

Diabetic Foot Ulcer and Wound Care

Lost feeling plus poor blood flow plus repeated rubbing equals a wound that does not heal.

THE STORY IN ONE SENTENCE

Lost feeling plus poor blood flow plus repeated rubbing equals a wound that does not heal.

“Take weight off the wound, cut out dead tissue, kill infection, and bring more blood to the foot.”

Normal anatomy — the body's job

Three shields keep your feet safe: nerve signals that make you feel pain and shift away from pressure, blood flow that feeds skin with oxygen and nutrients, and tough skin that locks germs out.

BODY'S JOB

Nerves in your feet are like smoke alarms—they scream 'Move!' when pressure builds or tissue gets hurt, so you shift your weight or take off your shoe before damage happens. Blood vessels are the delivery trucks: they bring oxygen, white blood cells, and building blocks (proteins, sugars) that skin needs to stay strong and heal cuts. Skin is the castle wall: when it is intact and well-fed, bacteria cannot break in. All three jobs must work together every second you stand, walk, or wear shoes.

1. Step on a pebble → nerves fire → you feel sharp pain → you lift your foot instantly → no wound forms.
2. Tiny cut on toe → blood rushes in → white blood cells eat bacteria → new skin cells close the gap in days → healed.
3. Pressure from shoe → nerves tell you 'too tight' → you loosen laces or change shoes → skin stays pink and healthy.

What breaks

High blood sugar over months and years poisons the nerves so they stop sending pain signals, clogs and stiffens the blood vessels so less blood reaches the foot, and makes skin dry and fragile. Without pain to warn you, without blood to heal, and without tough skin to protect, even a small bump or rub turns into a wound that will not close.

CLINICAL TERM

Diabetic peripheral neuropathy (loss of protective sensation) removes the warning system. Peripheral artery disease (PAD, narrowed leg arteries) causes starved blood flow (ischemia) (not enough blood feeding the tissue). Repetitive mechanical stress (pressure, shear, friction) at bony prominences (metatarsal heads, heel, malleoli) breaks down skin. High blood sugar (Hyperglycemia) impairs neutrophil function and collagen synthesis, slowing wound healing. Once the skin barrier is breached, polymicrobial infection (aerobes and anaerobes) invades soft tissue and bone.

How the body compensates

The body tries to fight infection by sending white blood cells and swelling the tissue to wall off bacteria, but without offloading pressure, cutting out dead tissue, and restoring blood flow, these defenses fail and the wound grows deeper. Vital signs (heart rate, blood pressure, temperature) may stay normal for weeks even as the ulcer quietly tunnels toward bone, so a stable-looking patient can still be on the path to amputation.

1. {'step': 'Local inflammation', 'why': 'The body floods the area with white blood cells and fluid (swelling, redness, warmth). This tries to trap bacteria and bring in repair crews, but poor blood flow means not enough soldiers arrive and dead tissue piles up.'}
2. {'step': 'Granulation tissue formation (when it works)', 'why': 'If pressure is removed and blood flow is decent, the wound bed fills with pink, bumpy granulation tissue (new capillaries and collagen). This is healing in action—but it only happens when the wound is offloaded and clean.'}
3. {'step': 'Systemic signs stay quiet early', 'why': 'Because the infection is local and blood flow is poor, bacteria and toxins do not spill into the bloodstream fast. Fever, high heart rate, and low blood pressure come late—often only after the infection is deep or sepsis starts.'}
4. {'step': 'Pain may never appear', 'why': 'Neuropathy blocks pain signals, so the usual alarm (throbbing, aching) is silent. The patient feels fine even as the wound opens wider.'}
5. {'step': 'Collateral vessels try to detour around blockages', 'why': 'The body grows tiny side-street blood vessels (collaterals) around clogged arteries, but in severe PAD these are not enough to feed a healing wound.'}

6. {'step': 'The tipping point: deep infection or critical starved blood flow (ischemia)', 'why': 'Once infection reaches bone (osteomyelitis) or blood flow drops below a critical threshold (ankle-brachial index <0.5, toe pressure <30 mmHg), the wound cannot heal without surgery (revascularization, debridement, or amputation). The window to save the foot closes fast.'}

