrest.
8. Watch the trend—stable numbers that are slowly worsening, or symptoms that are progressing (twitching → tetany → seizure; weak reflexes → no reflexes → no breathing)—not just a single lab value, because the crash point arrives suddenly once the body's narrow margin runs out.

Domino effect

1. Magnesium drops below 1.7 mg/dL, and the electrical gates (ion channels) in nerve and muscle cell walls lose their steady anchor. — Magnesium normally plugs into NMDA receptors (a type of calcium gate in nerves) and blocks extra calcium from flooding in; without enough magnesium, calcium rushes in and nerves fire over and over without a real signal—this causes muscle twitches, cramps, and spasms (tetany, where hands and feet curl up), and can trigger seizures if it reaches the brain.
2. At the same time, low magnesium opens the ROMK potassium channel in the kidney tubule too wide, so potassium pours into urine even when blood potassium is already low. — The kidney tries to save potassium by closing ROMK, but magnesium is the lock that keeps ROMK properly regulated—without it, the channel stays open and potassium leaks out (renal potassium wasting). Giving potassium by mouth or IV refills the blood temporarily, but it keeps draining into urine until magnesium is replaced first—this is refractory low potassium (hypokalemia), the hallmark clue that magnesium is the hidden problem.
3. Low magnesium also blocks PTH release from the parathyroid glands and makes bone and kidney cells ignore PTH, so calcium drops (hypocalcemia) and won't correct until magnesium is fixed. — Calcium and magnesium are partners: the calcium-sensing receptor on the parathyroid needs magnesium to work, and PTH needs magnesium to unlock calcium from bone and tell the kidney to save it. When magnesium is missing, the whole calcium-regulation system stalls, so patients get the numbness, tingling, and muscle cramps of low calcium on top of low magnesium.
4. In the heart, low magnesium and low potassium together prolong the repolarization phase (the reset between beats), stretching the QT interval on the EKG and triggering torsades de pointes—a rapid, twisting rhythm that looks like a spinning ribbon and can degenerate into cardiac arrest. — Magnesium and potassium both help close the potassium channels that reset the heart's electrical charge after each beat; without them, the reset drags out, the heart is vulnerable to extra beats (premature ventricular contractions), and one extra beat at the wrong moment can spin into torsades. This is the lethal endpoint of untreated low magnesium (hypomagnesemia).
5. When magnesium climbs above 4 mg/dL (hypermagnesemia), it floods the neuromuscular junction (the gap between nerve and muscle) and blocks calcium channels, so acetylcholine (the chemical signal to contract) can't be released—muscles go limp, deep tendon reflexes disappear, and the patient feels weak. — Muscle contraction depends on calcium flowing into the nerve terminal to trigger acetylcholine release; magnesium competes with calcium and wins when levels are high, so the signal never arrives. First the deep reflexes vanish (around 4–6 mg/dL), then skeletal muscles weaken (6–10 mg/dL), and finally the diaphragm stops contracting and breathing ceases (above 12 mg/dL)—this is why severe hypermagnesemia is a medical emergency.

6. High magnesium also relaxes smooth muscle in blood vessel walls, dropping blood pressure (hypotension), and slows the heart's pacemaker (bradycardia) by blocking calcium entry into SA node cells. — Blood vessels dilate because smooth muscle can't contract without calcium, so blood pressure falls; the SA node (the heart's natural pacemaker in the right atrium) fires more slowly because its automatic rhythm depends on calcium influx, and magnesium blocks that. If magnesium reaches very high levels, the heart can stop altogether (asystole).
7. Every one of these findings—twitching, refractory low potassium, low calcium, torsades, weak muscles, lost reflexes, slow breathing—is not the disease itself but the body's electrical and chemical system failing because magnesium, the hidden stabilizer, is missing or overwhelming the gates. — The body isn't misbehaving; it's revealing that one upstream control—magnesium—is broken. Fixing magnesium stops the whole domino line: replace magnesium and potassium stays in, calcium comes up, the heart rhythm steadies, and muscles relax. Remove excess magnesium (stop the infusion, give IV calcium to block its effects, use dialysis if kidneys can't clear it) and muscles wake up, reflexes return, blood pressure rises, and breathing restarts.
8. Trace backward from any symptom—shake, cramp, bad rhythm, weak muscle—and they all point to one broken step: magnesium homeostasis. Find the cause (diuretic, alcohol, diarrhea, PPI, kidney failure, caused by medical care (iatrogenic) load) and fix it, and the whole picture resolves. — This is the core of clinical reasoning for magnesium disorders: the constellation of findings (refractory low potassium (hypokalemia) + low calcium (hypocalcemia) + long QT, or absent reflexes (areflexia) + low blood pressure (hypotension) + respiratory depression) is not a list to memorize but a single mechanism to see—magnesium's role stabilizing ion channels and enzymes—so you recognize the pattern instantly and know exactly which upstream lever to pull.

