

Cellulitis

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THE STORY IN ONE SENTENCE

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“Cellulitis is an infection in the skin and soft tissue. Your job is to figure out how deep it goes, which bacteria caused it, and whether it's spreading fast.”

Normal anatomy — the body's job

Your skin has three layers: the thin outer epidermis, the thicker dermis (full of blood vessels, nerves, and immune cells), and the subcutaneous fat layer underneath. Lymphatic vessels drain fluid and trapped germs to lymph nodes, which filter them out. Immune cells patrol constantly and kill bacteria that sneak through tiny breaks. This keeps you safe every day.

BODY'S JOB

The skin's job is to be a wall that keeps bacteria out. The dermis brings blood (oxygen and nutrients) and immune cells to fight any germs that get in. Lymphatics carry away fluid and germs so they don't build up. If a break happens—like a cut, scrape, bug bite, cracked skin, athlete's foot, or eczema—bacteria can get through, and the body has to flood the area with extra blood flow and white blood cells to trap and kill them before they spread.

1. Picture the skin as a brick wall: the epidermis is the outer paint, the dermis is the bricks (full of blood vessels and nerves), and the subcutaneous fat is the insulation behind the wall.
2. Lymphatic vessels are like tiny drains that carry away trash and germs to filter stations (lymph nodes) in your armpits, groin, and neck.
3. Immune cells (white blood cells) are like guards patrolling the wall—they grab and kill bacteria before they can spread.
4. When the wall is intact, bacteria can't get in. When there's a break (cut, bug bite, athlete's foot crack), bacteria sneak through, and the body rushes extra blood, fluid, and immune cells to that spot to trap and kill them—that's what inflammation is.

5. If the bacteria multiply faster than the immune cells can kill them, the infection spreads deeper into the fat layer or even into the fascia (the tough tissue wrapping muscles), and that's when things get dangerous.

What breaks

A break in the skin lets bacteria in. The two most common bacteria are *Streptococcus* (strep) and *Staphylococcus* (staph). Strep spreads fast through the lymphatic vessels. Staph tends to form pus pockets (abscesses). Anything that damages the skin or weakens the immune system makes cellulitis more likely: cuts, scrapes, bug bites, athlete's foot, eczema, IV drug use (needles poke holes), diabetes (high blood sugar slows immune cells), lymphedema (swollen limbs from bad lymphatic drainage), and venous insufficiency (blood pools in the legs and the skin gets fragile).

CLINICAL TERM

Cellulitis happens when bacteria invade the dermis and subcutaneous tissue through a break in the skin. The most common organisms are beta-destroying red blood cells (hemolytic) *Streptococcus* (especially group A Strep, which spreads rapidly via lymphatics and causes lymphangitic streaking) and *Staphylococcus aureus* (which tends to form localized abscesses). Risk factors include traumatic wounds, tinea pedis (athlete's foot with toe-web maceration), eczema, insect bites, IV drug use, diabetes mellitus (impaired neutrophil function and microvascular disease), chronic lymphedema (from surgery, radiation, or filariasis), and chronic venous insufficiency (venous stasis ulcers). The infection triggers acute inflammation: vasodilation (warmth), redness (erythema) (redness from increased blood flow), swelling (edema) (swelling from capillary leak), and tenderness (from pressure on nerve endings). If bacteria spread systemically, the result is fever, a fast heart rate (tachycardia), and a high white blood cell count (leukocytosis). Severe infection—especially necrotizing fasciitis—causes tissue starved blood flow (ischemia) and tissue death (necrosis), appearing as bullae, a crackling feel or sound (crepitus) (subcutaneous gas from anaerobic bacteria), or dusky/black skin.

How the body compensates

The body tries to keep vital signs normal by flooding the infected area with blood, white blood cells, and fluid. Early on, blood pressure, heart rate, and oxygen levels may look fine even though the infection is spreading. The danger is that the team can miss how sick the patient really is. Watch for the tipping point: necrotizing fasciitis (flesh-eating infection) or sepsis (bacteria in the bloodstream causing organ failure). Once the body can't keep up, the patient

crashes fast—blood pressure drops, organs fail, and the window to save them closes. Look at the trend (is the patient getting better or worse?) not just one set of numbers.

