Necrotizing Soft-Tissue Infections: A Case-Based Review

Vidhi Desai, Undergraduate Student, Cumming School of Medicine, University of Calgary, Calgary, Alberta, Canada.
Susan Vokey, MD, FRCSC, Surgical Program Director, Health Sciences North, Sudbury Regional Hospital, Sudbury, Ontario, Canada.
Stephen Vaughan, MD, FRCPC, Clinical Associate Professor, Department of Medicine and Snyder Institute for Chronic Diseases, University of Calgary.
Ranjani Somayaji, MD, MPH, FRCPC, Associate Professor, Department of Medicine and Snyder Institute for Chronic Diseases, University of Calgary.

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GENERAL PURPOSE: To review the assessment and management of necrotizing fasciitis.

TARGET AUDIENCE: This continuing-education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES: After participating in this educational activity, the participant will:

1. Identify the etiologic pathogens for necrotizing fasciitis.
2. Summarize assessment guidelines for patients who present with signs of necrotizing fasciitis.
3. Explain recommended treatment protocols for patients who have necrotizing fasciitis.

ABSTRACT

Necrotizing fasciitis is a rapidly progressive soft-tissue infection with tissue necrosis and a high mortality rate. This case-based review provides an overview of an approach to the diagnosis and management of necrotizing fasciitis for clinicians.

KEYWORDS: infection, management, mortality, necrosis, necrotizing fasciitis, soft-tissue infection

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INTRODUCTION

Necrotizing soft-tissue infections are rare but highly lethal infections. The spectrum of disease encompasses necrotizing forms of cellulitis, myositis, and fasciitis. Also known as "flesh-eating disease," necrotizing fasciitis (NF) describes a clinical syndrome in which there is necrosis of subcutaneous tissue and fascia with relative sparing of the underlying muscle.[1] The incidence of NF increases with age, with NF occurring most often among adults older than 60 years. Men experience 2.5 times greater incidence of NF on average than women across all age groups.[2,3]

PATHOGENESIS

Necrotizing fasciitis is characterized by widespread necrosis of the subcutaneous tissue and fascia. Often, this is caused by bacterial invasion through the epidermis due to trauma (eg, injection, cut, chronic wound/boil) that compromises skin integrity.[4,5] Rarely, NF will develop in a healthy individual after a minor trauma without a break in the skin barrier. Organisms spread from the subcutaneous tissue along the superficial and deep fascial planes facilitated by bacterial enzymes and toxins. The infection spreads to involve the vascular

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supply to tissue causing vascular occlusion, ischemia, and tissue necrosis. Superficial nerves are often damaged producing localized anesthesia.

The development of NF is caused by interactions between virulence factors of pathogenic organisms and patient comorbidities, predisposing factors, and immune response. Toxins (ie, superantigens) are released by causative pathogens and initiate an endogenous cytokine reaction that ultimately results in hypotension and widespread tissue destruction. Bacteria rapidly multiply within viable tissue. Fibrous attachments between fascia and subcutaneous tissues limit the spread of bacteria (eg, in the feet and hands); however, there is a lack of fibrous attachments in the trunk and limbs, allowing the rapid spread of infection. Edema results from infection-induced cytokine release locally and from spread into venous and lymphatic channels. Breaching of the fascia can lead to infection of the muscle (myositis). *Clostridium* species can produce gas and lead to gas gangrene. With toxin-producing bacteria (ie, *Streptococcus pyogenes* and *Staphylococcus aureus*), toxic shock-like syndromes can occur.

MICROBIOLOGY

Necrotizing fasciitis is often divided into two classifications depending on the causative organism(s): type 1 and 2. Type 1 is more common and is caused by a mixture of aerobic and anaerobic bacteria. Causative pathogens are usually Gram-positive such as the *Bacteroides*, *Clostridium*, *Staphylococcus*, and *Streptococcus* species. Other less commonly reported causative organisms for type 1 NF include the Enterobacteriaceae and fungal species, specifically mucormycosis. Risk factors for type 1 disease include a history of diabetes, peripheral vascular disease, immune-compromised states, and recent surgery.