Symptoms · what the patient feels, and why

- Muscle twitches or cramps that won't let up: Magnesium helps muscle cells relax after they squeeze. When magnesium drops too low, the cells stay jumpy and fire off even when you don't want them to—like a light switch that won't turn off. If magnesium climbs way too high, muscles get so relaxed they turn weak and floppy.
- Numbness or tingling around the mouth or in the hands and feet: Low magnesium often drags down calcium as well, because magnesium is needed to release the hormone (PTH) that keeps calcium steady. When calcium falls, nerves misfire and send false alarm signals—tickles and pins-and-needles—even though nothing is touching the skin.

- Heart racing or skipping beats (palpitations): Magnesium steadies the electrical gates in the heart muscle that open and close to make each beat. Too little magnesium makes those gates unreliable, so extra beats fire off or the rhythm wobbles. Too much magnesium can slow the heart's electrical system dangerously.
- Seizure or whole-body shaking you can't control: The brain's nerve cells need magnesium to keep their electrical activity calm. When magnesium is very low, neurons fire wild storms of signals all at once—a seizure—because there's not enough brake on the system.
- Feeling weak all over or trouble moving: Very high magnesium blocks the signal that tells muscles to contract. The muscles stay loose and floppy, so even simple movements—lifting your arm, standing up—become hard or impossible.
- Nausea, throwing up, or losing your appetite: High magnesium slows the gut's squeezing waves and can irritate the stomach lining, making you queasy or causing you to vomit. Low magnesium can upset the gut's rhythm too, though nausea is more common when levels are high.
- Confusion or trouble thinking clearly: Severe magnesium imbalance—especially when it's very high—muddles the brain's electrical signals. Neurons can't communicate crisply, so thoughts turn foggy and memory slips.
- Taking medicines like proton-pump inhibitors (PPIs for heartburn), diuretics (water pills), or drinking a lot of alcohol: PPIs block acid in the stomach, and you need some acid to pull magnesium out of food. Diuretics make the kidneys flush out extra water—and magnesium goes with it. Alcohol makes the kidneys leak magnesium and also stops the gut from soaking it up properly.
- Long-lasting diarrhea or a bowel condition like Crohn disease: Most magnesium is absorbed in the small intestine. When diarrhea rushes food through, or inflammation damages the lining, the gut can't grab enough magnesium before it leaves the body.
- Kidney disease or taking magnesium-containing antacids or laxatives often: Healthy kidneys dump extra magnesium into the urine to keep levels steady. When kidneys fail, magnesium piles up in the blood. Taking a lot of magnesium by mouth (antacids, laxatives, supplements) can overwhelm even healthy kidneys and push the level dangerously high.

Focused exam

- Tremor, muscle twitching, or muscle stiffness: Low magnesium makes nerve and muscle cells fire too easily — like a light switch that flickers when you barely touch it. This happens because magnesium normally blocks calcium from rushing into nerve cells; without enough magnesium, the calcium floods in and the nerves fire over and over. This is the signature of low magnesium (hypomagnesemia) and tells you the cell membranes are unstable.