1. {'step': 'The body floods the infected area with extra blood flow (vasodilation).', 'why': 'More blood brings oxygen, nutrients, and white blood cells to fight the bacteria. This is why the skin feels warm and looks red.'}
2. {'step': 'Capillaries leak fluid into the tissue (edema).', 'why': 'The fluid carries antibodies and white blood cells to surround and trap bacteria. This is why the area swells.'}
3. {'step': 'White blood cells (neutrophils) rush in and release chemicals to kill bacteria.', 'why': 'The chemicals also cause pain (by stimulating nerve endings) and more swelling. This is why the area is tender.'}
4. {'step': 'If the infection is big or spreading, the whole body raises its temperature (fever) and makes more white blood cells (leukocytosis).', 'why': 'Heat makes it harder for bacteria to grow, and extra white blood cells give the immune system more soldiers.'}
5. {'step': 'Heart rate goes up (tachycardia) to pump more blood to the infected area and keep blood pressure steady.', 'why': 'The body is trying to deliver enough oxygen and immune cells to win the fight.'}
6. {'step': '"This compensation works—until it doesn't. Watch for the crash point: pain way worse than the redness suggests (necrotizing fasciitis), confusion or low blood pressure (sepsis), or skin turning black (tissue death).", 'why': '"Once the body can't keep up—because bacteria are multiplying too fast, toxins are destroying tissue, or organs are failing—the patient tips into shock. Blood pressure drops, organs shut down, and survival depends on acting in the next few hours (IV antibiotics, fluids, surgery if needed)."}'
7. {'step': 'The key is to catch the crash before it happens. Look at trends: Is the redness spreading? Is the pain getting worse? Is the heart rate climbing? Is the patient more confused?', 'why': '"One set of normal vital signs can be misleading. The body can look stable while it's working hard to compensate—then suddenly tip over. Serial exams (marking the border of redness with a pen and checking every few hours) and watching how fast things change tell you more than a single snapshot."}'

Domino effect

1. A break in the skin (cut, bug bite, athlete's foot crack, eczema) lets bacteria in. — The epidermis is your wall. When it's broken, bacteria (usually *Streptococcus* or *Staphylococcus*) can get through and start multiplying in the dermis and subcutaneous fat.

2. The body floods the area with blood, white blood cells, and fluid—this is inflammation. — The four classic signs are warmth (from extra blood flow), redness (from dilated blood vessels), swelling (from fluid leaking out of capillaries), and tenderness (from chemicals that irritate nerve endings). This isn't the sickness—it's the body trying to trap and kill the bacteria.
3. If *Streptococcus* is spreading through lymphatic vessels, you see red streaks pointing upward (lymphangitic streaking). — Strep bacteria travel through the lymphatics (tiny drainage tubes) toward the nearest lymph node. The lymphatics become inflamed and visible as red streaks on the skin. The lymph node swells and hurts because it's filtering out bacteria.
4. If the infection is big or bacteria spill into the bloodstream, you get fever and a high white blood cell count (leukocytosis). — Fever is the body turning up the heat to slow bacterial growth and speed up the immune system. A high white blood cell count (Leukocytosis) means the bone marrow is making extra white blood cells to fight. These are the body's general alarm signals.
5. If the infection spreads deep and fast—especially necrotizing fasciitis—tissue starts dying because blood vessels are destroyed and oxygen can't get through. — You'll see bullae (large fluid-filled blisters), skin turning purple or black (necrosis), a crackling feel or sound (crepitus) (a crackling feeling from gas under the skin made by anaerobic bacteria), and pain that's way worse than the redness suggests. This is a surgical emergency—you need to cut out the dead tissue and give IV antibiotics immediately, or the patient will die.
6. If bacteria get into the bloodstream (bacteremia) and trigger a whole-body inflammatory response, the result is sepsis. — Sepsis means the infection is so bad that organs start failing: blood pressure drops (septic shock), kidneys stop making urine, the brain gets confused, lungs fill with fluid. The body's defense system goes into overdrive and accidentally damages its own tissues. This is life-threatening and needs ICU care, IV fluids, antibiotics, and sometimes medications to keep blood pressure up.
7. Every sign (warmth, redness, streaks, fever, pus) is the body fighting back—not the disease itself. — The bacteria are the enemy. The inflammation is your body's army. Treat the bacteria (with antibiotics) and support the body (fluids, pain control, wound care). Don't just treat the redness—find and fix the break that let bacteria in (clean the wound, treat athlete's foot, control eczema) so it doesn't happen again.
8. Trace it backward: skin keeps bacteria out → a break lets them in → the body floods the area to trap and kill them → if the body wins, the infection clears; if the bacteria win, the infection spreads deeper and faster until you intervene. — Antibiotics tip the balance in the body's favor by killing bacteria faster than they can multiply. Drainage (if there's an abscess) removes the pus so antibiotics can reach live tissue. Fixing the break (healing the wound, treating tinea pedis, managing eczema or venous insufficiency) prevents bacteria from getting back in after you've cleared the infection.

