

# Necrotizing Soft Tissue Infection

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

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## THE STORY IN ONE SENTENCE

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

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

## Normal anatomy — the body's job

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The fascia is a thin, tough sheet of tissue that wraps around every muscle, organ, and bundle of blood vessels and nerves under your skin, keeping everything in separate compartments and protecting muscle from germs and injury.

## BODY'S JOB

The fascia's job is to hold muscles in place, guide the direction they pull, let them slide smoothly past each other without tangling, and act as the first barrier that keeps bacteria on the skin from reaching deeper tissue. Think of it like the cling-wrap that keeps food fresh and separate in the fridge — it divides things neatly and keeps the bad stuff out.

1. Skin is the outermost layer, like the peel of an apple.
2. Just under the skin sits a layer of fat for padding and insulation.
3. Below the fat lies the fascia, a slippery white sheet of connective tissue that looks like thin plastic wrap stretched tight over the muscles.
4. The fascia divides the body into compartments — each muscle group gets its own sealed space with its own blood supply and nerves running through it.
5. Because the fascia is one connected sheet that runs along the whole limb or body wall, an infection can slide along it like water running down a hallway, spreading far from the original cut in just hours.

## What breaks

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Bacteria that make powerful flesh-destroying toxins get through a break in the skin — even a tiny scratch, insect bite, injection site, or blister — and land in the fascia layer, where they multiply explosively and release poisons that melt tissue faster than the body can wall them off or kill them.

### CLINICAL TERM

Necrotizing soft tissue infection (NSTI), also called necrotizing fasciitis when the fascia is the main target. The break happens when virulent bacteria — most commonly group A *Streptococcus pyogenes* (GAS), *Clostridium perfringens* or other *Clostridium* species (causing gas gangrene), or polymicrobial mixtures including anaerobes, *Enterobacteriaceae*, and sometimes *Vibrio vulnificus* from seawater — enter through even minor trauma and secrete exotoxins (like streptococcal pyrogenic exotoxins, streptolysin, and clostridial alpha toxin) that destroy the fascia, subcutaneous fat, and muscle by breaking down collagen, triglycerides, and cell membranes, triggering massive local tissue death (necrosis) and systemic toxin-mediated shock.

## How the body compensates

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The body tries to fight the infection by sending white blood cells, forming blood clots to wall off the bacteria, and ramping up the whole immune system — but because the bacteria destroy tissue faster than the immune system can contain them, and because the fascia has poor blood flow, these defenses fail and the person spirals into shock. Vital signs may look almost normal for the first few hours, which is the most dangerous window, because the hidden destruction is already racing ahead.

1. The immune system detects the bacteria and floods the area with white blood cells and inflammatory signals, causing swelling, redness, and intense pain.
2. Blood vessels in the infected zone start to clot (thrombose) in an attempt to trap the bacteria and stop them from spreading into the bloodstream — but this also cuts off oxygen to healthy tissue, making the tissue die even faster.
3. The liver and bone marrow pump out more white blood cells, and the heart beats faster to deliver them — so early labs may show a very high white blood cell count and the heart rate may be 110 or 120 even while blood pressure is still normal.
4. The adrenal glands release cortisol and adrenaline (epinephrine) to keep blood pressure up and keep the person awake and alert — this is why in the first few hours the patient may look 'not that sick' even though the infection is already deep and spreading.

5. As the toxins break into the bloodstream, the blood vessels all over the body start to leak fluid, blood pressure drops, and the kidneys and lungs begin to fail — this is septic shock, and it is the crash point.
6. Once septic shock hits, the window to save the person's life shrinks fast — they need emergency surgery to cut out all the dead tissue (debridement) plus IV antibiotics and intensive care support for blood pressure, breathing, and organ function.
7. Even with perfect care, mortality is 25 to 35 percent, and it doubles if surgery is delayed more than 12 hours from symptom onset, so the key is recognizing the infection in that early 'quiet' window before the crash.

## Domino effect

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1. Bacteria enter through a small break in the skin — a cut, scrape, bug bite, blister, or injection site. — 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. — 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. — 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. — 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. — 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. — 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. — 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. — 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.