- Irregular heartbeat, especially torsades de pointes (a dangerous twisting pattern on the heart monitor): Magnesium stabilizes the heart muscle cells the same way it steadies other muscles. When it is low, the heart rhythm breaks and can twist into a life-threatening pattern called torsades de pointes. Seeing torsades points straight to low magnesium as the upstream cause.
- Low potassium (K less than 3.5 mEq/L) that will not go back up even after you give potassium replacement: Magnesium is the gatekeeper for potassium channels in the kidneys. Without enough magnesium, your body cannot hold onto potassium no matter how much you replace — it just keeps leaking out in the urine. This stubborn low potassium (called refractory low potassium (hypokalemia)) is the key clue that magnesium is the real problem.
- Low calcium (Ca less than 8.5 mg/dL) that also will not fix with calcium replacement: Low magnesium breaks the parathyroid glands (four tiny glands in the neck that control calcium). The parathyroid glands cannot release their hormone (parathyroid hormone, or PTH) properly, so calcium stays low. The calcium will not go back up until magnesium is fixed first.
- History of water pills (loop or thiazide diuretics), proton pump inhibitors (PPIs like omeprazole), or heavy alcohol use: These are the three big robbers of magnesium. Loop and thiazide diuretics make the kidneys dump magnesium into the urine. PPIs block magnesium absorption in the gut. Alcohol damages the gut lining and makes the kidneys waste magnesium. Finding the cause is the only way to stop the leak and keep magnesium from dropping again.
- Diarrhea or vomiting for several days: The gut absorbs magnesium from food in the small intestine. Diarrhea and vomiting flush magnesium out before the body can absorb it — like a bucket with holes in the bottom. This is a common physical cause of low magnesium.
- Muscle weakness, loss of reflexes, or low blood pressure (in high magnesium only): High magnesium (hypermagnesemia) paralyzes muscle cells by blocking the calcium channels they need to contract. This is the opposite of low magnesium and tells you the body is overwhelmed and shutting down, not firing too much. High magnesium usually happens when the kidneys fail and cannot clear magnesium, or when too much magnesium is given by IV or laxatives.

Diagnostics · every test answers a question

- Serum magnesium (blood test) → Is the magnesium level low, normal, or high?
- Serum potassium (blood test) → Is potassium low, and does it stay low even after replacement?
- Serum calcium (blood test) → Is calcium also low?

- 12-lead electrocardiogram (EKG) → Is the heart rhythm abnormal? Is the QT interval prolonged?
- 24-hour urine magnesium (urine collection test) → Is the body losing too much magnesium in the urine, or is the gut not absorbing it?
- Serum creatinine and estimated glomerular filtration rate (eGFR, kidney function tests) → Are the kidneys working well enough to clear magnesium, or are they failing and trapping it?
- Arterial blood gas (ABG) → Is there a metabolic alkalosis (the blood turning too alkaline) that could be linked to diuretic use or vomiting?

Treatment logic

- IV magnesium sulfate 1 to 2 grams over 1 hour for severe or symptomatic low magnesium; oral magnesium oxide 400 mg once or twice daily for mild, asymptomatic low magnesium → Restores magnesium to the blood, cells, and enzyme systems. This stops the tremor, muscle twitching, irregular heartbeat, and allows potassium and calcium to stay in the body again. IV works fast (within hours); oral works more slowly (over days).
- Find and stop the cause: review and adjust diuretics, stop or reduce PPIs, treat diarrhea, support alcohol cessation → Stopping the ongoing loss (through kidneys or gut) prevents magnesium from falling again and allows the body to rebuild its magnesium stores over time. This is the only way to keep the problem from coming back.
- Replicate potassium (usually 20 to 40 mEq oral or IV) and calcium (1 to 2 grams calcium gluconate IV or oral) after magnesium is corrected → Once magnesium is restored, the kidneys can hold onto potassium and the parathyroid glands can control calcium properly. Both will rise and stay stable. Without magnesium fixed first, potassium and calcium replacement will fail.
- Continuous cardiac monitor and 12-lead EKG to watch for torsades de pointes or prolonged QT interval → Early spotting of dangerous heart rhythms from low magnesium allows you to treat faster with IV magnesium and prevent sudden cardiac arrest. Monitoring shows you when the heart is stabilizing as magnesium rises.
- For high magnesium with symptoms (muscle weakness, lost reflexes, slow heartbeat, trouble breathing): give IV calcium gluconate 1 to 2 grams over 5 minutes, IV loop diuretic (furosemide 20 to 40 mg), and arrange urgent hemodialysis if kidney failure is present or magnesium is very high (above 4 mg/dL) → Calcium gluconate stabilizes the heart and muscle cell membranes against high magnesium — it acts like a shield that blocks magnesium from paralyzing the cells. Loop diuretics make the kidneys dump magnesium into the urine (if kidneys work). Dialysis physically removes magnesium from the blood when kidneys cannot.