Symptoms · what the patient feels, and why

- Warm, red, tender skin that spreads out without a clear border: Bacteria (usually strep or staph) invade the deeper skin layers and trigger the body's alarm system—blood vessels open up to rush immune cells to the spot, which makes the area red, warm, and puffy.
- Swelling of the infected area: Extra fluid leaks out of blood vessels into the tissue because the infection makes vessel walls leaky—like a garden hose with tiny holes spraying water into the yard.
- Pain and tenderness when you press on the area: Stretching from swelling and chemicals released by immune cells irritate nerve endings in the skin—those nerves then send pain signals to the brain.
- Warm skin over the red area: Blood flow increases to bring more germ-fighting white cells, and blood carries heat—so more blood means the skin feels warmer than the skin around it.
- Sharp, clear edge to the redness (suggests erysipelas, a type of cellulitis closer to the surface): Erysipelas stays in the top skin layer (dermis), so the infection front forms a clear boundary—like drawing a line with a marker instead of watercolor bleeding into paper.
- Fever and chills: The immune system resets the body's thermostat higher to help kill bacteria (many germs die at higher temperatures), and shaking (chills) generates heat quickly.
- A soft, squishy spot under the skin (fluctuance): Pus (dead white cells and bacteria) collects in a pocket called an abscess—pressing on it feels like pushing on a water balloon because liquid moves inside.
- Pain that feels much worse than the redness looks: Bacteria may be eating away deeper tissue layers (necrotizing fasciitis)—those dying tissues release chemicals that scream at nerve endings even when the surface looks fairly normal.

Focused exam

- Poorly marked warm red area, usually on the lower leg or foot, with edges that blend into normal skin: This is cellulitis — bacteria growing in the deep skin layers and the fat underneath. The edges blend because bacteria spread along tissue spaces, not in a straight line the way a burn or rash would.
- Tenderness and swelling that stays dented when you press it (pitting swelling (edema)): Pitting swelling (edema) means fluid is pooling in the tissue spaces. The body is flooding the area with white blood cells and fluid to fight the bacteria, and the swelling proves that response is happening.
- Red streaks running up the arm or leg toward lymph nodes (lymphangitic streaks): These streaks show bacteria are traveling up the lymph channels, the body's drainage pipes. It means the infection is spreading and the immune system is working overtime to catch up.