## Symptoms · what the patient feels, and why

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- 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.
- Fast heartbeat and breathing faster than normal: The infection triggers a whole-body alarm reaction called sepsis. The heart pumps faster and breathing speeds up to try to deliver more oxygen and fight the infection, because toxins in the blood lower blood pressure and oxygen levels.

- Swelling that spreads quickly, sometimes an inch or more every hour: The bacteria multiply very fast and release enzymes (chemicals that break down tissue) that destroy the walls holding tissue layers together, so the infection spreads along those layers like fire racing through a building.

## Focused exam

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- Pain way out of proportion to what the skin looks like: In necrotizing soft tissue infection, the bacteria destroy the fascia (the thin tough sheet under the skin that wraps muscles) much faster than the skin dies. The nerves in that fascia scream before the skin changes color. When pain is worse than redness, think necrotizing infection—this finding alone should trigger the alarm.
- Skin that looks dark purple, blue, or black in streaks or patches: Dead tissue turns dark. In necrotizing soft tissue infection, the fascia dies first, then the skin above it dies from losing its blood supply. The color tells you how far the death has spread—purple or black patches mean the tissue is already gone.
- Blisters filled with dark or bloody fluid (hemorrhagic bullae): These are not regular burn blisters. In necrotizing soft tissue infection, bacterial toxins break down the skin layers and fluid leaks out. The fluid is dark or bloody because the tissue underneath is dying and small blood vessels are breaking apart.
- Crackling or a crackling feel or sound (crepitus) when you press on the skin: This crackle means gas is trapped in the tissue spaces. Some bacteria that cause necrotizing infection—like *Clostridium*—make gas as they eat the fascia. You hear the crackle because air pockets form where healthy tissue used to be. A crackling feel or sound (Crepitus) is a late finding and means the infection is advanced.
- Swelling that spreads beyond the visible skin damage, crossing joints: In necrotizing soft tissue infection, the inflammation follows the fascia layers like highways. The swelling crosses joints and spreads faster than regular skin infections because it travels along the tissue planes under the skin, not just in the skin itself.
- Signs of septic shock: rapid heart rate (tachycardia), low blood pressure (hypotension), fever or being cold and clammy: Necrotizing soft tissue infection releases bacterial toxins (like streptococcal pyrogenic exotoxins) into the bloodstream. The body goes into shock trying to fight back—blood vessels leak, blood pressure drops, and organs begin to fail. This is the danger moment: septic shock and organ failure are coming unless surgery happens immediately.

## Diagnostics · every test answers a question

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- Serum lactate → Is the tissue getting enough oxygen, or is the body slipping into shock?

- Complete blood count (CBC) with differential → Is the immune system fighting hard, and are there signs of severe infection?
- Creatine kinase (CK) → Is muscle tissue being destroyed?
- Basic metabolic panel (BMP) with creatinine → Are the kidneys still working, and is the body in metabolic trouble?
- C-reactive protein (CRP) → How much inflammation is happening in the body?
- Blood glucose → Is the stress response pushing sugar dangerously high?
- Blood cultures (two sets, drawn before antibiotics) → Has the infection spread into the bloodstream, and which bacteria are causing it?
- Imaging: X-ray, CT, or MRI of the affected area → Is there gas in the tissue, or can I see fluid and swelling tracking along the fascia?

## Treatment logic

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- 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.
- 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.
- 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.
- 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.
- 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.

- Repeat surgical debridement every 24–48 hours until all dead tissue is gone → Removes any new dead tissue that was not clear at the first surgery. The infection boundary keeps moving inward, so the surgeon must go back and 'chase' the infection until only healthy, bleeding tissue remains.
- ICU-level supportive care: mechanical ventilation, dialysis, vasopressor support → Keeps vital organs (lungs, kidneys, heart) working while the body fights the infection. Mechanical ventilation takes over breathing if septic shock weakens the lungs (ARDS). Dialysis filters the blood if the kidneys fail from low blood pressure and toxins.

## Medications

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### **Piperacillin-tazobactam · Beta-lactam antibiotic (penicillin) plus beta-lactamase inhibitor**

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.