- Stop magnesium-containing laxatives (like milk of magnesia), antacids (like Maalox), and IV magnesium infusions → Stops the source of extra magnesium coming into the body, especially important in kidney failure when the body cannot clear magnesium. This prevents magnesium from rising higher.

Medications

Magnesium sulfate (IV) · Electrolyte replacement (magnesium salt)

Delivers magnesium straight into the bloodstream so cells can quickly refill their magnesium stores, steadying the electrical charges on nerve and muscle membranes and restarting hundreds of enzyme jobs that depend on magnesium.

1. Magnesium sulfate dissolves in IV fluid and enters the vein.
2. Magnesium ions spread through the blood to every tissue.
3. Cells take up magnesium through ion channels in their outer membrane.
4. Inside the cell, magnesium turns on ATP-using enzymes (the powerhouses that run most chemical reactions).
5. Magnesium also plugs into NMDA receptors on nerve cells, blocking extra calcium from rushing in and calming overactive nerves.
6. In the heart, magnesium stabilizes the sodium-potassium pump on the muscle-cell membrane, keeping the heart's rhythm steady.
7. In skeletal muscle, magnesium prevents calcium from locking muscle fibers in spasm.

SAFETY WATCH

Give slowly (1–2 grams over 1 hour) — pushing too fast can drop blood pressure suddenly or slow breathing. · Watch for flushing, warmth, or nausea during the infusion (signs the dose is going in too quickly). · In people with kidney failure, magnesium can pile up dangerously because the kidneys cannot clear it — check magnesium levels and use lower doses. · Do not give with calcium in the same IV line at the same time (they can form crystals that clog the line).

Magnesium oxide or magnesium chloride (oral) · Electrolyte replacement (magnesium salt)

Delivers magnesium through the gut so the small intestine can absorb it into the blood, gradually refilling the body's magnesium stores when the deficit is mild and the gut works well.

1. Magnesium salt dissolves in the stomach and small intestine.

2. Magnesium ions cross the intestinal wall through special magnesium channels (TRPM6 and TRPM7) in the cells lining the gut.
3. The bloodstream carries magnesium to tissues.
4. Cells take up magnesium and use it to power ATP-dependent enzymes, stabilize nerve and muscle membranes, and regulate calcium and potassium movement.
5. The kidneys hold onto magnesium (reabsorb it in the thick ascending limb of the loop of Henle) to keep the level from falling again.

SAFETY WATCH

Diarrhea is common (magnesium pulls water into the colon — start with a lower dose and build up slowly). · In kidney failure, oral magnesium can still build up to dangerous levels — check blood levels regularly. · Split the daily dose (for example 200–400 mg two or three times a day) instead of one large dose to reduce diarrhea and improve absorption. · Magnesium chloride is absorbed slightly better than magnesium oxide.

Potassium chloride (oral or IV) · Electrolyte replacement (potassium salt)

Replaces potassium in the blood and cells. Low magnesium keeps the kidneys from holding onto potassium, so you must fix magnesium first or the potassium will keep leaking out and the level will not rise.

1. Potassium enters the blood (from the gut or IV line).
2. Cells take up potassium through the sodium-potassium pump (an enzyme in the cell membrane that swaps three sodium ions out for two potassium ions in, powered by ATP).
3. Magnesium is a required cofactor for this pump — without enough magnesium, the pump slows and potassium leaks back out.
4. In the kidney, magnesium normally keeps potassium channels in the collecting duct partly closed so potassium is not dumped into urine; when magnesium is low, those channels stay open and potassium pours out.

SAFETY WATCH

Too much potassium (especially IV) can stop the heart — never push IV potassium quickly; dilute it and run it through a pump. · Oral potassium can irritate or ulcerate the stomach or esophagus — take with food and plenty of water. · In kidney failure, potassium can build up dangerously — check levels frequently and reduce the dose.

