

Bone infection

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THE STORY IN ONE SENTENCE

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“Bone infection — get imaging (X-ray then MRI), take a culture from the bone itself, give IV antibiotics for weeks, and cut out dead bone if medicine alone won't clear it.”

Normal anatomy — the body's job

Healthy bone has a hard outer shell (the cortex) and a spongy inside (the medulla) packed with blood vessels that deliver oxygen, immune cells, and nutrients. That constant flow keeps bacteria from taking hold.

BODY'S JOB

The bone's job is to support your body, protect organs, make blood cells in the marrow, and store minerals like calcium. The rich blood supply inside also patrols for invaders—white blood cells cruise through looking for bacteria and clean them up before they can multiply.

1. Tiny arteries branch into the bone through small holes in the cortex.
2. Blood flows through the spongy marrow and out through veins.
3. The periosteum (a thin, tough skin covering the outside of the bone) holds more blood vessels and nerves.

What breaks

Bacteria reach the bone and multiply faster than the immune system can kill them. Blood vessels get squeezed or clotted off by swelling, which starves the bone and traps infection inside.

CLINICAL TERM

Osteomyelitis is infection of bone caused by bacteria (most often *Staphylococcus aureus*, including MRSA) reaching the bone through direct inoculation (trauma, surgery, contiguous spread from an adjacent soft-tissue infection or ulcer) or hematogenous seeding (bloodstream spread). Inflammation and pus increase intraosseous pressure, compress blood vessels, cause starved blood flow (ischemia) and tissue death (necrosis), and form sequestra (islands of dead bone) that antibiotics cannot penetrate.

How the body compensates

The body sends extra white blood cells and walls off infection with scar tissue, which buys time but also raises pressure inside the bone, chokes off blood flow, and creates pockets of dead bone that antibiotics can't reach.

1. {'step': 'Inflammation and pus form at the infection site.', 'why': 'White blood cells flood in to kill bacteria, releasing chemicals that cause swelling, heat, and pain—the classic signs you see on exam.'}
2. {'step': 'Pressure inside the bone rises.', 'why': 'The hard cortex can't stretch, so swelling squeezes the blood vessels, cutting off oxygen and nutrients to the bone.'}
3. {'step': 'Dead bone (sequestrum) forms.', 'why': 'Starved of blood, chunks of bone die and become a safe hiding place for bacteria, because antibiotics travel in blood and can't reach dead tissue.'}
4. {'step': 'New bone (involucrum) grows around the infection.', 'why': 'The periosteum lays down fresh bone to wall off the infection, but this shell can trap pus inside, turning the site into a chronic abscess.'}
5. {'step': 'Sinus tracts open to the skin.', 'why': 'Pus finds the path of least resistance and drains through the soft tissue to the outside, creating a tunnel that won't close until the bone infection is cleared.'}
6. {'step': 'Systemic inflammatory response may appear.', 'why': 'If bacteria or their toxins spill into the bloodstream, you see fever, chills, elevated white count, and elevated inflammatory markers (CRP, ESR)—but these can be absent in chronic low-grade infection.'}

Domino effect

1. Bacteria enter bone and begin to multiply. — Whether they arrive through a wound, surgical hardware, or the bloodstream, bacteria find a foothold faster than the immune system can clear them—especially in areas with poor blood flow.

2. Inflammation and pus build up inside the bone. — White blood cells rush in and release enzymes to kill the invaders, but the pus and swelling have nowhere to go inside the rigid bone, so pressure climbs.
3. Rising pressure squeezes blood vessels shut. — The bone's own shell (cortex) becomes a cage: swelling inside compresses the tiny arteries and veins, cutting off oxygen and trapping dead tissue and bacteria.
4. Islands of dead bone (sequestra) form. — Without blood flow, chunks of bone die. Antibiotics can't reach dead tissue, so bacteria hide inside these islands and keep the infection alive even after weeks of treatment.
5. Chronic infection and sinus tracts develop. — Pus burrows through soft tissue to drain at the skin, creating a tunnel (sinus tract) that won't heal as long as infected dead bone remains inside—leading to months or years of drainage, pain, and flare-ups.
6. Without treatment, bone destruction spreads or sepsis occurs. — Ongoing infection eats away more bone, weakens it to the point of fracture, or seeds the bloodstream (sepsis), which can damage distant organs and become life-threatening.
7. Surgery removes dead bone; antibiotics clear remaining bacteria. — Cutting out the sequestrum and any infected hardware lets blood flow return and antibiotics reach live tissue, which is the only way to truly cure deep bone infection and close those draining wounds.