Domino effect

1. Neuropathy erases the pain alarm, so repetitive pressure goes unnoticed. — Without pain, you do not feel the blister forming under the ball of your foot or the corn rubbing at the toe tip. You keep walking, and the skin breaks open.
2. An ulcer forms at a pressure point—usually the ball of the foot (metatarsal head), heel, or side of the big toe. — These spots carry the most weight and rub the most with every step. Once skin breaks, you have an open door for bacteria.
3. Poor blood flow (PAD) means the wound does not heal; it stays open for weeks or months. — Healing needs oxygen, nutrients, and immune cells delivered by blood. When arteries are narrow, not enough supply arrives, so the wound edges stay pale and ragged instead of closing.
4. Bacteria colonize the wound, then invade deeper tissue (soft tissue infection, abscess). — Skin flora (like Staph and Strep) and gut bacteria (if the ulcer is dirty) multiply in the warm, moist wound. The crippled immune system cannot clear them, so infection spreads into fat and muscle.
5. Infection tunnels down to bone, causing osteomyelitis (bone infection). — At pressure points, bone sits just beneath the skin. Bacteria follow the path of least resistance—through dead tissue and along tendons—straight into bone. Once bone is infected, oral antibiotics often cannot reach high enough levels to cure it.
6. The wound drains pus, smells foul, or probes to bone (you can touch bone with a sterile cotton swab through the ulcer). — These are the bedside clues that infection is deep. Probe-to-bone has roughly 90% positive predictive value for osteomyelitis when you can feel hard bone through the wound base.
7. Systemic infection (sepsis) or gangrene (dead black tissue) can develop if the wound is not treated. — Bacteria spill into the bloodstream, causing fever, confusion, low blood pressure (septic shock). Or blood flow drops so low that tissue dies and turns black (gangrene), and the only option left is amputation to stop the infection from spreading up the leg.
8. Amputation (toe, foot, or below-knee) becomes necessary if infection or starved blood flow (ischemia) cannot be controlled. — When bone infection will not clear with antibiotics and surgery, or when blood flow is so poor that tissue is dead and cannot recover, cutting off the dying part saves the person's life and stops pain or sepsis. Diabetes is the leading cause of non-trauma amputation in the U.S.

Symptoms · what the patient feels, and why

- Open sore on the foot that won't heal: High blood sugar damages nerves (so you don't feel pain that would make you rest the spot) and also slows down the body's repair crew (white blood cells and new skin cells can't work as well), so small breaks in the skin stay open and grow deeper instead of closing up.
- Loss of feeling in the feet (numbness or can't feel light touch): Years of high blood sugar poison the tiny nerve fibers in the feet, like frayed wires that can't send signals anymore, so your brain stops getting pain and pressure warnings from that area.
- Skin around the ulcer looks red, swollen, or feels warm: Bacteria have gotten into the wound and your immune system is sending extra blood and fighter cells to the spot (inflammation), which causes heat, redness, and puffiness.
- Foul-smelling drainage or pus coming from the sore: Bacteria are multiplying inside the wound and breaking down tissue; the smell comes from the waste products those germs make and from dead tissue rotting.
- Foot feels cold or has weak pulses: Diabetes damages and clogs the arteries (like rust narrowing a pipe), so less oxygen-rich blood reaches the foot; without enough blood the tissue stays cold and can't heal.
- Blackened or dead-looking tissue at the edges of the wound: The tissue has died (gangrene) because it got zero blood flow or because infection killed the cells; dead tissue turns black or gray and feels leathery.
- Probe-to-bone test is positive (a sterile metal probe touches bone at the bottom of the ulcer): The ulcer has eaten so deep through skin, fat, and muscle that it reaches bone; when the probe hits something hard at the ulcer base it means the bone is exposed and very likely infected (osteomyelitis, which means bone infection).
- Ankle-brachial index (ABI) is low (less than 0.9): The ABI compares blood pressure at the ankle to blood pressure in the arm; a low number means the leg arteries are clogged and not delivering enough blood, so the wound can't get the oxygen and nutrients it needs to close.

Focused exam

- Painless ulcer at a pressure point (heel, ball of foot, or base of big toe): When a wound on the foot does not hurt, the nerves are damaged (called neuropathy). The foot cannot send pain signals to warn you about damage. Pressure points get squeezed hardest when you walk, so skin there breaks down when you cannot feel it happening. This single finding tells you the root problem: broken nerves plus constant pressure.