- **Fever and chills:** When cellulitis bacteria spill into the bloodstream, the body cranks up its internal temperature to kill them. Fever is the body's weapon, not the disease itself.
- **A break in the skin** — cut, crack, athlete's foot, eczema, or old wound — in or near the red area: This is the door bacteria used to get in. Without a break, cellulitis does not start. Find and treat this break or cellulitis will come back.
- **Pain** that is way worse than what you see on the skin, especially with purple blisters or a crackling feeling under the skin (crepitus): This is a red flag for necrotizing fasciitis (flesh-eating infection), not simple cellulitis. Necrotizing fasciitis destroys tissue fast and needs emergency surgery plus antibiotics, not just antibiotics alone.
- **A soft squishy pocket of pus** inside the swollen area (fluctuance): An abscess (walled-off pocket of pus) has formed. Cellulitis can turn into an abscess if staph bacteria wall off the infection. Antibiotics cannot reach pus — the pocket must be drained.

Diagnostics · every test answers a question

- **Complete blood count (CBC) with differential** → Is the body making extra white blood cells to fight bacteria?
- **C-reactive protein (CRP)** → How much inflammation is the body making in response to the infection?
- **Blood cultures** (two sets from different sites) → Are bacteria in the bloodstream (bacteremia)?
- **Wound culture or aspirate** from the leading edge of redness (if pus-filled (purulent)) → Which bacteria are causing the infection?
- **Ultrasound** of the swollen area → Is there a hidden abscess pocket that needs to be drained?
- **Creatine kinase (CK) level** — if severe pain or concern for necrotizing infection → Is muscle being destroyed (a sign of necrotizing fasciitis)?

Treatment logic

- **Find the break in the skin** (wound, athlete's foot, eczema, IV site) and classify cellulitis as pus-filled (purulent) (with pus or abscess) or non-pus-filled (spreading redness only) → Pus-filled (Purulent) cellulitis is usually caused by Staph aureus (including MRSA). Non-pus-filled is usually Streptococcus. This one choice tells you which antibiotic family to use.
- **Non-pus-filled (purulent) cellulitis:** give cephalexin 500 mg by mouth four times a day, or cefazolin 1-2 g IV every 8 hours (IDSA 2014) → These drugs kill Streptococcus pyogenes and methicillin-sensitive Staph aureus in the skin layers where they are spreading. Stops bacteria from multiplying and lets the immune system catch up.

- Pus-filled (Purulent) cellulitis or suspected MRSA: give clindamycin 300–450 mg by mouth three times a day, doxycycline 100 mg twice a day, trimethoprim-sulfamethoxazole (TMP-SMX) 1–2 double-strength tablets twice a day, or vancomycin 15–20 mg/kg IV every 8–12 hours (IDSA 2014) → These drugs kill Staph aureus including MRSA strains that hide in pus pockets and resist many older antibiotics. Targets the bacteria that cause abscesses.
- Drain any fluctuant abscess with needle aspiration or surgical incision and drainage → Removes the pus pocket where bacteria cluster at very high numbers. Lowers the bacterial load so antibiotics can reach and kill the remaining bacteria more easily.
- Elevate the affected limb above the level of the heart and apply cool compresses → Improves blood and lymph drainage out of the swollen area. Reduces the four inflammation signs (warmth, redness, swelling, pain) and gives the immune system breathing room to work.
- Treat the underlying skin break: antifungal cream for athlete's foot (tinea pedis), moisturizer and steroid cream for eczema, compression stockings for venous insufficiency → Closes the door bacteria used to get in so they cannot re-enter after this infection heals. Stops the cellulitis cycle from repeating.
- Admit to hospital for IV antibiotics if patient has systemic toxicity (high fever, low blood pressure, confusion), failed outpatient treatment, or cannot take pills → IV antibiotics reach higher blood levels faster and work when the gut is not absorbing pills well. Close monitoring catches worsening infection (spreading redness, rising heart rate or fever) before it becomes life-threatening.

Medications

Cephalexin (oral) or Cefazolin (IV) · First-generation cephalosporin (a beta-lactam antibiotic)

These antibiotics punch holes in the cell wall of certain bacteria—especially Streptococcus (strep) and some Staphylococcus (staph) that don't resist them. Without a solid wall, the bacteria burst and die, stopping the infection from spreading.

