

Osteoporosis and Fragility Fracture

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THE STORY IN ONE SENTENCE

Bones become thin and full of tiny holes inside, so they break from small bumps or falls that would never hurt a healthy person.

“Osteoporosis makes bones weak and hollow inside like a sponge losing its struts, so get a DEXA scan (a special X-ray that measures how thick your bones are) at 65 for women or 70 for men, take 1,000 to 1,200 mg of calcium and 800 to 1,000 units of vitamin D every day, do weight-bearing exercise like walking, and start a bisphosphonate pill (like alendronate) or a denosumab shot if your T-score is minus 2.5 or lower or if you already broke a bone from a small fall.”

Normal anatomy — the body's job

Bone is living tissue that rebuilds itself every day. Osteoclasts (digger cells) carve out old bone, and osteoblasts (builder cells) fill the space back in with fresh, strong bone. This balance keeps bones thick, dense, and able to hold your weight without cracking.

BODY'S JOB

The builder-and-digger dance keeps your skeleton strong enough to support your body, protect your organs like the brain and heart, store calcium for your muscles and nerves, and make new blood cells inside the marrow. When the two teams stay matched, bone mineral density stays high and bones do not break from everyday bumps.

1. Osteoclasts (the digger cells) release acid and enzymes that dissolve a small pit of old bone on the surface, like a tiny construction crew breaking up old concrete.
2. The pit sends a chemical signal that calls osteoblasts (the builder cells) to move in and lay down fresh collagen (the scaffolding, like steel beams) and then pack it with calcium and phosphate crystals (the concrete that hardens around the beams).
3. Over about three to four months, the new bone hardens and becomes as strong as the bone that was there before, so the skeleton stays solid.

4. This cycle repeats across millions of spots in your skeleton every day, replacing about ten percent of your whole skeleton each year and keeping bones young and strong.
5. Estrogen in women and testosterone in men put the brakes on the digger cells, so builders can keep up and bone mass stays thick and dense.

What breaks

The digger cells speed up or the builder cells slow down, so more bone gets carved out than gets filled back in. Over years, the bone becomes thin and full of holes inside like a sponge losing its struts, even though it looks normal on the outside and you feel fine.

CLINICAL TERM

Increased osteoclast-mediated bone resorption or decreased osteoblast-mediated bone formation tips the remodeling balance toward net bone loss, reducing bone mineral density and disrupting trabecular microarchitecture. The result is low bone mass—osteopenia (T-score -1.0 to -2.5) or osteoporosis (T-score ≤ -2.5)—which raises fracture risk even from low-energy trauma.

How the body compensates

Osteoporosis has no early warning signs because bone has no nerves to send pain signals and the body keeps pulling calcium from bone to keep blood calcium normal, so you feel completely fine. The skeleton thins silently for years, and you only find out when a bone snaps from a small fall or even a sneeze. The job is to screen with a DEXA scan and treat before the first fracture, because each break doubles the risk of the next one.

1. For years or even decades, bones thin silently while blood tests (including serum calcium and phosphate) and daily life stay completely normal—no pain, no fatigue, no swelling, no visible change on the outside.
2. The body keeps blood calcium steady by pulling it from bone through PTH, so even severe osteoporosis shows a normal serum calcium level on routine labs, which is why blood tests do not catch the disease.
3. The first fracture is often in the spine (a vertebral compression fracture, where one of the back bones collapses and causes sudden sharp back pain or slow height loss over time) or the wrist (a Colles fracture from putting your hand out to catch yourself in a fall).
4. Many spine fractures happen without any fall at all—just bending over, lifting a bag of groceries, or sneezing can crush a vertebra if the bone is thin enough, and some people never feel pain, so they only notice they are getting shorter or more hunched.