- Probe-to-bone test positive (a sterile metal probe can touch bone through the wound): If you can touch bone through the ulcer, infection has traveled all the way down into the bone itself (called osteomyelitis). Bone infection is serious. It means you need strong IV antibiotics for 4 to 6 weeks, not just pills or ointment. It also raises the chance of amputation if not treated right away.
- Cannot feel the 10-gram monofilament touching the foot (loss of protective sensation): A monofilament is a thin piece of nylon that bends when pressed. If the patient cannot feel it, their foot has lost protective sensation. That means they cannot feel sharp things, pressure, or blisters. This is why the ulcer formed in the first place—the body had no warning system.
- Weak or absent pulses in the foot, or ankle-brachial index (ABI) below 0.90: Wounds need oxygen and nutrients from blood to heal. If pulses are weak or the ABI is low, the arteries are too narrow (peripheral artery disease). Without enough blood flow, the ulcer will not close and infection can spread faster. You must check blood flow early.
- Swelling, warmth, redness, or pus draining from the ulcer or between the toes: These are signs of active infection spreading in the tissue. People with diabetes often cannot feel pain from infection because nerves are damaged, so swelling and pus may be the only clues that bacteria are moving deeper into the foot.
- Thick callus or hard skin around the edge of the ulcer: Callus forms when the same spot gets rubbed or pressed over and over. The body builds thick skin to try to protect itself, but that thick skin actually traps pressure underneath and makes the ulcer dig deeper instead of healing.

Diagnostics · every test answers a question

- Plain X-ray of the foot (anteroposterior, lateral, and oblique views) → Is there bone destruction, gas in the soft tissue, or foreign material inside the wound?
- Red blood cell (Erythrocyte) sedimentation rate (ESR) and C-reactive protein (CRP) → Is there ongoing inflammation or infection in the body?
- Ankle-brachial index (ABI) → Is blood flow to the foot strong enough for healing?
- Wound culture (deep tissue or bone sample, NOT swab of surface) → Which bacteria are causing the infection, and which antibiotics will kill them?
- Hemoglobin A1c (HbA1c) → How well has blood sugar been controlled over the past 2 to 3 months?
- MRI of the foot (with and without contrast) → Is there bone infection (osteomyelitis), abscess, or deep soft-tissue infection that X-rays cannot see?

Treatment logic

- Offload pressure with a total contact cast, removable cast walker (if adherent), or custom orthotic → Takes all weight and pressure off the exact spot where the ulcer is, so the skin can finally rest and start healing instead of getting crushed every time you step. Studies show offloading heals ulcers in about 12 weeks when done correctly.
- Sharp debridement (surgical removal of dead tissue, callus, and infected material) → Cuts away the dead and infected tissue so you can see how deep the wound really goes and so healthy tissue can grow in. Removes the hard callus around the edge that traps pressure. Infection cannot survive in clean, fresh tissue.
- Revascularization (angioplasty or bypass surgery) if ABI is below 0.50 or toe pressure is below 30 mmHg → Opens up the blocked arteries so oxygen-rich blood can reach the foot again. Without blood flow, wounds cannot heal no matter what else you do. Revascularization can save the limb and help ulcers close.
- Antibiotics: oral (e.g., amoxicillin-clavulanate, doxycycline, or trimethoprim-sulfamethoxazole) for mild infection; IV (e.g., vancomycin plus piperacillin-tazobactam or ceftriaxone plus metronidazole) for moderate-to-severe or bone infection → Kills the bacteria spreading through the tissue or bone. Stops the infection from destroying more tissue and causing sepsis. For osteomyelitis, you need at least 4 to 6 weeks of IV antibiotics, and sometimes surgery to remove dead bone.
- Tight blood sugar control: target HbA1c below 7% (ADA) with medication adjustment, insulin if needed, and diabetes education → Lowers blood sugar so white blood cells and platelets work better and wounds heal faster. High sugar feeds bacteria and slows healing. Controlled sugar also protects nerves and blood vessels from more damage.
- Daily foot inspection and patient education on proper footwear, moisturizing (not between toes), and when to call for help → Teaches the patient to catch new blisters, cuts, or red spots early before they become deep ulcers. Proper shoes spread pressure evenly instead of crushing one spot. Early detection prevents emergencies.

