

Necrotizing Fasciitis

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THE STORY IN ONE SENTENCE

Flesh-eating bacteria race along the layers between muscles, destroying tissue faster than the immune system can reach them.

“Necrotizing fasciitis is a fast-spreading infection where bacteria eat through the fascia (the wrapping between muscle groups), making toxins that kill tissue, and only emergency surgery to cut out all the dead tissue can stop it.”

Normal anatomy — the body's job

Fascia is a thin, tough sheet of connective tissue that wraps around and separates muscle groups all over the body, like layers of plastic wrap between packages of meat. It holds muscles in place and lets them slide smoothly past each other when you move.

BODY'S JOB

Fascia has very few blood vessels running through it, which is fine when it's healthy — it just needs to be strong and smooth. But that low blood supply becomes a huge problem if bacteria get in, because your white blood cells (the infection fighters in your blood) and antibiotics have a hard time reaching fascia. That makes fascia a perfect hiding spot for bacteria to multiply fast without much resistance.

1. Picture your muscles as bundles wrapped in thin, slippery sheets — that's fascia.
2. These sheets stack and slide, letting you bend, twist, and move without muscles sticking together.
3. Because fascia doesn't need much energy or oxygen for its job, it has only a trickle of blood flow compared to muscle.
4. That trickle is enough to keep fascia healthy, but it's not enough to deliver a flood of immune cells or medicine when infection strikes.
5. So once bacteria land in fascia, they face almost no resistance and can spread sideways along the tissue plane very quickly, sometimes inches per hour.

What breaks

Bacteria break through the skin and invade the fascia, then multiply and make poisons that destroy tissue faster than the body can fight back.

CLINICAL TERM

The most common culprit is group A *Streptococcus* (also called *Strep pyogenes*, the same bug that causes strep throat), but other bacteria can do it too: *Clostridium* species (which make gas and thrive without oxygen), *Vibrio vulnificus* (from seawater or raw shellfish), or a mix of different bacteria working together (polymicrobial infection, often with gut bacteria like *E. coli* and *Bacteroides* after abdominal surgery or in people with diabetes). The bacteria enter through a break in the skin — a cut, scrape, puncture wound, insect bite, injection site, surgical incision, or sometimes a tiny invisible crack. Once in the fascia, they release toxins (such as exotoxins from *Strep* that act like superantigens, overloading the immune system, or enzymes that dissolve collagen and tissue). Some bacteria also produce gas as they digest tissue. The toxins kill fascia, fat, and muscle, and the dead tissue (necrosis) spreads along the fascial plane. Because fascia has so little blood flow, white blood cells and antibiotics arrive late and in small numbers, so the infection races ahead. The toxins spill into the bloodstream, triggering a massive inflammatory response (systemic inflammatory response syndrome, or SIRS) that leads to septic shock: blood vessels leak, blood pressure drops, and organs fail from low oxygen and toxic overload.

How the body compensates

At first, the body tries to fight the infection by ramping up heart rate, breathing, and immune signals, so vital signs might look almost normal or only a little off. This hides how bad things really are. By the time blood pressure crashes and organs fail, the window to save tissue and life is closing fast. The key is to catch the infection in that dangerous middle zone — when pain is severe, skin looks angry, and labs show inflammation — before full shock hits.

1. The body floods the infected area with inflammatory signals (cytokines like TNF and interleukins) to try to wall off the bacteria and call in immune cells.
2. The heart beats faster (tachycardia) to pump more blood and deliver oxygen and white blood cells to the tissues.
3. Breathing speeds up (tachypnea) to bring in more oxygen and blow off carbon dioxide as the body's metabolism revs up fighting infection.
4. Blood vessels in the skin and non-essential areas squeeze down (vasoconstriction) to keep blood pressure up and protect the brain and heart.