1. The drug enters your bloodstream and reaches the infected tissue.
2. It binds to proteins (penicillin-binding proteins) that bacteria use to build their cell walls.
3. The bacteria can no longer finish making their protective wall.
4. Water rushes into the bacteria through weak spots, and they pop like over-filled water balloons.
5. Your immune system cleans up the debris, and swelling and redness start to fade.

SAFETY WATCH

Allergy: some people allergic to penicillin can also react to cephalosporins—mention any past antibiotic rash to your doctor. · Diarrhea is common; severe watery or bloody diarrhea (C. difficile) needs urgent attention. · Kidney function: the dose may need to be lowered if your kidneys don't clear the drug well. · Drug interactions: tell your provider about all medicines, especially blood thinners.

Dicloxacillin (oral) · Penicillinase-resistant penicillin (a beta-lactam antibiotic)

Dicloxacillin blocks the same cell-wall-building step as cephalexin, but it is built to resist the enzymes (beta-lactamases) that regular penicillin-sensitive staph bacteria make. It works best against non-MRSA staph and strep.

1. You swallow the pill; it is absorbed in your intestine and enters your bloodstream.
2. The drug travels to the infected skin and binds to penicillin-binding proteins on staph or strep bacteria.
3. These proteins normally help weave strong cell walls; the drug jams the weaving machinery.
4. The bacteria try to grow but split open because their walls are too weak.
5. Your white blood cells finish clearing the dead bacteria, and the red, hot swelling begins to shrink.

SAFETY WATCH

Penicillin allergy: anyone with a history of severe penicillin reaction (hives, swelling, trouble breathing) should avoid this drug. · Upset stomach or diarrhea; take with food to reduce nausea. · Liver enzymes can rise; rarely causes serious liver inflammation. · Less effective if you miss doses or stop early—finish the full course even when you feel better.

Clindamycin (oral or IV) · Lincosamide antibiotic

Clindamycin gets inside bacteria and jams their protein-making factory (the ribosome). Without new proteins, the bacteria cannot grow, repair themselves, or make toxins. It works against many staph (including some community-acquired MRSA) and strep strains.

1. The drug enters your bloodstream and concentrates in infected tissue.
2. It slips into bacterial cells and locks onto the 50S part of the ribosome.
3. The ribosome can no longer string amino acids together to make proteins.
4. The bacteria stop growing and eventually die because they run out of essential parts.
5. Inflammation drops as the bacterial load falls, and your skin begins to heal.

SAFETY WATCH

C. difficile colitis: clindamycin is one of the antibiotics most likely to trigger severe, watery, sometimes bloody diarrhea caused by this gut bacterium—stop the drug and call your doctor if diarrhea starts. · Nausea and metallic taste are common. · Liver and kidney function should be monitored in long courses. · Rare but serious: severe skin reactions (Stevens-Johnson syndrome) if you develop blistering rash.

Doxycycline (oral) · Tetracycline antibiotic

Doxycycline also blocks the bacterial ribosome, stopping protein production. Many community-acquired MRSA strains are still sensitive to it, along with strep. It is taken by mouth and penetrates tissue well.

1. You swallow the capsule; it is absorbed in your stomach and small intestine.
2. The drug circulates in your blood and enters infected skin.
3. Inside bacteria, it binds to the 30S ribosomal subunit and prevents new protein chains from starting.
4. Bacteria cannot repair damage or multiply, so the infection stops spreading.
5. Your immune cells clean up the weakened bacteria, and swelling and pain decrease.

SAFETY WATCH

Sun sensitivity: your skin can burn much more easily—use sunscreen and cover up outdoors. · Upset stomach: take with food (but not dairy, which blocks absorption). · Do NOT use in pregnancy or in children under 8 years—can stain developing teeth and bones. · Rare: swelling of the brain (pseudotumor cerebri) causing headache and vision changes.

Trimethoprim-sulfamethoxazole (TMP-SMX, oral) · Folate synthesis inhibitor (combination sulfonamide antibiotic)

This combination drug blocks two different steps in how bacteria make folate (a vitamin they need to build DNA). Without folate, bacteria cannot copy their genetic code or multiply. It works well against many MRSA strains.

