

Necrotizing Soft Tissue Infection

NSTI is a flesh-eating infection that spreads in hours along the fascia — only immediate surgery saves lives.

THE ONE-LINE TRUTH

Bacteria eat through the tissue layer under the skin so fast that without emergency surgery most people die.

The chain — how it unfolds

1. Bacteria enter through a small break in the skin — a cut, scrape, bug bite, blister, or injection site. — why: The skin is the body's main barrier against germs, so any break, no matter how tiny, gives bacteria a door into the tissue underneath, especially if the person's immune system is weakened by diabetes, cancer, or other illness.
2. The bacteria land in the fascia and start multiplying and releasing toxins within hours. — why: The fascia has fewer immune cells and less blood flow than muscle, so bacteria can grow explosively there. Group A strep releases toxins like streptococcal pyrogenic exotoxin that punch holes in cells, and Clostridium releases alpha toxin that destroys cell membranes and makes gas — both melt tissue fast.
3. The patient feels severe pain that seems way out of proportion to the size of the wound. — why: The toxins damage nerve endings in the fascia, and the fascia itself is packed with pain sensors (nociceptors), so even though the skin looks almost normal, the deep tissue destruction triggers alarm signals that feel like the worst pain the person has ever had.
4. The skin over the infected area turns dusky, purple, blue, or black, and feels tight and swollen. — why: The toxins and inflammation cause blood vessels in and under the skin to clot off (thrombose), cutting off blood flow (ischemia), so the skin dies from lack of oxygen and turns dark — this is called bruising (ecchymosis) and tissue death (necrosis).
5. Blisters (bullae) form, filled with dark red or purple fluid that looks like blood mixed with serum. — why: The dying blood vessels leak plasma and red blood cells into the tissue, and the body tries to wall off the toxins by forming fluid-filled pockets, which show up as blisters on the skin surface.

6. A crackling or popping sound (crepitus) can be heard or felt under the skin, especially with Clostridium infections. — why: Clostridium bacteria ferment tissue and make carbon dioxide and hydrogen gas, which collects in bubbles under the skin and pops when you press on it or move the limb — this is called subcutaneous emphysema.
7. The person develops fever or sometimes low temperature, fast heart rate, low blood pressure, confusion, and decreased urine output — signs of sepsis and septic shock. — why: The bacterial toxins spill into the bloodstream and trigger the whole immune system to release cytokines (inflammatory messengers like TNF-alpha and interleukin-1), which make blood vessels leak, drop blood pressure, and impair organs like the kidneys, lungs, and brain — this is systemic inflammatory response syndrome (SIRS) and septic shock.
8. Without emergency surgery to remove all the dead tissue, the infection spreads along the fascia, more tissue dies, and the person goes into multi-organ failure and dies in one to three days. — why: Antibiotics cannot reach dead tissue because there is no blood flow, so the bacteria keep multiplying and making toxins. Only cutting out every bit of dead and dying tissue (often requiring amputation or multiple surgeries) stops the source, and even then the systemic damage may be too far along to reverse.

Spot it — what you see, and why

- Pain much worse than you would expect from how the skin looks: The bacteria release toxins (poisons) that destroy muscle and soft tissue deep under the skin, and the dying tissue triggers pain nerves long before the skin shows all the damage on the outside.
- Skin turns dark red, purple, or blue-black: The infection kills blood vessels under the skin, so blood cannot reach the tissue anymore. When tissue dies from lack of oxygen, it turns dark, like a bruise that never heals.
- Blisters filled with dark red or purple fluid: The toxins from the bacteria damage the walls of tiny blood vessels, so blood leaks out into pockets between the layers of skin, forming blisters filled with blood instead of clear fluid.
- Crackling or popping feeling under the skin when you press on it: Some of the bacteria make gas as they digest tissue. The gas bubbles get trapped under the skin, and when you press on them, they crackle like bubble wrap—this is called a crackling feel or sound (crepitus).
- The skin feels numb or you cannot feel touch in the infected area: The infection destroys the nerves that carry feeling signals from the skin to the brain, so even though the tissue is badly damaged, it stops hurting and you lose sensation there.
- High fever and chills that shake your whole body: The bacteria flood into the bloodstream and the immune system cranks up the body's thermostat (in the hypothalamus, the brain's temperature control center) to fight the infection, causing fever. Chills happen when the body tries to make heat to reach the new set point.

