

Osteoporosis and Fragility Fracture

A plain-English guide for you and your family

WHAT IS HAPPENING

Your bones are alive and busy rebuilding themselves every single day. Special cells called osteoclasts dig out old bone, and other cells called osteoblasts fill the holes back in with fresh, strong bone. In a young adult, these two teams stay balanced, so bones stay thick and solid. But after menopause, estrogen (a hormone that keeps the digger cells from working too fast) drops, so the diggers speed up and carve out more bone than the builders can replace. The same thing happens with aging, long-term steroid medicines like prednisone, smoking, heavy alcohol, low testosterone in men, and not getting enough calcium or vitamin D. Year after year, tiny holes spread through the bone like a sponge getting airier and airier, but you feel nothing because bone itself has no nerve endings to tell you it hurts. Then one day a small trip, a sneeze, or lifting a grocery bag snaps a bone that should have held strong—often the hip, spine, or wrist. That fracture is the first signal that the balance tipped long ago. A DEXA scan catches the thinning early, before a break happens, and medicines like bisphosphonates or denosumab slow the digger cells down so the builders can catch up and make bones thicker again. Calcium, vitamin D, and walking or lifting light weights give the builders the raw materials and the signal they need to keep your skeleton strong.

What you might feel

- Silent bone loss with no pain or warning
- Fracture from a minor fall or bump (fragility fracture)
- Loss of height over time (getting shorter)
- Sudden sharp back pain (especially mid or lower back)
- Stooped or hunched posture (kyphosis)
- Bone pain or tenderness in the spine, hips, or ribs
- Fracture that happens with little or no injury
- Risk factors present: being postmenopausal, low body weight, smoking, heavy alcohol use, long-term steroid use, or family history of osteoporosis or fractures
- Difficulty standing up from a chair or climbing stairs (muscle weakness)

- Early satiety or trouble breathing (in severe kyphosis)

Call the doctor right away if...

- 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)

What the care team will do

- 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)
- 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
- 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
- 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
- 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
- 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
- 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

- 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

Medicines you may be given — and why

Calcium (1,000-1,200 mg per day) plus vitamin D (800-1,000 IU per day)

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.

Alendronate (Fosamax), risedronate (Actonel), zoledronic acid (Reclast)

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.

Denosumab (Prolia)

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.

Teriparatide (Forteo), abaloparatide (Tymlos)

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.

Romosozumab (Evenity)

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.

Raloxifene (Evista)

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).

Calcitonin (Fortical, Miacalcin) — now RARELY used

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.

Where this information comes from

- 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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