1. You swallow the tablet; both drugs are absorbed and enter your bloodstream.
2. They concentrate in infected tissue and penetrate bacterial cells.
3. Trimethoprim blocks one enzyme; sulfamethoxazole blocks another—both are needed to make folate.
4. Bacteria run out of folate and cannot make DNA or RNA.
5. Without new genetic material, bacteria stop dividing and die; your immune system finishes the job.

SAFETY WATCH

Allergy: sulfa drugs can cause rash (including serious blistering reactions like Stevens-Johnson syndrome) or liver and blood-cell problems—stop immediately if rash appears. · Drink plenty of water to prevent kidney crystals. · Can raise potassium, especially in people with kidney disease or on certain blood-pressure medicines. · Not safe in late pregnancy (can harm the baby) or in infants under 2 months. · Drug interactions: can boost blood thinners and diabetes medicines—watch for bleeding or low blood sugar.

Vancomycin (IV) · Glycopeptide antibiotic

Vancomycin is a large molecule that blocks cell-wall building from the outside of the bacterial membrane. It binds to the wall pieces before they are stitched together, so the wall stays weak and the bacteria burst. It is the go-to IV drug for serious MRSA infections.

1. The drug is given through an IV line into a vein.
2. It travels through your bloodstream to the infected area.
3. Vancomycin wraps around the building blocks (D-Ala-D-Ala peptides) of the bacterial cell wall.
4. Enzymes cannot link the blocks together, so the wall remains full of holes.
5. Bacteria swell and pop; inflammation calms as the infection clears.

SAFETY WATCH

Red man syndrome: if infused too fast, can cause flushing, itching, and low blood pressure—the IV must run slowly. · Kidney damage: especially if combined with other kidney-toxic drugs or in people with existing kidney disease—levels are monitored with blood tests. · Hearing loss (rare) with high levels or long courses. · Vein irritation: the IV site can become sore or inflamed.

Piperacillin-tazobactam (IV) · Extended-spectrum penicillin plus beta-lactamase inhibitor

Piperacillin punches holes in bacterial cell walls like other penicillins, but it works on a much wider range of bacteria (Gram-positive, Gram-negative, and anaerobes). Tazobactam shields piperacillin from enzymes that would break it down, so the combination can kill tougher bugs often found in diabetic foot infections or dirty wounds.

1. The drug is given through an IV.
2. Piperacillin reaches infected tissue and binds to the wall-building machinery of many different bacteria.

3. Tazobactam inactivates beta-lactamase enzymes that bacteria produce to defend themselves.
4. With their enzymes blocked, bacteria cannot repair or finish building their walls.
5. Bacteria lyse (burst), and the immune system clears the debris, reducing swelling, redness, and pain.

SAFETY WATCH

Penicillin allergy: cross-reacts in people allergic to penicillin—mention any past reactions. · Diarrhea and risk of *C. difficile* colitis. · Can lower platelets or cause bleeding in rare cases. · Kidney function and electrolytes (especially sodium and potassium) should be monitored. · Drug interactions with blood thinners and some other antibiotics.

Ertapenem, Meropenem, or Imipenem (IV carbapenems) · Carbapenem (a very broad-spectrum beta-lactam antibiotic)

Carbapenems work like penicillins—breaking bacterial cell walls—but they resist nearly all the enzymes bacteria use for defense, so they kill an extremely wide range of bacteria (Gram-positive, Gram-negative, anaerobes). Reserved for severe or complicated infections, such as diabetic foot with mixed bacteria or when other antibiotics have failed.

1. The drug is infused into a vein.
2. It spreads through blood to infected tissue.
3. Inside the tissue, it binds to penicillin-binding proteins on many types of bacteria.
4. Cell-wall synthesis stops; bacteria burst under their own internal pressure.
5. Infection burden drops rapidly, and inflammation begins to resolve.