Medications

Doxycycline · Tetracycline antibiotic

Doxycycline stops bacteria from making the proteins they need to grow and multiply. Without these proteins, the bacteria cannot survive or spread in the wound. We use this for mild, shallow (superficial) infections in diabetic foot ulcers when the infection has not reached the bone or deep tissue.

1. Doxycycline enters the bacterial cell.
2. It attaches to the ribosome, which is the bacteria's protein-making factory.

3. The ribosome cannot read its instructions anymore, so it stops making new proteins.
4. Without proteins, the bacteria cannot repair itself, grow, or multiply.
5. The bacteria die or stop spreading, and the body's immune system clears them out.

SAFETY WATCH

Can cause upset stomach or nausea; take with food to reduce this. · Makes skin very sensitive to sunlight—wear sunscreen and cover skin outdoors. · Avoid in children under 8 years old because it can stain developing teeth. · Do not take with calcium, iron, or antacids—they block absorption. · Can harm a developing baby; tell your doctor if you are pregnant or might become pregnant.

Trimethoprim-sulfamethoxazole (TMP-SMX, Bactrim) · Sulfonamide antibiotic combination

TMP-SMX is two medicines working together to stop bacteria from making DNA, which is the instruction manual every cell needs to live. Without DNA, bacteria cannot grow or make copies of themselves. We use this for mild, shallow diabetic foot infections when the bacteria are common skin germs like staph or strep.

1. Trimethoprim and sulfamethoxazole both enter the bacterial cell.
2. Sulfamethoxazole blocks the first enzyme bacteria need to make folic acid (a vitamin-like chemical).
3. Trimethoprim blocks the second enzyme in the same pathway.
4. With both steps blocked, the bacteria cannot make folic acid.
5. Without folic acid, the bacteria cannot build DNA.
6. The bacteria cannot multiply or repair damage, so they die.

SAFETY WATCH

Can cause a serious skin rash (Stevens-Johnson syndrome) in rare cases—stop the medicine and call your doctor if you get a painful rash or blisters. · May raise potassium levels in the blood, especially in people with kidney disease or taking ACE inhibitors. · Do not use if you are allergic to sulfa drugs. · Can lower white blood cell counts in some people—your doctor may check blood tests. · Drink plenty of water to protect your kidneys.

Vancomycin (IV) · Glycopeptide antibiotic (IV)

Vancomycin destroys the cell wall that holds bacteria together. Without a strong wall, the bacteria burst open and die. We use IV vancomycin for deep, severe diabetic foot infections

that reach muscle or bone, especially when MRSA (a resistant staph bacteria) is suspected or proven.

1. Vancomycin enters the bloodstream through the IV line.
2. It travels to the infected tissue in the foot.
3. Vancomycin attaches to the building blocks bacteria use to make their cell wall.
4. The bacteria cannot finish building a strong wall.
5. The weak cell wall breaks, and the bacteria burst open and die.
6. The infection stops spreading, and the immune system clears out the dead bacteria.

SAFETY WATCH

Must be given slowly through an IV—too fast can cause red man syndrome (sudden flushing, itching, and low blood pressure). · Can damage the kidneys, especially in older adults or people already on other kidney-toxic drugs—your doctor will check blood tests regularly. · Can damage hearing—tell your doctor if you notice ringing in your ears or hearing loss. · Levels in the blood must be monitored to keep the dose safe and effective. · Tell your doctor if you have kidney disease—the dose may need to be lowered.

Piperacillin-tazobactam (Zosyn, IV) · Beta-lactam antibiotic combination (penicillin plus beta-lactamase inhibitor)

Piperacillin breaks the cell wall that holds bacteria together, causing them to burst and die. Tazobactam protects piperacillin from bacterial enzymes that would destroy it. Together, they kill a wide range of bacteria—including gram-negative bacteria like *Pseudomonas* and mixed infections common in deep diabetic foot ulcers.

1. Piperacillin-tazobactam enters the bloodstream through the IV.
2. It travels to the infected tissue.
3. Piperacillin attaches to enzymes that build the bacterial cell wall.
4. The bacteria cannot finish building the wall.
5. Tazobactam blocks the enzymes (beta-lactamases) that some bacteria use to destroy piperacillin.
6. With the wall incomplete, the bacteria burst and die.