5. The liver makes acute-phase proteins, and the bone marrow pours out more white blood cells, so labs show high or very low white count, high C-reactive protein (CRP), and high procalcitonin.
6. For a while — hours to a day or so — blood pressure and oxygen levels may stay in a range that looks 'okay,' masking the disaster brewing underneath.
7. Once the toxins overwhelm the system, blood vessels start to leak (capillary leak), blood pressure drops despite the fast heart rate, and organs (kidneys, lungs, liver, brain) don't get enough oxygen, leading to multi-organ failure.
8. At that point, even aggressive treatment may not reverse the damage, so the goal is to recognize and treat the infection in the compensation phase — when the body is working hard but still holding on — before the crash into irreversible shock.

Domino effect

1. Bacteria enter through a break in the skin and reach the fascia. — Any cut, puncture, or surgical wound can let bacteria slip past the skin barrier; sometimes the entry point is so small it's never found, but the bacteria find their way into the poorly defended fascia.
2. Bacteria multiply in fascia and release toxins that destroy tissue. — Fascia has very little blood flow, so immune cells and antibiotics arrive slowly; meanwhile, the bacteria make enzymes (like hyaluronidase and collagenase) that dissolve the 'glue' holding tissue together, and toxins (like streptococcal pyrogenic exotoxins) that kill cells directly, causing tissue death (necrosis).
3. Pain becomes severe, far worse than the appearance of the skin would suggest. — The toxins and dying tissue release inflammatory chemicals (like bradykinin and prostaglandins) that fire pain nerves intensely, and the infection is destroying deep layers (fascia and muscle) even when the skin on top looks only red or slightly swollen.
4. Skin changes appear: dark red or purple color, blisters filled with blood or dark fluid (hemorrhagic bullae), and sometimes a crackling feeling under the skin (crepitus from gas). — As fascia dies, blood vessels in and above it are destroyed, so blood leaks into blisters and the skin turns dusky from poor blood flow; gas-producing bacteria (like *Clostridium*) release bubbles that crackle when you press the skin.
5. The infected area may go numb. — Toxins and lack of blood kill the small nerves running through the fascia and skin, so even though the infection is raging, the area loses feeling — this is a very bad sign that tissue is dying.
6. Fever, fast heart rate, fast breathing, confusion, and feeling terribly ill (toxicity) set in. — Bacterial toxins and chunks of dead tissue (endotoxins and cell debris) spill into the bloodstream, triggering a whole-body inflammatory response (SIRS); the immune system releases cytokines (like TNF-alpha, IL-1, and IL-6) that cause fever, speed up the heart and lungs, and fog the brain.

7. Blood pressure starts to drop (hypotension), and organs begin to fail (septic shock and multi-organ dysfunction). — The massive inflammation causes blood vessels to leak fluid into tissues (capillary leak), so blood volume in the vessels falls and pressure drops; at the same time, toxins and low oxygen damage the kidneys (rising creatinine, less urine), lungs (trouble breathing, low oxygen), liver (rising enzymes), and brain (confusion, sleepiness), leading to multi-organ failure.
8. Without emergency surgery to remove all dead tissue (debridement), the infection spreads further, shock worsens, and death is almost certain. — Antibiotics alone cannot reach the dead, bloodless tissue where bacteria are thriving; only cutting out every bit of dead (necrotic) fascia, fat, and muscle stops the source of toxins and gives antibiotics and the immune system a chance to clear what's left — delay of even hours sharply raises the risk of losing limbs, going into irreversible shock, or dying.

Symptoms · what the patient feels, and why

- Severe pain that seems way worse than what you can see on the skin: Bacteria release toxins (poisons) that destroy tissue deep under the skin, killing nerves and muscle. The damage races ahead of the visible redness, so the tissue screams even when the surface looks fairly normal.
- Skin turns dark purple or bronze, sometimes with blood blisters (bullae): The infection cuts off blood flow to the skin by clotting small vessels and filling tissue with toxins. Without oxygen, the skin tissue dies and turns dark; blood leaks into blisters because vessel walls break down.
- A crackling feel or sound (Crepitus) — a crackling or popping feel when you press the skin: Some bacteria (especially *Clostridium*) make gas as they digest tissue. The gas gets trapped under the skin, so when you push on it you feel and hear tiny bubbles popping.
- The painful area feels numb or you cannot feel touch there: Toxins and swelling crush the tiny nerves in the tissue, cutting off their ability to send signals. When nerves die, the skin goes numb even though the deep pain remains.
- Fever, often high (above 101°F or 38.3°C): The hypothalamus (the brain's thermostat) raises body temperature in response to bacterial toxins and immune signals called cytokines. Heat helps fight infection but also means the body is under serious attack.
- Fast heart rate (tachycardia, often above 100 beats per minute): The body releases adrenaline (epinephrine, a stress hormone) to pump more blood and oxygen to tissues. The heart speeds up to try to keep blood pressure from dropping as vessels leak fluid.
- Low blood pressure (hypotension, systolic below 90 mmHg): Bacterial toxins make blood vessels relax and leak fluid into tissues (distributive shock). Less fluid stays in the bloodstream, so pressure falls and organs do not get enough blood.