SAFETY WATCH

Seizures: especially in people with kidney disease, brain injury, or on high doses—dose must be adjusted for kidney function. · Allergy: can cross-react with penicillin allergies. · *C. difficile* diarrhea. · Meropenem and imipenem require more frequent dosing than ertapenem; choice depends on kidney function and coverage needs. · Should be reserved for truly resistant or life-threatening infections to preserve effectiveness.

Red flags

- Pain that is far worse than what you see on the skin (may signal necrotizing fasciitis, a fast-moving infection that destroys muscle and tissue)
- Purple or black patches, blisters filled with blood, or skin that feels numb (signs tissue is dying)

- Fever over 100.4°F with confusion, very fast heart rate, or feeling faint (signs bacteria may be in the bloodstream—sepsis)
- Swelling that spreads quickly over hours (suggests aggressive infection needing urgent treatment)
- A pocket of pus (abscess) that needs drainage, not just antibiotics

Nursing priorities

1. Mark the border of the redness with a pen and write the date and time next to it. — Cellulitis spreads outward as bacteria multiply in the dermis (middle skin layer) and subcutaneous fat; marking the edge lets the next nurse see if antibiotics are stopping the infection or if it is getting worse and needs stronger drugs or IV therapy.
2. Elevate the infected arm or leg above the level of the heart whenever the patient sits or lies down. — Gravity pulls fluid and white blood cells down into tissue; elevation uses gravity to drain interstitial fluid (fluid between cells) back into veins and lymph vessels, which reduces swelling (edema) and pain and speeds antibiotic delivery to the infection site.
3. Give the first dose of antibiotic within one hour of the order and teach the patient to take every dose on time at home. — Bacteria double in number every 20 to 30 minutes; delaying antibiotics lets the colony grow and invade deeper layers or enter the bloodstream, and missing doses at home creates low drug levels that allow resistant bacteria to survive and multiply.
4. Inspect the skin closely for any drainage, blisters, black spots, or areas that feel hard or numb, and report these findings immediately. — Pus-filled (Purulent) drainage means an abscess that needs incision and drainage; bullae (large blisters), purple or black patches (ecchymosis or tissue death (necrosis)), and woody hardened tissue (induration) (hard, board-like tissue) signal necrotizing fasciitis or clostridial gas gangrene, both surgical emergencies.
5. Check temperature, heart rate, blood pressure, and mental status every 4 hours and use the SIRS criteria or qSOFA score to screen for sepsis. — Cellulitis can seed bacteria into the bloodstream (bacteremia), triggering sepsis (dysregulated immune response causing organ damage); early sepsis shows as fever or hypothermia, a fast heart rate (tachycardia), fast breathing (tachypnea) (fast breathing), low blood pressure (hypotension), and altered mentation, and catching it early cuts mortality in half.
6. Apply warm, moist compresses to the red area for 20 minutes three to four times a day. — Heat causes vasodilation (widening of blood vessels), which increases blood flow and brings more neutrophils (infection-fighting white cells) and antibiotics into the infected tissue; warmth also improves lymphatic drainage to clear dead bacteria and toxins.

7. Treat the portal of entry: apply antifungal cream to athlete's foot, antibiotic ointment to cuts, or emollient lotion to cracked eczema skin. — Tinea pedis (athlete's foot) cracks the toe webs and lets Streptococcus or Staphylococcus enter; untreated portals allow reinfection even after antibiotics cure the cellulitis, creating a cycle of recurrent infections that damage lymph vessels and lead to chronic leg swelling.
8. Teach the patient to finish all antibiotic pills even when the redness fades and to return if fever comes back or the red area grows despite two days of treatment. — Stopping antibiotics early leaves dormant bacteria (persisters) in tissue that restart infection within days; failure to improve in 48 to 72 hours means the bacteria are resistant to the chosen drug or an abscess is hidden under the skin and needs imaging and drainage.

Sources

- IDSA Skin and Soft Tissue Infections Guideline 2014
- CDC
- UpToDate
- American Academy of Dermatology

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