Calcium gluconate (IV) · Electrolyte replacement (calcium salt)

Raises blood calcium to stop dangerous muscle spasms (including the airway and heart) caused by low calcium, which often drops when magnesium is low because the parathyroid

gland needs magnesium to release PTH (the hormone that pulls calcium from bone and tells the kidney to hold onto it).

1. Calcium gluconate dissolves in the blood.
2. Ionized calcium floods the space outside cells.
3. Calcium binds to receptors and channels on nerve and muscle membranes, raising the threshold for an action potential (the electrical spike that triggers contraction or a nerve signal).
4. In the heart, calcium steadies the plateau phase of the cardiac action potential, preventing dangerous extra beats or chaotic rhythms.

SAFETY WATCH

Give slowly (over 10 minutes) — fast infusion can cause heart rhythm problems or a sudden drop in blood pressure. · Do not mix with bicarbonate or phosphate in the same IV line (calcium will form crystals). · Extravasation (leaking out of the vein into tissue) can cause severe skin injury — make sure the IV is in the vein before you give calcium. · Calcium can worsen digitalis (digoxin) toxicity — use cautiously if the patient is on that drug.

Amiloride · Potassium-sparing diuretic (epithelial sodium-channel blocker)

Blocks sodium channels in the kidney's collecting duct so less sodium is pulled back into the blood, which indirectly keeps the kidney from dumping magnesium and potassium into the urine. Used when a genetic defect or a loop/thiazide diuretic is causing magnesium wasting.

1. Amiloride enters the urine in the kidney tubule.
2. It plugs the ENaC channel on the surface of the tubule cell, blocking sodium from flowing in.
3. Less sodium reabsorption means less negative charge is created in the tubule, so the kidney stops pulling magnesium and potassium out through paracellular channels (the spaces between cells).
4. Magnesium and potassium stay in the blood instead of being lost in urine.

SAFETY WATCH

Can raise potassium dangerously high (hyperkalemia), especially in kidney disease or if combined with ACE inhibitors, ARBs, or spironolactone — check potassium regularly. · May worsen kidney function in people who already have chronic kidney disease. · Avoid in severe kidney failure (GFR below 30) — potassium and magnesium can build up.

Loop diuretic (furosemide) and IV saline (for severe hypermagnesemia) · Loop diuretic (blocks sodium-potassium-chloride cotransporter in the kidney)

Furosemide forces the kidney to dump sodium, water, and magnesium into the urine, lowering blood magnesium quickly. IV saline expands blood volume so the kidney can filter and excrete magnesium faster. Used only when magnesium is dangerously high (usually above 4 mEq/L with symptoms).

1. Furosemide blocks the NKCC2 transporter (sodium-potassium-chloride cotransporter) in the thick ascending limb.
2. Sodium, potassium, chloride, and magnesium all stay in the urine instead of being pulled back into the blood.
3. IV saline increases blood flow to the kidney, raising the glomerular filtration rate (the speed at which blood is filtered).
4. The kidney flushes magnesium, sodium, and water into the urine, dropping the blood magnesium level within hours.

SAFETY WATCH

Can cause severe dehydration and low blood pressure if not given with enough IV saline — monitor fluid balance closely. · Flushes out potassium, sodium, and calcium along with magnesium — check and replace those electrolytes. · In kidney failure, furosemide may not work well and can worsen kidney function — dialysis is safer. · Older adults and people with heart failure can develop dangerous drops in blood pressure or worsening heart failure if too much fluid is removed too quickly.

Calcium gluconate (IV, for severe hypermagnesemia) · Electrolyte replacement (calcium salt)

Quickly reverses the dangerous effects of very high magnesium on the heart and breathing muscles by flooding receptors and channels with calcium, which competes with magnesium and restores normal muscle and nerve excitability. This is an emergency rescue, not a way to lower magnesium itself.

1. Calcium gluconate is injected into the vein and spreads through the blood instantly.
2. Calcium ions compete with magnesium at voltage-gated calcium channels on heart and nerve cells.
3. Normal calcium levels restore the action-potential shape, bringing back normal heart rhythm and muscle contraction.
4. Breathing muscles wake up so the patient can inhale and exhale normally.