5. After one fracture, the risk of another fracture within the next year doubles, because the rest of the skeleton is just as thin and fragile, and the person may move less out of fear or pain, which thins bones even more from lack of weight-bearing stress.
6. Hip fracture (usually the femoral neck, the narrow part of the thighbone just below the ball of the hip joint) is the most dangerous: it almost always requires surgery, half of people never walk independently again, and twenty to thirty percent die within one year from complications like pneumonia, blood clots, and muscle wasting from being stuck in bed.
7. Screening with a DEXA scan (dual-energy X-ray absorptiometry, a quick, low-radiation X-ray that measures bone mineral density at the hip and spine) at age 65 for all women and age 70 for all men—or earlier if you have risk factors like steroid use, early menopause, low body weight, smoking, or a family history of hip fracture—catches low bone density before any fracture happens.
8. The DEXA scan gives a T-score, which compares your bone density to a healthy 30-year-old. A T-score of -1.0 to -2.5 is osteopenia (low bone mass), and -2.5 or lower is osteoporosis. If you already broke a bone from a small fall (a fragility fracture), you have osteoporosis no matter what the DEXA score is.
9. Treating osteoporosis with bisphosphonates (like alendronate, risedronate, ibandronate, or zoledronic acid), denosumab (a shot every six months), or other bone medicines, plus 1,000 to 1,200 mg of calcium and 800 to 1,000 units of vitamin D every day, and weight-bearing exercise like walking or lifting light weights, can rebuild bone density by five to ten percent over one to three years and cut fracture risk in half.

Domino effect

1. Estrogen drops after menopause, or steroids like prednisone are started, or aging slows the builder cells, or calcium and vitamin D intake runs too low for years. — Each of these triggers tips the bone remodeling balance so osteoclasts (digger cells) carve out more bone than osteoblasts (builder cells) can replace—nothing hurts yet because bone itself has no nerve endings to send pain signals, and blood calcium stays normal because the body just pulls more calcium out of the thinning skeleton.
2. Over months to years, tiny holes spread through the trabecular bone (the spongy inner part that looks like a honeycomb), making it thinner and weaker inside while the outer cortical shell (the hard outer layer) still looks normal on the outside. — The diggers hollow out the bone's internal struts faster than the builders can fill them back in, so bone mineral density falls and the skeleton loses its strength like a sponge losing its framework—but you feel fine and have no idea this is happening because there are no symptoms.

3. The first fracture happens from a small fall from standing height or less, a sneeze, a cough, or lifting something light—most often in the spine (vertebral compression fracture), wrist (Colles fracture, named after the surgeon who described it), or hip (femoral neck or intertrochanteric fracture). — When bone mineral density drops below a critical point (T-score ≤ -2.5 on a DEXA scan), bones cannot handle normal everyday stress anymore, so forces that would never break healthy bone—like body weight squishing down on a vertebra or the twist of catching yourself in a fall—now snap the weak, hollowed-out skeleton.
4. Spine fractures cause the vertebrae (the small bones stacked up to make your backbone) to collapse and wedge forward, so the person loses height (sometimes one to three inches over years), develops a hunched-forward back called kyphosis (a dowager's hump), and has chronic aching or sharp pain in the mid or lower back. — Each vertebra is mostly spongy trabecular bone, so when it thins, body weight crushes it like stepping on a marshmallow, and the spine curves forward because the front of the bone collapses more than the back, which also squashes the lungs and belly and makes breathing and eating harder.
5. Hip fractures almost always happen from a fall and require surgery (a metal rod and screws to hold the bone together, or a hip replacement); many people never walk independently again, lose the ability to live alone, and twenty to thirty percent die within one year from pneumonia, blood clots in the legs or lungs, or muscle wasting from being stuck in bed. — The femoral neck (the narrow part of the thighbone just below the ball of the hip joint) is under constant stress from body weight every time you stand or walk, so when it thins, even a small fall from standing snaps it, and the long, painful recovery keeps people in bed or a wheelchair, where immobility causes life-threatening complications like pressure sores, pneumonia from shallow breathing, and a clot in a deep leg vein (deep vein thrombosis).
6. After one fracture, the risk of another fracture doubles within the next year, because the rest of the skeleton is just as thin and fragile and the person may move less out of fear or pain, which thins bones even more because bones need weight-bearing stress to stay strong. — Osteoporosis is a whole-body problem, not a single weak spot, so every bone is at risk, and the cycle of fracture → fear → less activity → weaker bones → more fractures keeps spinning unless treatment stops it—this is why the first fracture is called a 'sentinel event' that demands immediate action.