- Confusion or drowsiness (altered mental status): Low blood pressure and toxins mean the brain does not get enough oxygen and glucose (its fuel). The prefrontal cortex (the brain's thinking center) shuts down first, causing confusion or sleepiness.
- Swelling that spreads quickly, sometimes several inches per hour: Bacteria multiply fast and release enzymes that dissolve the tissue barriers (fascia) between muscle groups. Toxins and fluid pour into new areas, pushing the infection outward at a racing pace.

Focused exam

- Pain way out of proportion to what you see on the skin: Bacteria are destroying tissue deep under the skin, along the fascia (the thin, strong layer that wraps your muscles like plastic wrap). The nerves buried in that layer are dying, and they scream. The skin may look almost normal, but the pain is huge. This mismatch is the single biggest warning sign that necrotizing fasciitis is happening.
- Skin that looks dark purple, blue, or black in patches: The bacteria and the poisons they make are killing the tiny blood vessels that feed the skin. When tissue dies, it turns dark. Seeing dark patches means necrotizing fasciitis has already been spreading for hours.
- Bullae (large blisters filled with blood or dark fluid): When the fascia dies, the skin above it loses its blood supply and blisters form. Blood inside the blisters means the tissue is truly dying, not just infected on the surface.
- A crackling feel or sound (Crepitus) (a crackling or popping feeling when you press on the skin): Some bacteria (like Clostridium) make gas as they grow and destroy tissue. You feel the gas bubbles under the skin, like pressing on bubble wrap. A crackling feel or sound (Crepitus) is a sign that gas-making bacteria are at work in necrotizing fasciitis.
- Numbness or loss of feeling in the skin over the area: The bacteria spreading along the fascia kill the nerves that carry feeling. When the skin goes numb, it means necrotizing fasciitis has destroyed the nerve layer.
- Fever, fast heart rate (over 110 beats per minute), low blood pressure (under 90 systolic), and confusion: The body is losing the fight. Bacterial toxins are flooding the bloodstream, and organs (brain, heart, kidneys) are shutting down from lack of oxygen and blood flow. This is septic shock caused by necrotizing fasciitis spreading poison everywhere.

Diagnostics · every test answers a question

- Lactate (serum lactate level) → Is the body's tissue getting enough oxygen, or is shock starting?
- Complete blood count (CBC) with white blood cell count and differential → Is the body mounting a big immune response to infection?

- Basic metabolic panel (BMP) with creatinine and sodium → Are the kidneys still working, and is the body losing salt and water from shock?
- C-reactive protein (CRP) → How much inflammation is happening in the body right now?
- Creatine kinase (CK) → Is muscle being destroyed along with the fascia?
- CT scan with IV contrast of the affected area → Is there gas, fluid, or swelling along the fascial planes that confirms necrotizing fasciitis?
- Blood cultures (two sets from different sites) → What bacteria are in the bloodstream, and are they causing sepsis?