Type 2 NF is a monomicrobial infection caused by group A *Streptococcus*, *Clostridium* species, or *S aureus*.[1,6] Group A *Streptococcus* and *Clostridium* species share the ability to infect all the compartments of the soft tissues, including fascia and muscle. They also cause a rapidly progressive and severe form of NF medicated through exotoxins (ie, secreted toxin).

Two lesser-known NF classifications are type 3 NF, which is generally caused by *Vibrio vulnificus*, *Clostridium*, and *Klebsiella* species, and type 4 NF, which has a fungal etiology, including *Candida* species and *Zygomycetes*.[7] Neither of these classifications is well understood.

CLINICAL MANIFESTATIONS

Clinical manifestations of NF range from localized to systemic findings. Most studies show a predilection for infection of the extremities followed by perineal and truncal infections.[5,8] Of note, truncal infections are more common in children than adults. Predictors of NF include having a history of recent surgery (past 90 days) or trauma at the affected site, IV drug use, alcoholism, comorbidities (ie, diabetes mellitus, obesity, and peripheral vascular disease), and the use of immunosuppressive drugs.[9]

The initial presentation of NF may vary: NF can follow a hyperacute presentation with sepsis and subsequent multiorgan failure, or, conversely, a subacute course of progression can occur, with a soft-tissue infection followed by sudden deterioration. Initial signs and symptoms of NF include localized swelling, erythema, and pain, as are seen with other types of skin and soft-tissue infections.[10] Notably, pain and systemic symptoms can be disproportionate to physical findings in NF cases.

Individuals with NF will often be tachycardic at initial presentation. As the infection progresses, systemic symptoms such as diaphoresis, chills, and rigor can be observed. Hypotension and shock are common in advanced disease consistent with sepsis. Without intervention, patients with NF will develop severe sepsis and septic shock with altered mental status and cardiovascular and/or pulmonary collapse and will require intensive care and physiologic support. Other organ systems can also be compromised, including the renal system, as indicated by decreased urine output and acute kidney injury.

Locally, advanced infection will often manifest as tense edema outside of the area of compromised skin, pain disproportionate to the wound appearance, and a salmon-colored skin discoloration.[6,11] Vascular thrombosis manifests as a blue discoloration resembling ecchymosis followed by bullae and necrosis of the skin. At this point in the infection progress, nerve damage also occurs, and physical examination may reveal areas of hyperesthesia combined with anesthetic portions of the skin.

DIFFERENTIAL DIAGNOSIS

Diagnosing NF can be challenging because early disease may be difficult to differentiate from a simple cellulitis.[12] Clinicians must have a high index of suspicion for NF as a result.[13] Distinguishing manifestations in NF include sites of infection displaying pain out of proportion with examination findings, skin necrosis, crepitus, and ischemia (with hemorrhagic bullae). In addition, the presence of the following systemic signs and symptoms can indicate NF: tachycardia, fever, hypotension, and tachypnea.

The laboratory risk indicator for NF and finger tests are important tools for NF diagnosis. The former is based on laboratory markers, and the latter is a surgical assessment that can be performed at the bedside or in the OR. In rare cases where diagnosis is still not clear, a fascial biopsy can confirm the diagnosis but should not delay definitive management in cases of NF.

Particularly at the early stages, NF may be mistaken for other soft-tissue infections such as cellulitis or erysipelas. Cellulitis is a soft-tissue infection characterized by inflammation extending to the subcutaneous tissues, whereas

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Table. NF CLINICAL FEATURES, SUGGESTED INVESTIGATIONS, TREATMENT, AND FOLLOW-UP CARE