Symptoms · what the patient feels, and why

- Pain right over the infected bone that gets worse when you press or move it: Bacteria multiply inside the bone, triggering swelling and inflammation in a tight space that cannot stretch — like blowing up a balloon inside a brick — which presses on nerves and causes sharp, deep pain.
- Warmth, redness, and swelling of the skin over the bone: The body rushes more blood to the infected area to deliver white blood cells and oxygen — extra blood makes the skin warmer and redder, and leaky blood vessels let fluid escape into the tissue, causing swelling.
- Fever and chills: The immune system detects bacteria in the bone and releases chemical messengers called cytokines that reset the brain's thermostat in the hypothalamus (the body's temperature control center) to a higher setting, causing fever to help white blood cells fight better.
- An open sore or draining wound near the bone that won't heal, especially in people with diabetes or poor circulation: Chronic wounds break the skin barrier and let bacteria travel down to the bone; once bacteria are deep in bone tissue, the infection keeps the wound from healing because bacteria constantly re-seed the surface and inflammation damages tissue faster than it can repair.

- New or worsening limping, inability to bear weight, or refusal to use an arm or leg (especially in children): Moving or putting weight on the infected bone stretches inflamed tissue and bone lining, sending sharp pain signals — children and adults naturally protect the painful limb by avoiding movement.
- Bone tenderness or a positive probe-to-bone test in someone with a diabetic foot ulcer: In people with diabetes and chronic foot ulcers, skin and soft tissue often break down deep enough that bacteria reach the bone underneath; a sterile metal probe inserted into the ulcer will hit hard bone instead of soft tissue if infection has eaten into the bone.
- Fatigue, irritability, and poor appetite (especially in babies and young children): The body diverts energy to fight infection — cytokines (immune signaling molecules) suppress appetite and make children feel tired and cranky, and fever burns extra calories that leave less energy for normal activity.
- Visible pus draining from a surgical site, fracture, or puncture wound near bone: White blood cells attack bacteria in the bone and die, forming thick yellow or green pus; pressure from swelling forces the pus to drain out through any break in the skin, such as a wound or surgical incision.

Focused exam

- Probe-to-bone test over a diabetic foot ulcer: Bone infection (osteomyelitis) often starts when bacteria from a long-lasting wound tunnel down and reach the bone underneath. If you push a sterile probe into the ulcer and it touches hard bone, osteomyelitis is very likely.
- Local swelling, warmth, and drainage at the site: The body sends white blood cells (infection-fighting cells) and extra fluid to the infected spot. This puffiness, heat, and pus or cloudy fluid draining out are your clues that osteomyelitis is spreading in the tissues around the bone.
- Fever, chills, or feeling very sick (septic): When bone infection spills bacteria into the bloodstream, your whole body sounds the alarm with fever and shaking chills. This tells you the osteomyelitis is serious and spreading beyond the local spot.
- Sharp, throbbing pain over the bone that does not stop with rest: Bone pain from osteomyelitis is deeper and worse than regular muscle or skin pain because infection irritates the bone's own nerves and blood vessels. The pain throbs with your heartbeat and does not ease when you lie down.
- Back pain with fever in a patient with recent surgery or IV drug use: Bacteria traveling in the blood can land in the bones of the spine (vertebral osteomyelitis). The combo of new back pain plus fever is a red flag that infection has reached the spine, especially if the person had recent surgery or used needles that carried germs into the blood.

- Redness, warmth, and drainage near surgical hardware, screws, or pins: Metal implants in bone can trap bacteria and form a slimy coating (biofilm) that shields the germs from antibiotics. When osteomyelitis grows around hardware, you see pus, redness, and heat right at that spot because your body cannot clear the infection on its own.