Treatment logic

- Emergency surgical debridement (cutting out all dead and dying fascia, fat, and muscle) → Removes the rotting tissue that is making toxins and feeding bacteria, so the infection stops spreading and poisoning the whole body. Surgery is the only thing that actually stops necrotizing fasciitis from killing more tissue.
- Broad-spectrum IV antibiotics: piperacillin-tazobactam 3.375–4.5 g every 6 hours OR a carbapenem (meropenem 1 g every 8 hours or imipenem 500 mg every 6 hours) → Kills the mix of bacteria (Group A Streptococcus, Clostridium, gut bacteria like E. coli, Klebsiella, and Bacteroides) that cause necrotizing fasciitis before they can make more toxins. These antibiotics cover both oxygen-loving and oxygen-hating bacteria.
- Vancomycin IV 15–20 mg/kg every 8–12 hours (dose adjusted to keep trough level 15–20 mcg/mL) → Kills Group A Streptococcus and MRSA (methicillin-resistant Staph aureus) that the other antibiotics might miss, especially in areas where resistant bacteria are common or in patients who had recent healthcare contact.
- Clindamycin IV 600–900 mg every 8 hours → Shuts down the bacterial ribosome (the tiny factory inside bacteria that makes proteins), so Group A Streptococcus stops making the toxins (like streptococcal pyrogenic exotoxins) that destroy tissue. This happens even while the bacteria are still alive and dying from other antibiotics.
- Aggressive IV fluid resuscitation (30 mL/kg crystalloid bolus within the first 3 hours) and vasopressors (norepinephrine) if blood pressure stays low → Fills up the blood vessels so blood can reach the organs (brain, kidneys, heart) and keeps blood pressure high enough to push oxygen into tissue. This buys time for antibiotics and surgery to work before organs shut down from septic shock.
- Repeat surgical debridement every 12–24 hours until no more dead or dying tissue is seen → Finds and removes bacteria and toxins that were hiding deeper in the tissue planes during the first surgery. Necrotizing fasciitis spreads so fast that a single surgery almost never gets all the infection. Each trip to the operating room removes more fuel from the fire.

- Intravenous immunoglobulin (IVIG) 1–2 g/kg as a single dose (in severe Group A *Streptococcus necrotizing fasciitis* with toxic shock) → Gives the patient donated antibodies from thousands of healthy people. These antibodies bind to and neutralize the toxins made by Group A *Streptococcus*, especially the superantigens that overactivate the immune system and cause shock.

Medications

Piperacillin-tazobactam · Beta-lactam antibiotic + beta-lactamase inhibitor

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.

1. Piperacillin sticks to proteins (penicillin-binding proteins) on the inside of the bacterial wall that glue new wall pieces together.
2. Without those proteins working, the wall gets weak and full of gaps.
3. Water rushes into the bacteria, swells it up, and bursts it open.
4. Tazobactam locks onto beta-lactamase enzymes and stops them from snipping the piperacillin molecule, so the antibiotic stays active longer.

SAFETY WATCH

Watch for allergic reactions, especially hives, throat swelling, or trouble breathing — about 1 in 10 people allergic to penicillin will also react to piperacillin. · Can cause diarrhea, sometimes severe (*C. diff* colitis), because it kills good gut bacteria too. · Check kidney function daily in the hospital — dose must be lowered if kidneys are struggling. · Rarely lowers blood counts (white cells, platelets); labs are checked every few days.

Meropenem (or imipenem) · Carbapenem antibiotic

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.

1. Meropenem attaches to penicillin-binding proteins inside the bacterial wall, blocking the enzymes that stitch new wall pieces together.
2. The wall becomes riddled with weak spots and holes.
3. Pressure inside the bacteria (from water rushing in) breaks the weak wall, and the bacteria explodes and dies.

4. Carbapenems resist most bacterial scissors (beta-lactamases), so they keep working even when other antibiotics fail.

SAFETY WATCH

Can trigger seizures, especially in people with kidney problems or a history of epilepsy — dose is adjusted for kidney function. · Watch for severe allergic reactions (rash, swelling, wheezing); cross-allergy with penicillin is possible but less common than with other penicillins. · Can cause diarrhea and C. diff colitis by wiping out healthy gut bacteria. · Liver enzyme levels may rise; checked with routine blood work.

Vancomycin · Glycopeptide antibiotic

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.

1. Vancomycin binds tightly to the ends of peptidoglycan chains (the bricks of the bacterial wall) that are waiting to be glued together.
2. By blocking the glue site, it stops the wall from being built and repaired.
3. The weak wall cannot hold the bacteria's shape under pressure, so the bacteria leaks and dies.
4. Vancomycin is too large to cross the outer membrane of gram-negative bacteria, so it does not work against those — that is why a second antibiotic is always needed.