SAFETY WATCH

Can cause allergic reactions, especially in people allergic to penicillin—tell your doctor about any drug allergies. · May cause diarrhea or a serious gut infection called *C. difficile* colitis—call your doctor if you have watery diarrhea or stomach cramps. · Can lower blood cell counts—your doctor may check blood tests. · High doses can cause seizures in people with kidney disease or brain disorders. · Contains sodium—may be a concern if you need to limit salt.

Metronidazole (Flagyl) · Nitroimidazole antibiotic

Metronidazole enters bacterial cells and damages their DNA so they cannot make copies of themselves or repair damage. It is especially good at killing anaerobic bacteria (bacteria that live without oxygen), which are common in deep, foul-smelling diabetic foot infections. Often used with other antibiotics to cover a broad range of bacteria.

1. Metronidazole enters the bacterial cell.
2. The bacteria's enzymes change metronidazole into an active toxic form.
3. The toxic form damages the bacteria's DNA strands.
4. The bacteria cannot repair the DNA or make new proteins.
5. The bacteria die, and the infection stops spreading.

SAFETY WATCH

Do NOT drink alcohol while taking metronidazole and for 3 days after stopping—it causes severe nausea, vomiting, flushing, and headache. · Can cause a metallic taste in the mouth. · Rarely, can cause nerve damage (peripheral neuropathy) with long-term use—tell your doctor if you feel tingling or numbness. · Can cause dark or reddish-brown urine (this is harmless). · Tell your doctor if you are pregnant—it is usually avoided in the first trimester.

Ertapenem (Invanz, IV) · Carbapenem antibiotic

Ertapenem destroys the bacterial cell wall, causing bacteria to burst and die. It works against a very wide range of bacteria, including many resistant strains. We use it for severe diabetic foot infections when multiple types of bacteria are present or when simpler antibiotics have failed.

1. Ertapenem enters the bloodstream through the IV (or deep muscle injection).
2. It travels to the infected tissue.
3. Ertapenem binds to the enzymes that build the bacterial cell wall.
4. The bacteria cannot finish building the wall.
5. The weak wall breaks, and the bacteria burst and die.

SAFETY WATCH

Can cause allergic reactions, especially in people allergic to penicillin or other beta-lactam antibiotics. · May cause diarrhea or C. difficile colitis—call your doctor for watery diarrhea. · Can lower the seizure threshold—use caution in people with a history of seizures or brain injury. · Tell your doctor if you have kidney disease—the dose may need adjustment. · Can cause pain or swelling at the injection site.

Ceftriaxone (Rocephin, IV) · Third-generation cephalosporin antibiotic

Ceftriaxone breaks the bacterial cell wall, causing bacteria to burst and die. It works against many common bacteria that infect diabetic foot ulcers, including some that are resistant to older antibiotics. Often used as a once-daily IV treatment for moderate to severe infections.

1. Ceftriaxone enters the bloodstream through the IV (or deep muscle injection).
2. It travels to the infected tissue.
3. Ceftriaxone binds to the enzymes that build the bacterial cell wall.
4. The bacteria cannot finish the wall.
5. The bacteria burst and die.

SAFETY WATCH

Can cause allergic reactions—people with penicillin allergy have a small risk of cross-reaction. · May cause diarrhea or C. difficile colitis. · Can cause gallbladder sludge or stones, especially in children—usually harmless and goes away after stopping the drug. · Do NOT mix with calcium-containing IV fluids in newborns—can cause dangerous crystals. · Tell your doctor if you have liver or kidney disease.

Linezolid (Zyvox) · Oxazolidinone antibiotic

Linezolid stops bacteria from making the proteins they need to survive. It works by blocking the very first step of protein assembly inside the bacterial cell. We use it for MRSA infections in diabetic foot ulcers, especially when vancomycin cannot be used or has failed. It can be given by mouth or IV.

1. Linezolid enters the bacterial cell.
2. It binds to the ribosome at the very start of protein assembly.
3. The ribosome cannot begin making new proteins.
4. Without proteins, the bacteria cannot grow, repair damage, or multiply.
5. The bacteria die or stop spreading.

SAFETY WATCH

Can lower blood cell counts (especially platelets and white blood cells) with long-term use—your doctor will check blood tests regularly. · Can damage the optic nerve and cause vision problems with prolonged use—tell your doctor if your vision changes. · Can cause nerve damage (peripheral neuropathy) with long-term use. · Avoid foods high in tyramine (aged cheese, cured meats, soy sauce, draft beer) while taking linezolid—can cause dangerous spikes in blood pressure. · Do not take with certain antidepressants (SSRIs, MAOIs)—can cause serotonin syndrome.