1. Piperacillin binds to proteins (penicillin-binding proteins) on the inside of bacterial cell walls.
2. This stops the bacteria from building and repairing their protective outer shell.
3. Without a strong wall, water rushes into the bacteria and they pop like overfilled water balloons.
4. Tazobactam blocks beta-lactamase, an enzyme some bacteria make to break down piperacillin, so the antibiotic stays active longer.

#### **SAFETY WATCH**

Can cause allergic reactions, especially in people allergic to penicillin (hives, swelling, rarely a life-threatening reaction called anaphylaxis). · May trigger diarrhea or a serious gut infection called C. diff colitis if it kills off healthy gut bacteria. · High doses can rarely cause seizures, especially in people with kidney problems or brain disease. · Can lower platelet count (cells that stop bleeding) and raise the risk of bruising or bleeding.

### **Vancomycin · Glycopeptide antibiotic**

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.

1. Vancomycin binds to the building blocks (D-alanyl-D-alanine) at the ends of cell-wall chains.
2. This stops enzymes from cross-linking the chains into a strong mesh.
3. The bacterial wall becomes weak and leaky.
4. Pressure inside the bacteria causes them to burst and die.

#### **SAFETY WATCH**

Can damage the kidneys, especially at high doses or when combined with other kidney-toxic drugs; doctors check kidney function and vancomycin blood levels regularly. · Can damage hearing (ototoxicity), causing ringing in the ears or permanent hearing loss, especially with high doses or long use. · Rapid infusion can cause 'red man syndrome' (flushing, itching, red rash on the face, neck, and chest) because it triggers histamine release; slow the infusion to prevent this. · Rarely causes severe allergic reactions or a drop in white blood cells.

### **Clindamycin · Lincosamide antibiotic**

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.

1. Clindamycin enters bacterial cells and binds to the 50S subunit of the ribosome (the machine that reads genetic instructions and builds proteins).
2. This stops the ribosome from linking amino acids together into proteins.
3. Without proteins, bacteria cannot repair themselves, make toxins, or reproduce.
4. In group A Strep and Clostridium, blocking protein production stops the release of deadly toxins (like streptococcal pyrogenic exotoxins and clostridial alpha toxin) that destroy tissue and cause shock.

#### **SAFETY WATCH**

Can cause severe diarrhea and C. diff colitis (a dangerous infection of the colon caused by killing healthy gut bacteria); stop the drug and call a doctor if diarrhea is watery, bloody, or comes with fever and belly pain. · Rarely causes allergic reactions, including rash and, very rarely, a severe skin reaction (Stevens-Johnson syndrome). · May rarely cause liver enzyme elevations or low white blood cell counts.

## **Intravenous immunoglobulin (IVIG) · Immune globulin (pooled human antibodies)**

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.

1. IVIG delivers millions of antibodies that directly bind to and neutralize bacterial superantigens (toxins that overstimulate the immune system).
2. By neutralizing these toxins, IVIG prevents them from triggering massive T-cell activation and cytokine release (the 'cytokine storm' that causes shock).
3. IVIG also blocks Fc receptors on immune cells, dampening the body's runaway inflammatory response.
4. Together, these actions reduce tissue damage, stabilize blood pressure, and improve survival in toxic shock.

### **SAFETY WATCH**

Can cause infusion reactions (fever, chills, headache, nausea, muscle aches) during or shortly after the infusion; slowing the rate usually helps. · Rarely causes serious allergic reactions, especially in people with IgA deficiency. · Can rarely cause blood clots (deep vein clot formation (thrombosis), stroke, heart attack), kidney damage, or aseptic meningitis (inflammation of the brain lining without infection). · Very expensive and not always available; reserved for the sickest patients with toxic shock.

## **Intravenous fluids (crystalloids: normal saline or lactated Ringer's) · Intravenous fluid resuscitation**

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.

1. Sterile salt water (normal saline) or balanced salt solution (lactated Ringer's) is infused rapidly through a large IV line.
2. The fluid expands blood volume, raising the pressure inside blood vessels.
3. Higher pressure pushes blood forward, delivering oxygen and nutrients to organs and tissues.