Feature	Description
Clinical features	
Precipitating triggers	Accidental trauma, superficial graze, blunt trauma lacking skin break, burns, lacerations, penetrating injury, insect and animal bites, skin ulcer, surgery, or childbirth **Type III:** *Clostridium*: infections via wounds or needles *Vibrio*: infected seafood, exposure of open areas to salt/brackish water
Regional manifestations	- Pain disproportionate to physical findings - Red, painful swelling of infected areas of the fascia - Tissue ischemia followed by skin and soft-tissue necrosis - Systemic symptoms range from fevers/chills to septic shock and are nonspecific **Common regions of infection:** Extremities (ie, legs, arms), scrotum, perineum (note: Fournier gangrene), abdominal wall, trunk, head, and neck
Suggested investigations	
Performing swab or tissue for culture	**Levine technique** Clean the wounded area with saline solution to avoid surface organism contamination. Apply pressure to a 1-cm^2 area of viable tissue near the center of the wound base for 5 s
Laboratory procedures	**LRINEC** Differentiates NF from other infections; a score of ≥6 indicates NF. LRINEC scoring considers CRP, white blood cell, hemoglobin, creatinine, sodium, and glucose levels **PCT** PCT is a precursor of calcitonin. During bacterial infection, PCT level will increase (reference value, 0.01 mg/L). Check PCT levels 2 d postoperation to assess for ongoing tissue damage or worsening of NF **CRP** Although CRP measurements are included in LRINEC scoring, they can also be tested individually; CRP levels greater than 150 mg/L can indicate NF **Blood cultures** Blood cultures are commonly completed in the hospital setting; cultures may be positive for pathogens associated with NF
Surgical procedures for diagnosis	**Finger test** Following application of anesthesia, incise approximately 2 cm in suspected area and visually inspect tissues. The absence of normal blood flow and presence of dirty (brownish)-colored fluid and fat discoloration favors diagnosis of NF. Perform a finger sweep at the fascial level. If tissues are readily dissected, this favors an NF diagnosis and indicates the need for debridement. Tissue and fluid may be obtained at this stage for microbiologic diagnosis
Treatment	
Initial treatment	**Surgical treatment** Immediate aggressive debridement. Conduct daily explorative surgery for further debridement until the affected area is completely debrided (surgical decision) **Antibiotic Treatment** Prescribe empiric antibiotics to manage infection. Until wound cultures are available, use broad-coverage antibiotics. Adjust antibiotic treatments to wound cultures and antibiotic sensitivity tests' results. Nutrition, fluids, and antibiotics can be provided intravenously
Antibiotic coverage	**Type 1:** Broad coverage of both anaerobic and aerobic bacterial strains can be achieved via vancomycin, linezolid, or daptomycin along with piperacillin-tazobactam, carbapenem, ceftriaxone and metronidazole, or fluoroquinolone plus metronidazole **Type 2:** MSSA: cephalosporins or penicillin and clindamycin (for 72 h) MRSA: vancomycin, linezolid, or daptomycin **Type 3:** *Clostridium*: clindamycin and penicillin *Vibrio*: tetracyclines and cephalosporins Approximate duration of therapy is 2 wk, although this may be increased based on the extent of infection and presence of necrotic tissue requiring further surgical debridement. Second-line antibiotics may be considered based on allergy/intolerance to beta-lactams or known culture results

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Table. NF CLINICAL FEATURES, SUGGESTED INVESTIGATIONS, TREATMENT, AND FOLLOW-UP CARE, CONTINUED

Feature	Description
Follow-up care	
	- Perform split skin graft with or without prior vacuum-assisted closure therapy - Follow-up every 3 to 6 mo to monitor progress - Physical therapy may assist with pain management and improving function - In the case of partial or full amputation, patients may experience psychological distress; emotional services, social work, or counseling may be beneficial

Abbreviations: CRP, C-reactive protein; LRINEC, laboratory risk indicator for necrotizing fasciitis; MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-susceptible *Staphylococcus aureus*; NF, necrotizing fasciitis; PCT, procalcitonin.

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erysipelas extends to the dermis. The progression, clinical symptoms, and systemic signs of illness can help to differentiate these infections from NF. Other forms of necrotizing infections with varying depths include necrotizing cellulitis and myositis. However, the former tends to have a less severe presentation than NF, and the latter is quite rare and tends to have fewer overlying skin changes.

In subacute presentations, other entities may be considered on the differential diagnosis, but these disorders are rare, primarily occur in different clinical contexts, and do not have the rapid onset and progression seen with NF. Nonnecrotizing fasciitis has an absence of necrosis but otherwise may have similar symptoms as NF. Eosinophilic fasciitis, another mimic of NF, involves infiltration of superficial muscle fascia collagen by lymphocytes, plasma cells, and eosinophils[14] and can be distinguished based on clinical and radiographic features. Proliferative fasciitis is thought to originate from local stress reactions that result in eventual fibrous proliferation. Follow-up imaging is helpful to evaluate for tissue destruction and support the diagnosis.[15] Graft-versus-host disease, which occurs in persons who have undergone stem-cell transplants, is a noninfectious entity that presents with subcutaneous perimuscular/intramuscular edema and involves donor T lymphocytes damaging the epithelial cells that line the recipient target organ.[16]