Diagnostics · every test answers a question

- Red blood cell (Erythrocyte) sedimentation rate (ESR) and C-reactive protein (CRP) → Is there ongoing inflammation in the body that suggests bone infection?
- Blood cultures (two sets from different sites before antibiotics) → Is there bacteria in the bloodstream, and can we grow it to find out exactly which germ is causing the infection?
- Bone biopsy with culture and histology → What germ is living in the bone itself, and is the bone truly infected or just inflamed?
- MRI of the affected bone with and without contrast → How far has the infection spread in the bone and surrounding soft tissue, and is there an abscess (pocket of pus) that needs to be drained?
- Plain X-ray of the affected bone (two views) → Is there visible bone destruction or new bone formation that suggests chronic osteomyelitis?
- Complete blood count (CBC) with differential → Are white blood cells elevated, showing the body is fighting infection?
- Hemoglobin A1c (HbA1c) in patients with diabetes → Is blood sugar control poor, making it harder for the body to fight infection and heal bone?

Treatment logic

- Blood cultures (two sets) and bone biopsy before starting antibiotics (unless patient is septic and unstable) → Grows the exact germ causing osteomyelitis so you know which antibiotic kills it, instead of guessing. Stops you from treating the wrong bacteria and lets you narrow therapy once results come back (usually 2-3 days).
- Empiric IV antibiotics started immediately if patient is septic: vancomycin 15-20 mg/kg every 8-12 hours PLUS ceftriaxone 2 g daily or ciprofloxacin 400 mg every 12 hours → Kills *Staphylococcus aureus* (including MRSA) with vancomycin and covers gram-negative bacteria (like *E. coli* and *Pseudomonas*) that can infect bone, especially in diabetic foot or spine infections. Vancomycin penetrates bone tissue where many other drugs cannot reach.
- Surgical debridement: remove all dead bone (sequestrum), pus, and infected hardware if present → Takes away infected tissue and dead bone that antibiotics cannot reach because they have no blood supply. Lets fresh blood flow back into the cleaned bone so white blood cells and antibiotics can finish the job. Removes biofilm on hardware that shields bacteria.

- Pathogen-directed IV antibiotics for 4–6 weeks total (2–4 weeks IV, then switch to oral with high bioavailability like fluoroquinolones, linezolid, or trimethoprim-sulfamethoxazole based on culture results) → Long therapy gives bone time to heal and new bone to grow in where infection was removed. Switching to oral antibiotics after the first few weeks lets you finish treatment at home instead of staying in the hospital for six weeks.
- Optimize blood sugar control ($HbA1c < 7\%$) and stop smoking; evaluate blood vessels in diabetic foot with ankle-brachial index or vascular ultrasound → Better blood flow and lower blood sugar help white blood cells reach the infection and oxygen reach the bone so it can heal. Stopping smoking opens up small blood vessels. Fixing blocked arteries (revascularization) restores blood supply to blood-starved (ischemic) bone.
- Monitor ESR and CRP weekly; repeat MRI if clinical improvement stalls after 2–3 weeks of treatment → Falling ESR and CRP show the infection is shrinking. If numbers stay high or climb, the infection is not controlled — you may need different antibiotics, longer treatment, or more surgery. Repeat MRI finds hidden abscesses or new bone destruction.

Medications

Vancomycin · Glycopeptide antibiotic

Vancomycin kills bacteria by punching holes in the wall they build around themselves. Without a strong wall, the bacteria burst open and die. It works especially well against *Staphylococcus aureus*, including the tough MRSA kind (methicillin-resistant *Staph aureus*, a strain that ignores many common antibiotics). Bone infections often start with *Staph* bacteria, so vancomycin is a go-to choice when MRSA is suspected or proven.

1. Vancomycin travels through the bloodstream and reaches the infected bone.
2. It latches onto building blocks (called peptidoglycan units) that bacteria use to construct their cell wall.
3. By blocking these building blocks, vancomycin stops the bacteria from finishing or repairing their wall.
4. The bacteria's wall becomes weak and full of gaps.
5. Water rushes into the bacteria through the gaps, causing them to swell and burst apart.
6. The dead bacteria are then cleared away by the body's immune cells.