SAFETY WATCH

Can damage the kidneys, especially if blood levels get too high or if combined with other kidney-toxic drugs — blood levels (trough) are checked every few days and the dose is adjusted. · Can harm hearing and balance (ototoxicity) if levels stay high for too long — ask about ringing in the ears or dizziness. · Rapid IV push can cause 'red man syndrome' — flushing, itching, and a red rash on the face, neck, and chest. Slowing the infusion to at least 1 hour prevents this. · Rarely causes severe allergic reactions (rash, fever, low blood counts).

Clindamycin · Lincosamide antibiotic (protein synthesis inhibitor)

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.

1. Clindamycin slips into the bacteria and attaches to the 50S subunit of the ribosome (the part that strings amino acids together into proteins).
2. By blocking that site, it stops the ribosome from linking amino acids, so new proteins cannot be made.
3. Without proteins, the bacteria cannot repair itself, grow, or churn out toxins.
4. Toxin levels in the blood drop within hours, which helps stop the runaway inflammation and tissue death that toxins cause.

SAFETY WATCH

High risk of *C. difficile* colitis (severe, sometimes bloody diarrhea caused by overgrowth of bad bacteria in the colon) — tell the team right away if diarrhea starts, even weeks after treatment ends. · Can cause nausea and a bad taste in the mouth. · Rarely triggers severe allergic skin reactions (Stevens-Johnson syndrome) — watch for blistering rash or peeling skin. · May worsen myasthenia gravis (a muscle weakness disease) by blocking signals at the muscle.

Daptomycin · Lipopeptide antibiotic

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.

1. Daptomycin binds to calcium in the blood, then inserts itself into the bacteria's membrane.
2. It forms little holes (pores) that let potassium ions (K⁺) rush out of the bacteria.
3. Loss of potassium collapses the electrical gradient the bacteria needs to make ATP (its energy currency).
4. Without energy, the bacteria cannot repair, grow, or survive — it dies within minutes.

SAFETY WATCH

Can damage muscles (rhabdomyolysis) — check CPK (creatine phosphokinase) blood levels twice a week and stop the drug if muscle pain, weakness, or dark urine appears. · Can harm the kidneys, especially if combined with other kidney-toxic drugs — kidney function is monitored daily in the hospital. · Rarely causes lung inflammation (eosinophilic pneumonia) — watch for new cough, shortness of breath, or fever.

Linezolid · Oxazolidinone antibiotic (protein synthesis inhibitor)

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.

1. Linezolid binds to the 23S part of the 50S ribosomal subunit (the larger half of the protein-making machine).
2. By sitting on that spot, it prevents the 30S and 50S subunits from linking up to start building a protein chain.
3. No initiation means no protein production, which shuts down toxin release and bacterial growth.
4. The bacteria starves and dies.

SAFETY WATCH

Can drop blood counts (especially platelets and white cells) if used longer than 2 weeks — complete blood count is checked twice a week. · Can harm the optic nerve and cause vision loss if used for many weeks — report any vision changes right away. · Can trigger serotonin syndrome (confusion, fast heartbeat, sweating, shaking) if combined with certain antidepressants (SSRIs, MAOIs) — tell the team about all medications. · Can cause severe lactic acidosis (acid from oxygen-starved tissues) (acid buildup in the blood) with long-term use — watch for deep, fast breathing, nausea, and muscle weakness.

Intravenous immunoglobulin (IVIG) · Pooled human antibodies (immunoglobulin)

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.

1. IVIG antibodies (IgG) recognize and stick to streptococcal toxins floating in the blood and tissue.
2. By binding the toxins, they block the toxins from attaching to immune cells (T cells, macrophages) and triggering a massive cytokine release (the 'cytokine storm').
3. The immune system calms down, blood pressure stabilizes, and organ damage slows.

