

Necrotizing Soft Tissue Infection

A plain-English guide for you and your family

WHAT IS HAPPENING

Necrotizing soft tissue infection (NSTI) is when bacteria get into the fascia, the thin tough sheet of tissue that wraps around muscles and keeps everything in place under your skin. Normally the fascia acts like shrink-wrap protecting muscle from germs and holding organs in neat compartments. But when certain bacteria — most often group A streptococcus (the strep that causes strep throat, but in a much more dangerous form), or Clostridium (germs that live in dirt and make gas), or a mix of germs from the gut or ocean water like Vibrio — slip in through even a tiny cut, scrape, bug bite, or needle poke, they release powerful poisons called toxins that melt the fascia and the fat and muscle around it. The infection races along that tissue layer in hours, not days. The very first sign is pain that feels way worse than the small wound looks like it should cause. Then the skin over the area turns purple, blue, or black because blood vessels are dying. You might hear a crackling sound under the skin from gas bubbles the bacteria make. Dark blisters filled with blood and fluid appear. The person's whole body goes into shock — fever or low temperature, racing heart, confusion, blood pressure drops — because the toxins flood the bloodstream and the immune system goes into overdrive trying to fight back. This is a true emergency. The only treatment that works is rushing the person to the operating room to cut out all the dead and dying tissue, plus strong antibiotics through an IV. Even with perfect care, one in three people do not survive, and many who do survive lose fingers, toes, or limbs. The key is recognizing the mismatch early — terrible pain that does not match a small wound — and getting to surgery within hours, because every hour of delay doubles the risk of death.

What you might feel

- Pain much worse than you would expect from how the skin looks
- Skin turns dark red, purple, or blue-black
- Blisters filled with dark red or purple fluid
- Crackling or popping feeling under the skin when you press on it
- The skin feels numb or you cannot feel touch in the infected area
- High fever and chills that shake your whole body

- Fast heartbeat and breathing faster than normal
- Swelling that spreads quickly, sometimes an inch or more every hour

Call the doctor right away if...

- 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

What the care team will do

- 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
- Draw blood cultures from two different sites before any antibiotics, then give piperacillin-tazobactam plus vancomycin plus clindamycin within 60 minutes of recognition
- Mark the borders of redness with a pen every hour and measure the area in centimeters; photograph the site if possible
- Notify the surgeon immediately for bedside evaluation and do not wait for lab results or imaging to call
- Check lactate level (a waste product from cells not getting oxygen) and repeat every 2 to 4 hours; notify provider if lactate is above 2 or rising
- Monitor temperature, heart rate, respiratory rate, and oxygen saturation every 15 minutes; watch for fever above 101 or temperature below 96 degrees
- Measure urine output every hour with a Foley catheter and alert provider if output drops below 30 milliliters per hour
- Keep patient NPO (nothing by mouth) and prepare for emergency operating room transport with signed consent and pre-operative checklist completed

Medicines you may be given — and why

Piperacillin-tazobactam

Piperacillin punches holes in the cell walls of many bacteria, so they burst and die. Tazobactam stops bacteria from making an enzyme (beta-lactamase) that would otherwise chew up and

destroy the piperacillin. Together they kill a wide range of germs, including many of the gut and skin bacteria that can invade dying tissue.

Vancomycin

Vancomycin blocks the building of bacterial cell walls from the outside. It targets gram-positive bacteria (bacteria with thick walls), including MRSA (methicillin-resistant *Staph aureus*), which often causes necrotizing soft tissue infections. It stops the bacteria from linking together the bricks that form their protective shell, so they fall apart and die.

Clindamycin

Clindamycin stops bacteria from making the proteins they need to live and multiply. It also has a special job in necrotizing infections: it shuts down toxin production by bacteria like group A *Streptococcus* and *Clostridium*. Even when bacteria are not growing fast (because tissue is dying and there is little oxygen), clindamycin still blocks the toxins that destroy muscle and skin.

Intravenous immunoglobulin (IVIG)

IVIG is a concentrated mix of antibodies collected from thousands of healthy donors. In necrotizing soft tissue infections complicated by streptococcal toxic shock syndrome, it floods the bloodstream with antibodies that neutralize bacterial toxins (especially superantigens from group A Strep) and calm the immune system's overreaction. This helps stop the toxins from triggering a massive, harmful immune response that causes shock and organ failure.

Intravenous fluids (crystalloids: normal saline or lactated Ringer's)

Necrotizing soft tissue infections cause massive fluid loss because toxins and inflammation make blood vessels leaky, and fluid pours out of the blood into the tissues and the space around dying muscle. This drops blood pressure dangerously low (septic shock). IV fluids rapidly refill the bloodstream, restore blood pressure, and keep oxygen and nutrients flowing to the brain, heart, kidneys, and other organs.

Vasopressors (norepinephrine)

In septic shock from necrotizing infection, blood vessels become floppy and dilated (widened) because toxins and inflammation paralyze the muscles in vessel walls. Blood pressure crashes, and organs do not get enough blood. Norepinephrine is a synthetic version of the body's natural adrenaline-like signal. It tightens the muscles in blood vessel walls, squeezing the vessels narrower and raising blood pressure so blood can reach the brain, heart, and kidneys.

Hyperbaric oxygen therapy (HBOT)

Some necrotizing infections (especially those caused by *Clostridium* bacteria, which thrive in low-oxygen tissue) release toxins and gas that destroy muscle and skin. Hyperbaric oxygen

puts the patient in a pressurized chamber and delivers 100% oxygen. The high pressure forces extra oxygen to dissolve into the blood and reach dying, low-oxygen tissue. This extra oxygen poisons anaerobic bacteria (bacteria that cannot live in oxygen), helps white blood cells kill germs, reduces swelling, and supports tissue repair.

Where this information comes from

- IDSA Clinical Practice Guideline for Skin and Soft Tissue Infections 2014
- Surgical Infection Society
- CDC
- World Journal of Emergency Surgery consensus guidelines

Last reviewed: 2026-07-07