

Necrotizing Fasciitis

A plain-English guide for you and your family

WHAT IS HAPPENING

Fascia is a thin, tough layer of tissue that wraps around and separates your muscle groups, like plastic wrap between packages of meat. It has very few blood vessels, so your immune cells and antibiotics have a hard time getting there. When bacteria break through the skin — from a cut, a scrape, surgery, an injection, or sometimes no obvious reason — they can slip into that fascia layer. Once there, they multiply fast and make poisons (toxins) that destroy the tissue and sometimes produce gas. Because blood flow is so poor in fascia, your body's defense troops can't reach the infection in time. The bacteria spread sideways along the fascia, sometimes several inches in an hour. The pain is extreme, far worse than the skin looks, because the toxins are melting tissue and firing off pain nerves deep inside. Soon the skin above turns dark red or purple, blisters filled with blood or dark fluid appear, and you might feel or hear crackling under the skin (that's gas from the bacteria). The area may go numb because the nerves die. Meanwhile, bacterial toxins pour into the bloodstream, triggering septic shock: blood pressure crashes, organs start to fail, and without treatment, death comes quickly. The only real cure is emergency surgery to cut out every bit of dead and infected tissue, plus strong antibiotics and intensive care to support the body while it fights back. Speed is everything — every hour of delay raises the risk of losing limbs or life.

What you might feel

- Severe pain that seems way worse than what you can see on the skin
- Skin turns dark purple or bronze, sometimes with blood blisters (bullae)
- A crackling feel or sound (Crepitus) — a crackling or popping feel when you press the skin
- The painful area feels numb or you cannot feel touch there
- Fever, often high (above 101°F or 38.3°C)
- Fast heart rate (tachycardia, often above 100 beats per minute)
- Low blood pressure (hypotension, systolic below 90 mmHg)
- Confusion or drowsiness (altered mental status)
- Swelling that spreads quickly, sometimes several inches per hour

Call the doctor right away if...

- Pain that is far worse than the redness or swelling you can see — never dismiss severe pain as 'just cellulitis'
- Skin that turns dark purple, bronze, or black, or blood blisters that pop up
- Crackling under the skin (crepitus), numbness in the painful area, or skin that feels wooden and hard
- Fast heart rate, low blood pressure, confusion, or signs of shock — these mean the infection is spreading into the bloodstream
- Swelling or redness that races across the skin, growing visibly over a few hours

What the care team will do

- Alert the surgeon and operating room immediately and prepare the patient for emergency surgery within one hour.
- Start two large-bore IV lines and begin aggressive IV fluid resuscitation with at least 30 milliliters per kilogram of normal saline or lactated Ringer solution in the first hour.
- Administer broad-spectrum IV antibiotics immediately—do not wait for culture results—using piperacillin-tazobactam 3.375 grams every 6 hours or a carbapenem like meropenem 1 gram every 8 hours, plus vancomycin 15 to 20 milligrams per kilogram every 8 to 12 hours, plus clindamycin 600 to 900 milligrams every 8 hours.
- Monitor blood pressure every 5 to 15 minutes and prepare to start vasopressor medications like norepinephrine if systolic blood pressure stays below 90 after fluid resuscitation.
- Measure urine output every hour with a Foley catheter and monitor for at least 0.5 milliliters per kilogram per hour.
- Check lactate level, complete blood count, creatinine, and calculate the LRINEC score using lab values to assess severity of infection.
- Assess pain level and do not rely on lack of skin redness to rule out serious infection; document exact skin appearance, any bullae, skin color changes, and presence of a crackling feel or sound (crepitus).
- Prepare the patient and family for the possibility of amputation or disfiguring surgery and arrange ICU bed for postoperative care.

Medicines you may be given — and why

Piperacillin-tazobactam

Piperacillin punches holes in the outer wall of bacteria so they fall apart and die. Tazobactam blocks the scissors (enzymes called beta-lactamases) that some bacteria use to cut up

antibiotics, so the piperacillin can keep working. Together they kill a wide range of bacteria, including many that live in the gut and skin, which often sneak into deep tissue through cuts.

Meropenem (or imipenem)

A carbapenem is one of the strongest wall-busting antibiotics we have. It punches holes in the bacteria's outer shell so they burst. It works against almost all the common bacteria that cause necrotizing fasciitis, including those that live in soil, water, and the gut. Doctors often choose a carbapenem if the infection is very severe or if earlier antibiotics are not working.

Vancomycin

Vancomycin grabs onto the building blocks of the bacterial wall (peptidoglycan pieces) before they can lock together, so the wall stays weak and full of holes. It is the go-to drug for MRSA (methicillin-resistant *Staph aureus*) and other tough gram-positive bacteria that other antibiotics cannot kill. In necrotizing fasciitis, MRSA can be the main bug or sneak in alongside other bacteria, so vancomycin is added right away.

Clindamycin

Clindamycin stops bacteria from making the proteins they need to survive and, crucially, to make toxins. Group A Strep and *Clostridium* species pump out powerful poisons (exotoxins) that destroy tissue and trigger toxic shock. By jamming the bacteria's protein factory, clindamycin shuts down toxin production fast, even while other antibiotics are killing the bugs. It also helps antibiotics work better in dense, low-oxygen infected tissue where bacteria are packed tight.

Daptomycin

Daptomycin is a backup for MRSA if vancomycin cannot be used (kidney damage, allergy, or resistance). It punches holes directly in the bacteria's inner membrane (the sealed bag that holds all the bacteria's insides), letting critical ions (potassium) leak out. The bacteria loses the electrical charge it needs to make energy, and it dies quickly.

Linezolid

Linezolid is another backup for MRSA if vancomycin or daptomycin cannot be used. It blocks the very first step of protein-making inside the bacteria — it stops the two halves of the ribosome from snapping together, so no proteins can be built at all. Without proteins, the bacteria cannot make toxins, repair damage, or grow. It is one of the few anti-MRSA drugs that can be given by mouth, but in necrotizing fasciitis it is almost always given IV at first.

Intravenous immunoglobulin (IVIG)

IVIG is a concentrate of antibodies taken from the blood of thousands of healthy donors. When group A Strep causes necrotizing fasciitis, it can release super-toxins (like streptococcal

pyrogenic exotoxins) that overstimulate the immune system and trigger toxic shock syndrome (dangerously low blood pressure, organ failure, confusion). IVIG floods the bloodstream with ready-made antibodies that bind to and neutralize those toxins, calming the immune storm and buying time for antibiotics and surgery to work.

Hyperbaric oxygen therapy (HBOT)

Hyperbaric oxygen puts the patient in a sealed chamber and raises the air pressure to 2–3 times normal while they breathe 100% oxygen. This drives huge amounts of oxygen into the blood and deep into dying tissue. Clostridium bacteria (the cause of gas gangrene) cannot survive in high-oxygen environments and die. High oxygen also helps white blood cells kill bacteria better, speeds wound healing, and can slow the spread of toxins. HBOT is used as a helper treatment — it never replaces surgery or antibiotics.

Where this information comes from

- IDSA Skin and Soft Tissue Infection Guideline 2014
- World Society of Emergency Surgery (WSES) Necrotizing Soft Tissue Infections Consensus 2018
- CDC Necrotizing Fasciitis Information
- Cochrane Review of Necrotizing Soft Tissue Infections

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