7. Bisphosphonates (like alendronate or risedronate pills once a week, ibandronate once a month, or zoledronic acid IV once a year), denosumab (a shot under the skin every six months that blocks RANKL to stop osteoclasts), romosozumab (a shot that builds bone fast for one year in very high-risk patients), calcium (1,000 to 1,200 mg a day from food or pills), vitamin D (800 to 1,000 units a day), and weight-bearing exercise like walking, dancing, or lifting light weights slow or stop the bone loss, rebuild density by five to ten percent, and cut fracture risk in half. — These treatments put the brakes back on osteoclasts or give builders the raw materials and signals they need, so bone remodeling tips back toward building instead of breaking down, and the skeleton gets thicker and stronger again—fixing the one broken step upstream (the imbalance between diggers and builders) stops all the dominoes from falling and prevents the fractures that steal independence and shorten life.

Symptoms · what the patient feels, and why

- Silent bone loss with no pain or warning: Bones slowly lose their inner scaffold (the spongy mesh inside called trabecular bone) and become thin and fragile, but nerves in bone don't signal this loss until a break happens—so you can't feel it happening.
- Fracture from a minor fall or bump (fragility fracture): When bone loses its mineral density and inner structure, even a small force—like falling from standing height or bumping into furniture—can snap it. The hip, spine, and wrist are the most common spots because they bear a lot of stress.
- Loss of height over time (getting shorter): The vertebrae (the stacked bones in the spine) can collapse or compress when they become too weak to hold the body's weight, making the spine shorter and the person lose inches in height over months or years.
- Sudden sharp back pain (especially mid or lower back): A vertebra in the spine can crack or collapse (called a vertebral compression fracture) from the weight of the body or even just bending forward or lifting something light, causing sudden, knife-like pain.
- Stooped or hunched posture (kyphosis): Multiple vertebrae in the upper back collapse and wedge forward, curving the spine and creating a rounded hump (sometimes called a dowager's hump). This happens because the front of each vertebra crumbles more than the back.
- Bone pain or tenderness in the spine, hips, or ribs: Tiny cracks (microfractures) or a full fracture in weakened bone can cause constant aching or sharp pain when you touch the area or move. The bone is trying to heal, and that process triggers pain signals.
- Fracture that happens with little or no injury: Bone has lost so much strength that it can break spontaneously—sometimes just from coughing hard, lifting a light grocery bag, or stepping off a curb—because the normal force is now more than the bone can handle.

- Risk factors present: being postmenopausal, low body weight, smoking, heavy alcohol use, long-term steroid use, or family history of osteoporosis or fractures: After menopause, estrogen (the hormone that helps bones stay strong) drops sharply, speeding bone loss. Smoking and alcohol poison bone-building cells (osteoblasts) and ramp up bone-breakdown cells (osteoclasts). Steroids (like prednisone) also kill osteoblasts and block calcium absorption in the gut. Low weight means less mechanical stress to signal bones to stay strong, and family history suggests you inherited genes that make weaker bone or faster bone loss.
- Difficulty standing up from a chair or climbing stairs (muscle weakness): When bones are very weak, people often move less to avoid pain or fear of falling, so muscles shrink (atrophy). Also, vitamin D deficiency (common in osteoporosis) makes muscles weaker because vitamin D helps muscle fibers contract.
- Early satiety or trouble breathing (in severe kyphosis): When multiple spine fractures cause a severe forward curve, the ribcage presses down on the stomach and lungs, squeezing them and leaving less room for food and air.

Focused exam

- Height loss of more than 1.5 inches from young-adult height: Shows that the vertebrae (the stacked bones in the spine) are collapsing and squishing down from osteoporosis, making the person shorter over time — this is often the first quiet sign that bones are crumbling inside.
- Kyphosis — rounded upper back that makes a hump shape: Happens when the front part of several vertebrae (spine bones) crack and squish forward from weak osteoporotic bone, tilting the upper back into a curve (sometimes called a dowager's hump).
- New back pain or hip pain after a minor bump or fall from standing height: This is a fragility fracture — the bone broke from very little force because osteoporosis made it too weak to handle even normal everyday stress. This break is a red flag that bones are dangerously thin.
- Fracture of the wrist, hip, or spine after a fall from standing or less: These three spots (distal radius in the wrist, femoral neck in the hip, and vertebral bodies in the spine) are the classic fragility-fracture sites — they break first when osteoporosis weakens the skeleton.
- Low body weight (BMI under 19) or thin, small frame: People with less body mass have lower peak bone density to begin with and lose bone faster, so they reach the fracture-risk zone sooner in osteoporosis.
- Compression fractures visible on spine X-ray with no remembered injury: Shows that osteoporosis has been silently cracking vertebrae for months or years before the person felt symptoms — many spine fractures cause no pain at first but still signal high fracture risk.