Red flags — act now

- Pain far worse than the skin looks—this means deep tissue is dying even if the surface looks okay
- Skin turning dark purple, blue, or black—a sign that blood flow is cut off and tissue is dead
- Crackling or popping feeling under the skin—gas from bacteria eating tissue
- Numbness in the infected area even though it was painful before—nerves are destroyed
- Confusion, very low blood pressure, or not urinating—the infection has spread to the bloodstream and organs are shutting down

Treat it — in this order

1. Emergency surgical debridement (remove all dead and infected tissue) → Stops the bacteria from spreading deeper into healthy tissue by cutting away all dead fascia, muscle, and skin. Removes the source of toxins that are killing cells and poisoning the bloodstream. Surgery lowers the bacterial load instantly and gives antibiotics a chance to work.
2. Broad-spectrum IV antibiotics: vancomycin PLUS piperacillin-tazobactam (or a carbapenem like meropenem) PLUS clindamycin → Vancomycin kills group A Streptococcus (the most common cause) and methicillin-resistant Staphylococcus aureus (MRSA). Piperacillin-tazobactam or meropenem kills most aerobic and anaerobic bacteria (important for mixed infections). Clindamycin stops the ribosomes in bacteria so they cannot make toxins—this is critical because streptococcal pyrogenic exotoxins and Clostridium toxins drive tissue death and shock.
3. Aggressive IV fluid resuscitation and vasopressors (norepinephrine first-line) → Fills the blood vessels back up (septic shock causes massive fluid leak into tissues) and squeezes blood vessels tight so blood pressure stays high enough to push oxygen to dying tissues. Keeps the heart pumping hard enough to fight sepsis and deliver antibiotics.
4. Intravenous immunoglobulin (IVIG), especially for streptococcal toxic shock syndrome → IVIG is pooled antibodies from thousands of donors. It neutralizes streptococcal pyrogenic exotoxins (super-toxins made by group A strep) that trigger toxic shock and organ failure. IVIG also calms down the overactive immune response that damages organs.
5. Hyperbaric oxygen therapy (breathing 100% oxygen in a pressurized chamber) → Forces extra oxygen into the blood and into tissues at the edge of the infection. Higher oxygen levels slow the growth of anaerobic bacteria (like Clostridium) and help white blood cells (neutrophils) kill germs better. May reduce the need for amputation in limb infections.

Your top priorities at the bedside

1. Establish large-bore IV access (18-gauge or bigger) in two sites and start high-volume normal saline boluses while monitoring blood pressure every 15 minutes — why: Necrotizing infection releases toxins that make blood vessels leak fluid into tissues; patients lose circulating blood volume rapidly and go into septic shock where organs stop getting oxygen; early aggressive fluid replacement keeps blood pressure high enough to perfuse kidneys and brain
2. Draw blood cultures from two different sites before any antibiotics, then give piperacillin-tazobactam plus vancomycin plus clindamycin within 60 minutes of recognition — why: Blood cultures identify the exact bacteria so doctors can narrow antibiotics later; clindamycin stops bacterial toxin production even when bacteria are not actively growing; every hour of antibiotic delay increases death risk because bacteria double in tissue every 20 to 30 minutes
3. Mark the borders of redness with a pen every hour and measure the area in centimeters; photograph the site if possible — why: Necrotizing infection spreads along fascia (the tissue layer covering muscles) at 1 to 2 centimeters per hour; documented rapid spread despite antibiotics confirms the need for emergency surgery and helps the surgical team plan how much tissue to remove