THERAPY

Treating NF includes the principles of management of all surgical infections: source control, antimicrobial therapy, and physiologic support as needed (see Table). Early and complete surgical debridement is essential for reducing amputation rates and mortality; antimicrobial therapy is an adjunct to source control in the treatment of NF. Mortality estimates for NF range from 25% to 35%, largely due to delays in treatment, and may be even greater in low-resource settings.[17,18] As such, the importance of early recognition and initiation of treatment cannot be overemphasized.

Emergent NF treatment involves surgical debridement of necrotic and infected tissue.[19] Necrotic tissue located on extremities may require amputation based on joint involvement and rapidly progressing infection. Repeat debridement is often required at frequent intervals to ensure source control is achieved. Physiologic support includes adequate fluid resuscitation and oxygenation. In advanced stages, organ failure may necessitate intensive care with mechanical ventilation and pressor support with or without renal replacement therapies.

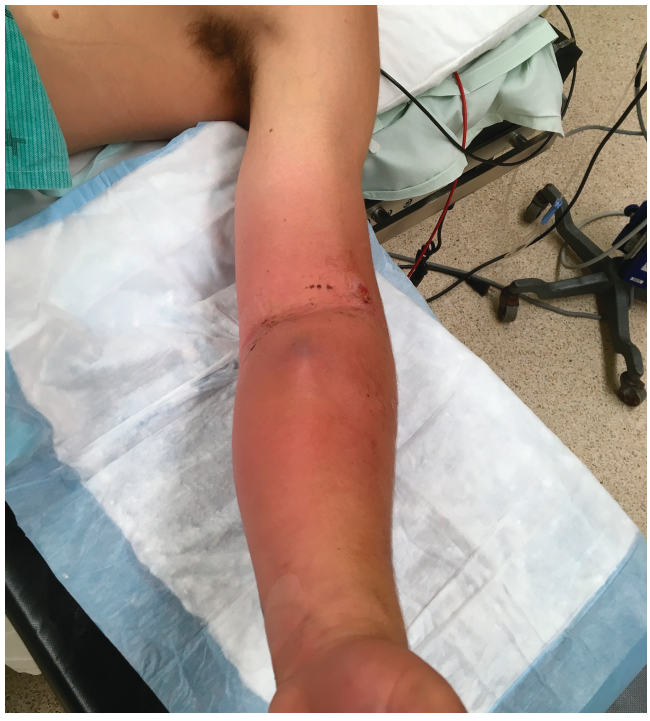
Antimicrobial therapy should be started early and include broad empiric coverage for Gram-positive, Gram-negative, and anaerobic species. Therapy targeting methicillin-resistant *S aureus* may also be used empirically based on local rates, risk factors, and clinical presentation. Specific antimicrobial therapy will differ based on whether the infection is monomicrobial (type 2) or polymicrobial (type 1). For polymicrobial infections, initial therapy can include vancomycin plus piperacillin-tazobactam or a carbapenem such as meropenem.[11] For patients with penicillin allergies, clindamycin is an acceptable replacement for piperacillin-tazobactam. Clinicians should perform blood cultures but not delay the initiation of antimicrobial therapy or surgical intervention. High-dose IV clindamycin may be added to therapy initially because it can help to reduce toxin production in cases of group A *Streptococcus*-associated NF (type 2). Intravenous immunoglobulin therapy can bind exotoxins and decrease inflammatory response in NF but is currently recommended only for critically ill patients who have streptococci as causative agents.[19] Hyperbaric oxygen therapy is not currently recommended in guidelines for NF management.

CASE REPORTS

Case 1

W.M. is a 26-year-old homeless man with amphetamine/opiate/IV drug use and hepatitis C virus who presented to the ED with redness and swelling of his left arm and erratic, confused behavior. His temperature was 38.3 °C with a heart rate of 140 beats per minute; other vital signs were stable. Blood tests revealed a white blood count of 19.9×10^9/L (reference range, 4.5–11 $\times 10^9$/L), C-reactive protein level of 152.5 mg/L (reference range, 0.3–1.0 mg/L), blood glucose concentration of 5.0 mmol/L (reference,

Figure 1. CASE 1 CLINICAL PRESENTATION



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<6.9 mmol/L), and creatinine levels of 106 mmol/L (reference range for men, 61.9-114.9 mmol/L). X-ray imaging of his left forearm identified two small areas of gas in the tissues, and he was urgently taken for surgery based on the medical team's suspicion of necrotizing infection (Figure 1).