Diagnostics · every test answers a question

- DEXA scan (dual-energy X-ray absorptiometry) of hip and lumbar spine → How dense and strong are the bones right now, and is the density low enough to call it osteoporosis?
- Serum calcium and phosphorus → Are calcium and phosphorus levels normal, or is another bone disease (like hyperparathyroidism or osteomalacia) mimicking osteoporosis?
- 25-hydroxyvitamin D level → Is there enough vitamin D to help bones absorb calcium and rebuild?
- Thyroid-stimulating hormone (TSH) → Is the thyroid gland overactive (hyperthyroidism), which speeds up bone breakdown?
- Parathyroid hormone (PTH) → Is the parathyroid gland pulling too much calcium out of bones to keep blood calcium normal?
- 24-hour urine calcium → Is the body losing too much calcium in urine, or absorbing too little from food?
- Serum testosterone (total) in men → Is testosterone low enough to explain bone loss in a male patient?
- Lateral spine X-ray or vertebral fracture assessment (VFA) on DEXA → Are there hidden compression fractures in the spine that the patient never felt?

Treatment logic

- Calcium 1000–1200 mg daily (from diet + supplement if needed) and vitamin D 800–1000 IU daily → Gives bones the raw calcium they need to rebuild, and vitamin D helps the gut absorb that calcium. Without enough of both, bone-building cells (osteoblasts) cannot lay down new bone to replace what is lost each day.
- Weight-bearing and resistance exercise (walking, jogging, dancing, lifting weights) most days → Physical stress on bones tells osteoblasts to build more bone and keep it thick. Regular load-bearing activity tips the balance toward building and slows age-related bone loss.
- Bisphosphonates (alendronate, risedronate, or zoledronic acid) for T-score ≤ -2.5 , prior fragility fracture, or high FRAX score → These drugs stick to bone and poison the osteoclasts (bone-breakdown cells), slowing their work. This tips the balance toward building, so bone density climbs 3–5% over 1–3 years and fracture risk drops by roughly 50%.

- Fall prevention — home safety check (remove rugs, add grab bars, improve lighting), vision correction, balance training, and review medications that cause dizziness (sedatives, blood-pressure pills) → Stops the fracture trigger. Even weak bones will not break if the person never falls. Reducing fall risk cuts fracture events as much as bone drugs do in many studies.
- Denosumab (Prolia) 60 mg subcutaneous every 6 months, if bisphosphonates fail, cause side effects, or cannot be used (severe kidney disease) → Blocks RANKL (the signal that turns on osteoclasts), so bone-breakdown cells never form. Bone density rises more than with bisphosphonates (up to 8–10% in 3 years), and spine-fracture risk drops sharply.
- Teriparatide (Forteo) or abaloparatide daily injection, or romosozumab (Evenity) monthly injection, for very high risk (T-score ≤ -3.5 , multiple fractures, or fracture on another osteoporosis drug) → These are anabolic agents — they directly wake up osteoblasts to build new bone fast. Teriparatide and abaloparatide are synthetic parathyroid hormone fragments; romosozumab blocks sclerostin (a brake on bone building). Bone density can jump 10–15% in 1–2 years.
- Treat secondary causes — taper or switch off glucocorticoids if possible; replace testosterone in men with levels < 300 ng/dL; correct hyperthyroidism, hyperparathyroidism, or malabsorption → Removes the upstream poison that was forcing bones to break down. Stopping chronic steroids or fixing hormone imbalances lets bone-building catch up again and makes other osteoporosis treatments work much better.

Medications

Calcium (1,000–1,200 mg per day) plus vitamin D (800–1,000 IU per day) · Bone minerals (supplements)

Calcium is the main building block of bone — like the bricks in a wall. Vitamin D helps your gut soak up calcium from food and tells bones to use it. Without enough of both, your body steals calcium from your own bones to keep your heart and nerves working, which leaves bones weaker. These supplements top up the supply so bone can repair itself.

1. You swallow calcium and vitamin D by mouth.
2. Vitamin D (turned active by your kidney and liver) tells the gut wall to let calcium pass into the blood.
3. Calcium travels in the blood to bones.
4. Osteoblasts (bone-building cells) grab the calcium and cement it into the bone scaffold, making bone denser and stronger.