SAFETY WATCH

Kidney damage: Vancomycin can hurt the kidneys, especially at high doses or when used with other drugs that stress the kidneys. Doctors check blood tests (creatinine level) regularly to watch kidney function. · Hearing loss: High levels of vancomycin in the blood can damage the inner ear, leading to ringing in the ears or permanent hearing loss. Blood levels are monitored to keep the dose safe. · Red Man Syndrome: If vancomycin is given too fast through the IV, it can cause a sudden flushing (red face and chest), itching, and a drop in blood pressure. Slowing the infusion prevents this. · Allergic reactions: Rarely, vancomycin can trigger hives, swelling, or trouble breathing. Tell the care team immediately if this happens.

Ceftriaxone · Third-generation cephalosporin antibiotic

Ceftriaxone kills bacteria by blocking the enzymes (proteins that do chemical jobs) they need to build their cell wall. Without a strong wall, the bacteria cannot hold their shape and they fall apart. It covers a wide range of bacteria, including many gram-negative bacteria (a type identified by how they stain in the lab) that can cause bone infections, especially when the infection spreads from a nearby ulcer, surgical site, or through the bloodstream.

1. Ceftriaxone enters the bloodstream through an IV or muscle injection and travels to the infected bone.
2. It slips through the bacteria's outer membrane and reaches the cell wall building zone.
3. Ceftriaxone binds tightly to penicillin-binding proteins (the bacteria's wall-building machines).
4. With these proteins stuck, the bacteria cannot connect the wall's building blocks together.
5. The cell wall becomes weak and incomplete.
6. The bacteria burst open under their own internal pressure.
7. The immune system cleans up the debris and the infection starts to clear.

SAFETY WATCH

Allergic reactions: People allergic to penicillin may also react to ceftriaxone (about 1 in 10 chance of cross-allergy). Watch for rash, hives, swelling, or trouble breathing. · Diarrhea and C. difficile infection: Antibiotics can kill good gut bacteria, allowing a harmful bacterium called *Clostridioides difficile* to overgrow and cause severe watery diarrhea, belly cramps, and fever. Tell the care team if diarrhea develops. · Gallbladder sludge: Ceftriaxone can form crystals in the gallbladder, especially in children and people getting high doses. This usually goes away after stopping the drug but can cause belly pain. · Blood cell changes: Rarely, ceftriaxone can lower white blood cells, red blood cells, or platelets. Blood tests are checked if treatment lasts a long time.

Piperacillin-tazobactam · Extended-spectrum penicillin combined with a beta-lactamase inhibitor

Piperacillin kills bacteria by blocking the enzymes they use to build their cell wall, causing them to burst. Tazobactam protects piperacillin from being destroyed by enzymes (called beta-lactamases) that many bacteria make to defend themselves. Together, they cover a very broad range of bacteria, including many gram-negative bacteria and some that live without oxygen (anaerobes), making this combination useful when the infection involves multiple types of bacteria or the exact cause is not yet known.

1. Piperacillin-tazobactam is given through an IV and travels through the bloodstream to the infected bone.
2. Tazobactam latches onto beta-lactamase enzymes and shuts them down, so they cannot destroy piperacillin.
3. Piperacillin then binds to the bacteria's penicillin-binding proteins, which are the machines that cross-link the cell wall.
4. With these proteins blocked, the bacteria cannot finish building or repairing their wall.
5. The bacteria's internal pressure causes them to swell and burst.
6. The immune system clears away the dead bacteria and healing begins.

SAFETY WATCH

Allergic reactions: Anyone allergic to penicillin antibiotics should avoid this drug. Watch for rash, hives, swelling, or trouble breathing. · Diarrhea and C. difficile infection: Like all broad antibiotics, this can upset the balance of gut bacteria and allow harmful Clostridioides difficile to overgrow, causing severe diarrhea. · Kidney damage: High doses or prolonged use can stress the kidneys, especially in people with existing kidney problems. Blood tests monitor kidney function. · Low potassium: Piperacillin can cause potassium levels in the blood to drop, leading to muscle weakness or irregular heartbeat. Electrolytes are checked regularly. · Bleeding risk: Rarely, piperacillin can interfere with platelet function (the cells that help blood clot), increasing the risk of bruising or bleeding.