Red flags

- Fever, chills, or feeling very sick all over (suggests the infection is spreading into the bloodstream)
- Red streaks running up the leg from the wound (sign that infection is traveling through the lymph vessels)
- Black or gray dead tissue, or a foul smell (means gangrene or deep infection that can lead to losing part of the foot)
- Sudden severe pain in a foot that used to feel numb (can mean a blood clot just blocked an artery or infection hit a nerve)

Nursing priorities

1. Assess the wound for size, depth, drainage color and smell, and presence of black or gray tissue, then check pulses in both feet and compare skin temperature. — Dead (Necrotic) tissue and absent pulses tell us if gangrene or critical starved blood flow (ischemia) (severe lack of blood flow) is present—these conditions require immediate vascular surgery consultation because the foot cannot heal without adequate oxygen delivery from blood, and infection in dead tissue spreads rapidly.
2. Obtain vital signs including temperature, and inspect the leg for red streaks or swelling tracking upward from the wound. — Fever or red streaks mean the infection is spreading beyond the local wound into the bloodstream or lymphatic system, triggering systemic inflammatory response syndrome (SIRS) that can progress to septic shock where blood pressure drops dangerously and organs begin to fail.

3. Ensure complete offloading of the ulcer by verifying the patient is using a total contact cast, cast walker boot, or staying entirely non-weight-bearing with crutches or a wheelchair. — Repeated pressure and shear forces (friction during walking) crush the fragile new tissue trying to form in the wound bed and collapse tiny capillaries (smallest blood vessels) that bring oxygen and healing cells—offloading is the single most important factor in whether a diabetic foot ulcer will close.
4. Check the most recent hemoglobin A1C and current blood glucose, and coordinate with the provider to tighten glucose control to a target below 180 mg/dL. — High blood sugar impairs neutrophil function (white blood cells that kill bacteria), slows fibroblast activity (cells that make new skin), and damages small blood vessels further through glycosylation (sugar coating proteins)—wounds heal roughly half as fast when glucose stays above 200 mg/dL compared to good control.
5. Prepare for or assist with sharp debridement by gathering sterile instruments, and after debridement apply the prescribed moisture-balancing dressing such as foam, alginate, or hydrogel. — Dead tissue and thick callus act as a wall that blocks white blood cells and antibiotics from reaching bacteria, and also release inflammatory chemicals that stop healing—removing this material converts a chronic stalled wound into an acute healing wound, and the right dressing keeps the wound bed moist enough for cell migration but dry enough to prevent maceration (tissue breakdown from too much moisture).
6. Administer prescribed oral antibiotics on time if the wound shows signs of infection, and teach the patient to complete the full course even if the wound looks better. — Stopping antibiotics early allows resistant bacteria (germs that survived the first few days of treatment) to multiply and cause a harder-to-treat infection—diabetic foot infections often contain multiple species of bacteria including *Staphylococcus aureus* and *Streptococcus*, and incomplete treatment is a major cause of treatment failure and amputation.
7. Teach the patient to inspect both feet every single day using a mirror for the bottom, looking for any new red areas, blisters, cuts, or warm spots. — Neuropathy (nerve damage) means the patient cannot feel a new injury forming, so visual inspection becomes the only early-warning system—catching a small blister or red pressure area within 24 hours allows simple offloading to prevent it from becoming a full-thickness ulcer that takes months to heal.
8. Coordinate vascular studies including ankle-brachial index (ABI) and toe pressures if pulses are diminished, the wound is not improving after two weeks, or the skin looks pale or dusky. — An ABI below 0.50 or toe pressure below 30 mmHg means the tissue is starving for oxygen and will not heal without revascularization (opening blocked arteries)—trying to heal a severely blood-starved (ischemic) ulcer with dressings alone wastes time while the tissue dies, leading to amputation that could have been prevented with angioplasty or bypass surgery.

Sources

- American Diabetes Association (ADA) Standards of Care
- Infectious Diseases Society of America (IDSA) Diabetic Foot Infection Guideline
- Society for Vascular Surgery Wound Care Guideline
- International Working Group on the Diabetic Foot (IWGDF)

Last reviewed: 2026-07-07