SAFETY WATCH

Too much calcium (over 2,000 mg a day) can cause kidney stones and may raise the risk of heart problems in some people. · Take calcium in divided doses (500 mg at a time) because your gut can only absorb so much at once. · Vitamin D toxicity is rare but can happen if you take very high doses (over 4,000 IU daily for months); it causes too much calcium in the blood, which can hurt your kidneys.

Alendronate (Fosamax), risedronate (Actonel), zoledronic acid (Reclast) · Bisphosphonates (bone anti-breakdown drugs)

Bones are constantly remodeled: osteoclasts (breakdown cells) dig out old bone, and osteoblasts (builder cells) fill in new bone. In osteoporosis, the osteoclasts work too fast — like digging too many holes — so the bone gets porous. Bisphosphonates glom onto bone and poison the osteoclasts, slowing the breakdown. With less digging, the builders catch up and bone density rises.

1. You swallow the pill (alendronate, risedronate) or get an IV infusion once a year (zoledronic acid).
2. The drug travels in your blood to bones.
3. Bisphosphonate molecules bind tightly to bone mineral where osteoclasts are working.
4. When an osteoclast tries to break down bone, it swallows the bisphosphonate.
5. Inside the osteoclast, the drug blocks a key enzyme (farnesyl pyrophosphate synthase, which the cell needs to stay alive).
6. The osteoclast dies, so bone breakdown slows.
7. Osteoblasts keep building, and over months your bone density goes up.

SAFETY WATCH

Stomach irritation or heartburn (pills can burn the esophagus); take on an empty stomach with a full glass of water, then stay upright for 30 minutes. · Very rare but serious: osteonecrosis of the jaw (bone death in the jaw, usually after dental surgery or tooth extraction); tell your dentist you take a bisphosphonate. · Very rare: atypical femur fracture (an odd break in the thigh bone after years of use); stop the drug if you get new thigh or groin pain. · After 5 years of oral bisphosphonate (or 3 years of IV), most people take a 1–2 year 'drug holiday' to let bone remodel naturally, unless fracture risk is very high.

Denosumab (Prolia) · RANK-ligand inhibitor (monoclonal antibody that blocks bone breakdown)

Osteoclasts (bone-eating cells) need a chemical signal called RANK-ligand to wake up and start digging. Denosumab is an antibody (a protein made in a lab) that grabs RANK-ligand and covers it up, like putting tape over a doorbell. Without the signal, osteoclasts never turn on, so bone breakdown almost stops. Builders keep working, and bone density climbs.

1. You get a shot under the skin (subcutaneous injection) every 6 months.
2. Denosumab circulates in your blood.
3. It binds to RANK-ligand (the chemical messenger that tells baby osteoclasts to grow up and start eating bone).
4. With RANK-ligand blocked, osteoclast precursors never mature.
5. Bone breakdown drops fast (within days).
6. Osteoblasts keep building, so bone density rises over months.

SAFETY WATCH

CRITICAL: Do NOT stop suddenly. Bone breakdown surges when the drug wears off, and you can suffer multiple spine fractures (rebound fractures). If you must stop, your doctor will usually switch you to a bisphosphonate to hold bone steady. · Low calcium in the blood (hypocalcemia), especially in the first weeks; make sure your calcium and vitamin D levels are good before each shot. · Same rare jaw and atypical femur fracture risks as bisphosphonates. · Slightly higher risk of skin infections; call your doctor if you get redness, swelling, or pain in your skin after the shot.

Teriparatide (Forteo), abaloparatide (Tymlos) · Parathyroid hormone (PTH) analogs (bone-building agents)

Parathyroid hormone (PTH) is made by tiny glands in your neck; it normally keeps blood calcium steady. In a twist, a small daily dose of PTH (or a drug that mimics it) wakes up osteoblasts (bone builders) more than osteoclasts (bone eaters), so you get a net GAIN in bone — especially in the spine. It is like sending in extra construction workers before the wrecking crew shows up. This is the only class that actually builds NEW bone rather than just slowing breakdown.

1. You inject the drug under your skin (subcutaneous) once a day.
2. The synthetic PTH fragment travels in your blood to bones.
3. It binds to PTH receptors on osteoblasts and their precursor cells.
4. Osteoblasts wake up, multiply, and start building new bone matrix (collagen and minerals).

5. Over weeks to months, bone density — especially in the spine — rises more than with any other drug.
6. After 18–24 months (the maximum safe duration), you switch to a bisphosphonate or denosumab to keep the new bone from breaking down.