4. This prevents or reverses shock and buys time while surgery removes dead tissue and antibiotics kill bacteria.

**SAFETY WATCH**

Giving too much fluid too fast can overload the heart and lungs, causing fluid to back up into the lungs (pulmonary swelling (edema)) and making breathing harder. · Can worsen tissue swelling in the arms, legs, or belly, sometimes requiring surgical release of pressure (fasciotomy). · Doctors monitor urine output, blood pressure, and oxygen levels closely to balance fluid needs.

**Vasopressors (norepinephrine) · Sympathomimetic vasopressor (alpha- and beta-adrenergic agonist)**

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.

1. Norepinephrine binds to alpha-1 receptors on blood vessel walls.
2. This triggers the muscle cells to contract, narrowing the vessels (vasoconstriction).
3. Narrower vessels raise blood pressure.
4. Norepinephrine also binds to beta-1 receptors in the heart, making it pump harder and faster, which further supports blood pressure and organ blood flow (perfusion).

**SAFETY WATCH**

Must be given through a central IV line (a catheter in a large vein near the heart); if it leaks into the tissue, it can cause severe tissue death and skin breakdown. · Can cause dangerously high blood pressure, irregular heart rhythms, or reduced blood flow to fingers, toes, or gut if the dose is too high. · Requires continuous blood pressure monitoring in an intensive care unit. · Weaning off too quickly can cause blood pressure to crash again.

**Hyperbaric oxygen therapy (HBOT) · Adjunctive therapy (not a medication; pure oxygen delivered under high pressure)**

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.

1. The patient breathes 100% oxygen inside a chamber pressurized to 2–3 times normal air pressure.
2. The high pressure forces oxygen to dissolve directly into the blood plasma, raising tissue oxygen levels far above normal.
3. High oxygen levels kill or slow the growth of anaerobic bacteria (like *Clostridium perfringens*) that produce gas and toxins.
4. Extra oxygen also enhances white blood cell killing of bacteria, reduces tissue swelling, and promotes blood vessel growth and wound healing.

#### **SAFETY WATCH**

Can cause ear pain or sinus pain from pressure changes (like diving too deep underwater); patients must equalize pressure by swallowing or yawning. · Rarely causes oxygen toxicity (seizures or lung damage) if pressure or duration is too high. · Can cause temporary worsening of vision (changes in the lens of the eye). · Delays surgery if the chamber is far away; surgery and antibiotics must come first. · Not available in all hospitals; transfer to a hyperbaric center may be needed.

## **Red flags**

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- 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

## **Nursing priorities**

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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 — 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 — 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 — 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
4. Notify the surgeon immediately for bedside evaluation and do not wait for lab results or imaging to call — Surgical debridement (cutting out all dead tissue) within 6 hours of symptom onset cuts death rate in half; imaging delays surgery and necrotizing infection is a clinical diagnosis based on exam findings; every hour without surgery allows more tissue death and toxin release into bloodstream
5. 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 — Elevated or rising lactate means tissues are starving for oxygen because septic shock has lowered blood pressure; this signals need for ICU transfer, central line placement, and vasopressor medications like norepinephrine to squeeze blood vessels and raise pressure
6. Monitor temperature, heart rate, respiratory rate, and oxygen saturation every 15 minutes; watch for fever above 101 or temperature below 96 degrees — High fever means bacterial toxins are triggering massive inflammation; low temperature (hypothermia) means the body can no longer regulate itself and septic shock is overwhelming defenses; both signal worsening sepsis and need for immediate provider notification
7. Measure urine output every hour with a Foley catheter and alert provider if output drops below 30 milliliters per hour — Kidneys need strong blood pressure to filter waste; low urine output means septic shock has cut blood flow to kidneys and acute kidney injury is starting; this guides fluid resuscitation and may require vasopressors to restore kidney blood flow (perfusion) before permanent damage occurs
8. Keep patient NPO (nothing by mouth) and prepare for emergency operating room transport with signed consent and pre-operative checklist completed — Patients need general anesthesia for surgical debridement; food in the stomach increases aspiration risk (breathing vomit into lungs) during intubation; having consent and checklist ready prevents delays when the operating room calls because minutes matter for tissue survival

## Sources

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

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