Levofloxacin or Ciprofloxacin · Fluoroquinolone antibiotic

Levofloxacin and ciprofloxacin kill bacteria by blocking enzymes (called DNA gyrase and topoisomerase IV) that bacteria need to unwind, copy, and repair their DNA. Without working DNA, the bacteria cannot multiply or survive. These drugs are especially useful for bone infections because they penetrate bone tissue well and cover many gram-negative bacteria and some gram-positive ones. They can be switched from IV to pills, which is helpful for the long treatment courses bone infections require.

1. The fluoroquinolone is given by IV or taken as a pill and absorbed into the bloodstream.

2. It travels to the infected bone and enters the bacteria.
3. Inside the bacteria, it binds to DNA gyrase and topoisomerase IV, locking them in place.
4. These enzymes can no longer unwind or relax the tightly coiled DNA strands.
5. The bacteria's DNA breaks apart and cannot be copied or repaired.
6. Without intact DNA, the bacteria die, and the immune system clears them away.

SAFETY WATCH

Tendon rupture: Fluoroquinolones can weaken tendons, especially the Achilles tendon at the back of the heel. This risk is higher in people over 60, those taking steroids, or people who have had a kidney transplant. Stop the drug and call the doctor if sudden pain, swelling, or trouble walking occurs. · Nerve damage (peripheral neuropathy): Rarely, these drugs can cause permanent nerve damage, leading to tingling, numbness, burning, or shooting pain in the hands or feet. Stop the drug and tell the care team right away if this happens. · Worsening of myasthenia gravis: Fluoroquinolones can make this muscle-weakening disease much worse. People with myasthenia gravis should avoid these drugs. · Mood and mental changes: Some people experience anxiety, confusion, depression, trouble sleeping, or even hallucinations. Older adults are at higher risk. · Blood sugar swings: These drugs can cause blood sugar to drop too low (hypoglycemia) or spike too high, especially in people with diabetes. · Heart rhythm problems: Rarely, fluoroquinolones can prolong the QT interval on an EKG, increasing the risk of a dangerous irregular heartbeat. Caution is needed in people already at risk for heart rhythm problems.

Linezolid · Oxazolidinone antibiotic

Linezolid stops bacteria from making the proteins they need to survive and multiply. It works by blocking the very first step of protein assembly inside the bacteria. It is particularly useful against MRSA and other tough gram-positive bacteria, and it can be given by mouth (not just IV), which is helpful for long bone-infection treatments after the first few weeks of IV therapy.

1. Linezolid is given by IV or taken as a pill and enters the bloodstream.
2. It travels to the infected bone and slips into the bacteria.
3. Linezolid binds to the 50S subunit of the bacterial ribosome, the machine that strings amino acids together to make proteins.
4. It blocks the ribosome from starting protein production.
5. Without new proteins, the bacteria cannot build enzymes, repair themselves, or multiply.
6. The bacteria weaken and die, and the infection begins to clear.

SAFETY WATCH

Low blood cell counts: Linezolid can lower red blood cells (causing anemia and tiredness), white blood cells (raising infection risk), and platelets (increasing bleeding risk), especially if used for more than 2 weeks. Blood counts are checked weekly during treatment. · Nerve damage (peripheral and optic neuropathy): Long-term use (more than 28 days) can damage nerves in the arms, legs, or eyes, causing tingling, numbness, vision loss, or color blindness. Vision and sensation should be monitored. · Serotonin syndrome: Linezolid can raise serotonin levels in the brain. If taken with certain antidepressants (SSRIs, SNRIs, MAOIs) or other serotonin-raising drugs, it can cause serotonin syndrome—confusion, agitation, fast heartbeat, high blood pressure, sweating, shivering, muscle twitching, and fever. This can be life-threatening. Tell the care team about all medications. · Lactic acidosis (acid from oxygen-starved tissues): Rarely, linezolid can cause a dangerous buildup of lactic acid in the blood, leading to nausea, vomiting, fast breathing, and confusion. Seek help immediately if these occur. · High blood pressure: Linezolid can raise blood pressure, especially in people eating foods high in tyramine (aged cheeses, cured meats, soy sauce, draft beer). Blood pressure should be monitored.