SAFETY WATCH

Can cause leg cramps, dizziness, or nausea in the first few weeks. · Raises blood calcium slightly; do not use if you already have high calcium or a history of kidney stones. · In rat studies, very high doses caused bone cancer (osteosarcoma) — this has NOT been seen in humans, but the drugs carry a black-box warning and are limited to 2 years lifetime use. Do not use if you have had radiation to bones, Paget disease of bone, or unexplained high alkaline phosphatase. · Daily injections can be a burden; some people get injection-site reactions (redness, bruising).

Romosozumab (Evenity) · Sclerostin inhibitor (dual-action: builds bone AND blocks breakdown)

Sclerostin is a protein made by osteocytes (cells embedded in bone) that puts the brakes on bone building. Romosozumab is an antibody that blocks sclerostin, so osteoblasts (builders) work overtime and osteoclasts (breakers) slow down — you get a double boost to bone density, faster than any other drug. It is like releasing the parking brake and stepping on the gas at the same time.

1. You get two shots under the skin (subcutaneous) once a month for 12 months (the maximum duration).
2. Romosozumab binds to sclerostin and neutralizes it.
3. With sclerostin blocked, osteoblasts ramp up bone building.
4. At the same time, osteoclast activity drops (probably because new bone signals them to slow down).
5. Bone density jumps fast — spine density can rise 10–15% in one year, more than with any other drug.
6. After 12 months, you switch to a bisphosphonate or denosumab to keep the new bone.

SAFETY WATCH

BLACK-BOX WARNING: may raise the risk of heart attack, stroke, or cardiovascular death. Do NOT use if you have had a heart attack or stroke in the past year. Your doctor will weigh fracture risk against heart risk. · Can cause joint or muscle pain. · Same rare risks of jaw osteonecrosis and atypical femur fracture as bisphosphonates and denosumab. · Injection-site reactions (pain, redness). · Reserved for very high-risk patients (multiple spine fractures, T-score below -3.5, or fracture on another osteoporosis drug) because it is expensive and carries cardiovascular concerns.

Raloxifene (Evista) · Selective estrogen receptor modulator (SERM)

After menopause, estrogen drops and bone breakdown speeds up because osteoclasts (bone eaters) lose their brake signal. Raloxifene acts like weak estrogen in bone — it binds to estrogen receptors on bone cells and slows osteoclast activity, so breakdown drops and bone density rises modestly. In the breast and uterus, raloxifene blocks estrogen (which is why it lowers breast-cancer risk and does NOT raise uterine-cancer risk like real estrogen can).

1. You swallow one pill daily.
2. Raloxifene travels in the blood to bones.
3. It binds to estrogen receptors on bone cells, mimicking estrogen's bone-protecting effect.
4. Osteoclast activity slows, so bone breakdown drops.
5. Bone density rises modestly (less than bisphosphonates), mainly in the spine.
6. In the breast, raloxifene BLOCKS estrogen receptors, which lowers the risk of estrogen-receptor-positive breast cancer.

SAFETY WATCH

Raises the risk of blood clots (deep vein clot formation (thrombosis), pulmonary embolism (a clot lodging in a lung artery)) about as much as estrogen does. Do NOT use if you have a history of clots or are immobile (e.g., after surgery, long flight). · Can worsen hot flashes and cause leg cramps. · May slightly raise the risk of fatal stroke in women with heart disease or stroke risk factors; use with caution in that group. · Does NOT increase uterine-cancer risk (unlike estrogen alone).

Calcitonin (Fortical, Miacalcin) — now RARELY used · Calcitonin (a hormone that weakly slows bone breakdown)

Calcitonin is a hormone made by the thyroid gland that tells osteoclasts (bone-eating cells) to slow down. A synthetic version (salmon calcitonin) is stronger than human calcitonin and was once used to treat osteoporosis. It lowers bone breakdown modestly and may ease bone pain after a fresh spine fracture. However, newer drugs work much better, so calcitonin is now

rarely prescribed for osteoporosis — it is mostly used short-term for pain relief after an acute vertebral fracture.

1. You spray it in your nose once a day (nasal spray) or get an injection under the skin.
2. Calcitonin travels in the blood to bones.
3. It binds to receptors on osteoclasts and temporarily shuts them down.
4. Bone breakdown slows for a few hours.
5. The effect wears off quickly, and the drug does NOT build lasting bone density.