4. IVIG also delivers antibodies that directly tag bacteria for destruction by the patient's own white blood cells.

SAFETY WATCH

Can cause infusion reactions (fever, chills, headache, nausea) — slow the infusion rate if these happen. · Can trigger kidney damage, especially in people who are dehydrated or already have kidney disease — IV fluids are given before and during the infusion. · Rarely causes blood clots (stroke, heart attack, a clot in a deep leg vein (deep vein thrombosis)) — higher risk in older adults and people with clotting disorders. · Can cause aseptic meningitis (headache, stiff neck, fever without infection) — usually resolves on its own after the infusion ends. · Very expensive and not always immediately available — used only in confirmed or highly suspected streptococcal toxic shock syndrome.

Hyperbaric oxygen therapy (HBOT) · Adjunctive therapy (not a drug — high-pressure oxygen delivered in a sealed chamber)

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.

1. The patient breathes 100% oxygen at 2–3 atmospheres of pressure (like being 33–66 feet underwater).
2. Oxygen dissolves directly into the blood plasma (not just carried by red blood cells), raising tissue oxygen levels 10–15 times higher than normal.
3. High oxygen poisons Clostridium and other anaerobic bacteria, which cannot handle oxygen and die.
4. Oxygen-starved white blood cells (neutrophils) regain their power to kill bacteria.
5. High oxygen also shrinks swelling, helps new blood vessels grow, and supports tissue repair.

SAFETY WATCH

Rarely available quickly in most hospitals — if the patient must be transferred far away for HBOT, it can delay life-saving surgery. · Can rupture the eardrum or sinuses if pressure is raised too fast — equalization techniques are taught before treatment. · Can cause oxygen toxicity (seizures, vision changes) if pressure is too high or treatment lasts too long — sessions are limited to 90–120 minutes. · Cannot be used if the patient has an untreated pneumothorax (collapsed lung) — the pressure change can make it worse. · Claustrophobia can make it hard to tolerate the small chamber.

Red flags

- 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

Nursing priorities

1. Alert the surgeon and operating room immediately and prepare the patient for emergency surgery within one hour. — Every hour of delay increases tissue death and risk of death because bacteria spread along the fascia at a rate of 2 to 3 centimeters per hour, so surgical debridement (cutting out dead tissue) is the only way to stop the infection from spreading.
2. 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. — Septic shock causes vasodilation (blood vessels open too wide) and capillary leak (fluid leaks out of blood vessels), so large volumes of IV fluid are needed to keep blood pressure up and organs perfused with oxygen.

3. 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. — Antibiotics kill bacteria in the bloodstream and slow spread, but they cannot reach dead tissue, so they must be started right away while the surgeon prepares; clindamycin also stops toxin production by bacteria, which reduces tissue damage.
4. 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. — Septic shock causes blood vessels to lose tone and blood pressure to drop dangerously low, and vasopressors squeeze blood vessels to keep blood pressure high enough to perfuse the brain, heart, and kidneys.
5. Measure urine output every hour with a Foley catheter and monitor for at least 0.5 milliliters per kilogram per hour. — Low urine output is an early sign that the kidneys are not getting enough blood flow from septic shock, and acute kidney injury (sudden kidney failure) is a common complication of necrotizing fasciitis.
6. Check lactate level, complete blood count, creatinine, and calculate the LRINEC score using lab values to assess severity of infection. — Lactate above 2 millimoles per liter means tissues are not getting enough oxygen; the LRINEC score (Laboratory Risk Indicator for Necrotizing Fasciitis) uses white blood cell count, hemoglobin, sodium, glucose, creatinine, and C-reactive protein to predict risk, with a score of 6 or more indicating high risk.
7. 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). — Early necrotizing fasciitis can look like minor cellulitis on the outside, but severe pain with minimal skin findings is the key warning sign that helps nurses and doctors catch the infection before tissue turns black and dies.
8. Prepare the patient and family for the possibility of amputation or disfiguring surgery and arrange ICU bed for postoperative care. — Surgical debridement often requires removal of large amounts of tissue, and sometimes a limb must be amputated to save the patient's life; the mortality rate is 20 to 30 percent even with treatment, and patients need ventilator support and close hemodynamic monitoring after surgery.

Sources

- 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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