Red flags

- Fever above 101.5°F (38.6°C) with bone pain, especially in a child or after surgery or injury
- Severe pain that does not improve with rest or over-the-counter pain medicine
- Red streaks spreading from the area, confusion, very fast heartbeat, or low blood pressure (signs bacteria are spreading into the bloodstream)
- A non-healing wound or ulcer over a bony area, especially in someone with diabetes, peripheral artery disease, or a weakened immune system
- New inability to move a limb, numbness, tingling, or loss of pulse below the infection site (may mean the infection is pressing on nerves or blood vessels)

Nursing priorities

1. Obtain two sets of blood cultures from different sites before giving any antibiotics, unless patient shows sepsis (low blood pressure, confusion, or organ failure) — Blood cultures catch bacteria floating in bloodstream and tell doctors which antibiotic will kill the germ (sensitivity testing); giving antibiotics first makes cultures falsely negative and leaves treatment guessing; septic patients need antibiotics within one hour so cultures are drawn fast then antibiotics start immediately

2. Check vital signs every 4 hours and watch for fever above 100.4 degrees, heart rate over 90, or blood pressure dropping below 90 systolic — Bone infection can release bacteria into blood (bacteremia) causing sepsis where blood vessels leak fluid and blood pressure drops (septic shock); early detection allows fluid resuscitation and ICU transfer before organs fail
3. Assess pain level every shift using 0 to 10 scale and give prescribed pain medicine; position affected limb with pillows to reduce swelling and pressure on bone — Infected bone swells inside hard bone shell causing severe pressure pain; pus buildup stretches the periosteum (bone covering) which has many pain nerves; controlling pain lets patient move and prevents blood clots from lying still
4. Check blood sugar (glucose) every 6 hours in diabetic patients and report values above 180 to doctor; give insulin as ordered to keep sugar between 80 and 180 — High blood sugar (hyperglycemia) stops white blood cells (neutrophils) from killing bacteria and slows wound healing; keeping sugar controlled helps antibiotics work better and bone repair faster; infection itself raises blood sugar creating a harmful cycle
5. Protect the infected area from weight or pressure by using special boots (for foot), bed rest (for spine), or slings (for arm); coordinate with physical therapy for safe movement — Putting weight on infected bone can crush weakened bone structure causing fracture (pathologic fracture); rest allows antibiotics to reach bone without mechanical disruption and prevents bacteria from spreading to nearby joints
6. Start IV antibiotics exactly on time (vancomycin plus ceftriaxone or fluoroquinolone) and monitor IV site for redness or swelling; flush IV line before and after each dose — Bone has limited blood supply so high antibiotic levels in blood are needed for 4 to 6 weeks; vancomycin kills MRSA (methicillin-resistant Staph aureus) common in healthcare infections; ceftriaxone or fluoroquinolone covers gram-negative bacteria from diabetic foot or urinary sources; missing doses lets bacteria grow resistant
7. Prepare patient for bone biopsy or surgical debridement by keeping nothing by mouth (NPO) after midnight and explaining that surgery removes dead bone (sequestrum) and pus that antibiotics cannot reach — Bone biopsy gets actual infected bone tissue to identify exact bacteria and resistance patterns; debridement removes dead (necrotic) (dead) bone that acts as hiding place for bacteria and blocks antibiotic penetration; removing infected hardware is often necessary because bacteria form biofilm (slime layer) on metal that antibiotics cannot penetrate
8. Monitor kidney function (creatinine and BUN lab tests) and ask about ringing in ears (tinnitus) or dizziness when patient is getting vancomycin, as these drugs can damage kidneys and hearing — Vancomycin is nephrotoxic (kidney-poisoning) especially with dehydration or other kidney-toxic drugs; aminoglycosides sometimes added for resistant infections damage inner ear causing permanent hearing loss (ototoxicity); catching early signs allows dose adjustment before permanent harm

Sources

- IDSA Clinical Practice Guideline for Osteomyelitis 2015
- CDC guidance on bone and joint infections
- American Academy of Orthopaedic Surgeons (AAOS)
- Cochrane reviews on diagnosis and treatment of osteomyelitis

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