SAFETY WATCH

Nasal spray can cause runny nose, nosebleeds, or nasal irritation. · Injection can cause nausea, flushing, or skin rash. · A safety review found a possible small increase in cancer risk with long-term use, which is another reason it is rarely used now. · Much less effective than other osteoporosis drugs, so it should NOT be your first choice.

Red flags

- Fracture from a fall from standing height or less (suggests severe bone fragility and very high risk for more fractures)
- Sudden severe back pain after bending, lifting, or coughing (may be a vertebral compression fracture—needs imaging right away)
- Loss of more than 1.5 inches in height or new stooped posture (suggests multiple spine fractures have already happened)
- Hip fracture (very high risk for disability and death, especially in older adults—about 20% of hip fracture patients die within one year)
- Multiple fractures or fractures in unusual places like the ribs or pelvis (may signal severe osteoporosis or a secondary cause like cancer, hyperparathyroidism, or multiple myeloma)

Nursing priorities

1. Assess for acute vertebral fracture by asking about new or sudden back pain, checking height loss greater than 1.5 inches from younger adult height, and observing for kyphosis (forward curve of upper back) — Vertebral compression fractures cause severe pain and disability but are often missed because they can happen without trauma; early detection allows pain control and prevents additional spine fractures that occur in 20 percent of patients within one year

2. Complete fall-risk assessment including gait observation, home safety review (loose rugs, poor lighting, bathroom grab bars), vision screening, and medication review for drugs causing dizziness like sedatives and blood pressure pills — Falls cause 90 percent of hip fractures; identifying and fixing fall hazards prevents fractures even in patients with low bone density, and some fracture risk comes from falling rather than bone weakness alone
3. Teach proper bisphosphonate administration if prescribed: take first thing in morning on empty stomach with full glass of plain water, stay upright (sitting or standing) for 30 minutes, no food or drink except water during that time — Bisphosphonates are poorly absorbed (only 1 percent gets into body), and lying down or eating blocks absorption completely; staying upright prevents esophagitis (irritation of the swallowing tube) and ulcers that cause severe chest pain
4. Calculate total daily calcium intake from diet and supplements to reach 1000 to 1200 mg, and ensure vitamin D supplementation of 800 to 1000 IU daily or check blood 25-hydroxyvitamin D level — Calcium is the building block of bone and vitamin D (a hormone that controls calcium absorption) is required for calcium to enter bones; without adequate amounts, bone medicine cannot work and bones continue weakening
5. Educate on weight-bearing and resistance exercises for 30 minutes most days, such as walking, dancing, climbing stairs, or lifting light weights, while avoiding high-impact activities that increase fracture risk — Mechanical stress from weight-bearing exercise signals bone cells called osteoblasts to build new bone and strengthens muscles that prevent falls; swimming and cycling do not stress bones enough to trigger bone formation
6. Review medications that increase fracture risk including proton pump inhibitors (omeprazole, pantoprazole) used longer than one year, SSRIs (antidepressants), and sedatives, and discuss alternatives with provider if appropriate — Proton pump inhibitors reduce calcium absorption; SSRIs and sedatives double fall risk through dizziness and slower reflexes; stopping or switching these medicines when safe reduces fractures separate from bone treatment
7. Schedule bone density DEXA scan (dual-energy X-ray absorptiometry) if not done recently and ensure patient understands T-score results: -1.0 to -2.5 is osteopenia (mild bone loss), -2.5 or lower is osteoporosis requiring medicine — DEXA scan T-score (comparison to young healthy bones) determines fracture risk and treatment decisions; T-score drops 1 point for every 10 to 12 percent bone loss, and fracture risk doubles with each point decrease
8. Counsel on limiting alcohol to one drink daily for women or two for men, and provide smoking cessation resources, as both directly poison osteoblasts (bone-building cells) and increase fracture risk 30 to 40 percent — Alcohol and tobacco are direct bone toxins that kill bone-forming cells and reduce blood flow to bones; quitting smoking and limiting alcohol allows bone-building cells to recover and improves response to bone medicines

Sources

- AACE/Endocrine Society Clinical Practice Guidelines 2020
- USPSTF Osteoporosis Screening Recommendation 2018
- National Osteoporosis Foundation (NOF) Clinician's Guide 2022
- WHO Fracture Risk Assessment Tool (FRAX)

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