SAFETY WATCH

Give slowly over 10 minutes — too-fast infusion can cause heart arrhythmias or cardiac arrest. · Watch for a slow heart rate (bradycardia) (slow heart rate) during infusion — stop if the heart rate drops suddenly. · Extravasation (leaking into tissue) causes severe skin tissue death (necrosis) — confirm IV is in the vein. · Can worsen digitalis toxicity in patients on digoxin.

Hemodialysis (for life-threatening hypermagnesemia with kidney failure) · Renal replacement therapy (mechanical filtration of blood)

A machine filters the patient's blood through a membrane that removes magnesium, potassium, and other waste while returning clean blood to the body. This is the only reliable way to lower magnesium quickly when the kidneys cannot clear it themselves.

1. Blood is pumped out of the body through a catheter (usually in the neck, chest, or groin).
2. Blood flows on one side of a semi-permeable membrane; dialysate (a clean salt solution with no magnesium) flows on the other side.
3. Magnesium diffuses from the blood (high concentration) into the dialysate (zero concentration) until the blood level falls.
4. Cleaned blood returns to the body through the catheter.
5. The process runs for 3–4 hours and removes most of the excess magnesium.

SAFETY WATCH

Requires a large-bore catheter in a central vein (risk of bleeding, infection, or pneumothorax during placement). · Can cause low blood pressure (hypotension) (low blood pressure) during the session as fluid is removed — monitor closely. · Risk of dialysis disequilibrium (brain swelling from rapid shifts in blood chemistry) if done too aggressively. · Removes other electrolytes (potassium, calcium, phosphate) — must check and replace after dialysis.

Red flags

- Heart rhythm suddenly turns very slow, very fast, or chaotic (arrhythmia) on the monitor
- Seizure or uncontrollable shaking
- Muscles so weak the person can't breathe deeply or move at all
- Confusion that gets worse quickly or the person becomes hard to wake up

Nursing priorities

1. Place patient on continuous cardiac monitor and obtain a 12-lead EKG immediately — Low magnesium prolongs the QT interval (the time the heart takes to reset between beats) and can trigger torsades de pointes, a deadly twisting heart rhythm that stops blood flow to the brain
2. Establish IV access and prepare to give IV magnesium sulfate 1 to 2 grams over 1 hour — IV magnesium replaces the body stores faster than oral forms and directly stabilizes the heart muscle membrane to prevent dangerous rhythms
3. Check potassium and calcium levels and prepare to replace both after magnesium is corrected — Low magnesium prevents potassium from entering cells (causing refractory low potassium (hypokalemia)) and blocks parathyroid hormone (a gland hormone that controls calcium), so these electrolytes will not correct until magnesium is fixed first
4. Assess deep tendon reflexes every 15 to 30 minutes during IV magnesium infusion — Loss of reflexes means magnesium level is rising too high (hypermagnesemia), which can cause muscle weakness, breathing failure, and cardiac arrest
5. Monitor respiratory rate and oxygen saturation closely during magnesium replacement — Too much magnesium depresses the brainstem (the part of the brain that controls breathing) and relaxes the diaphragm muscle, leading to slow shallow breaths or respiratory arrest
6. Review the medication list with the provider and discuss stopping or reducing diuretics and PPIs — Continuing the medicines that cause magnesium loss will make the problem come back even after replacement, creating a cycle of dangerous low magnesium
7. Assess for signs of tetany like Chvostek sign (facial twitching when you tap the cheek) or Trousseau sign (hand spasm when you inflate a blood pressure cuff) — These signs mean severe low calcium from low magnesium, which makes nerves fire too easily and can lead to seizures or laryngospasm (throat muscle spasm that blocks breathing)
8. For high magnesium with symptoms, prepare IV calcium gluconate 1 to 2 grams and stop all magnesium sources immediately — Calcium directly reverses the heart and muscle effects of high magnesium by competing at the same cell receptors, buying time while the kidneys clear excess magnesium or dialysis is arranged

Sources

- NEJM Clinical Practice review on magnesium disorders
- American Society of Nephrology (ASN) electrolyte guidelines
- UpToDate clinical reference on hypomagnesemia and hypermagnesemia
- Cochrane reviews on electrolyte replacement

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