Intraoperatively, the surgeon noted an approximately 10-cm abscess at the patient's left proximal forearm underlying the brachioradialis, proximally tracking to the distal lateral and volar forearm. Incision and debridement of the necrotic tissues and a volar fasciotomy were performed. Repeat debridement was performed 24 hours later (Figure 2). Gram stain revealed Gram-positive cocci and Gram-negative bacilli with tissue culture growing *Streptococcus anginosus* and an anaerobic Gram-positive bacillus.

Broad empiric treatment was prescribed with piperacillin/tazobactam and vancomycin with delayed primary wound closure. Subsequently, antibiotics were narrowed to ceftriaxone and metronidazole. The patient received a total of 10 days of antibiotic therapy prior to being discharged with ongoing wound care.

Case 2

S.P. is a 59-year-old male smoker who presented with fever (temperature of 38.5 °C) and a draining left foot wound of 3 days' duration (Figures 3 and 4). Left foot X-ray demonstrated diffuse soft-tissue prominence with subcutaneous gas but no bony changes. He had an elevated white blood cell count of 22.2 × 10^9/L, C-reactive protein level of 202 mg/L, blood glucose concentration of 15.8 mmol/L, and normal creatinine.

Urgent surgery was performed with debridement of the near-circumferential eschar and necrotic soft tissue, which extended proximally toward the lateral malleolus. Gram stain revealed Gram-negative coccobacilli and Gram-positive cocci, and tissue cultures grew *S aureus*, *Peptostreptococcus* species, and *Prevotella* species.

The patient was admitted to the ICU for postoperative care and treated empirically with piperacillin/tazobactam and clindamycin for his polymicrobial infection. Follow-up CT imaging revealed progression of the infection to the distal lower leg and posterior compartment with significant occlusive peripheral vascular disease. Because of a lack of reconstructive options for the left foot, the patient underwent a below-knee amputation. Following his amputation, he received a new diagnosis of type 2 diabetes and was transferred to the rehabilitation unit.

CONCLUSIONS

Necrotizing fasciitis is an uncommon but rapidly progressive soft-tissue infection with associated mortality. Early recognition of NF is crucial to implement appropriate management and improve outcomes. ●

Figure 2. CASE 1 POSTSURGERY

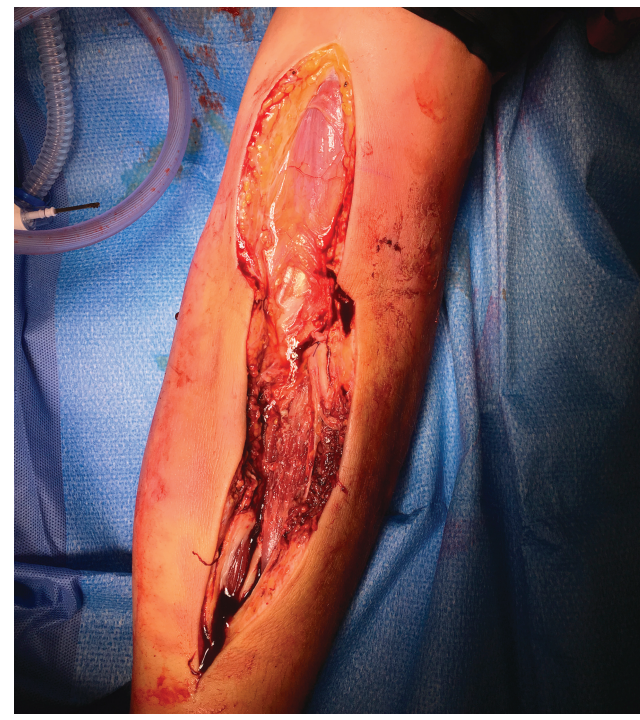

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Figure 3. CASE 2 CLINICAL PRESENTATION, DORSAL VIEW

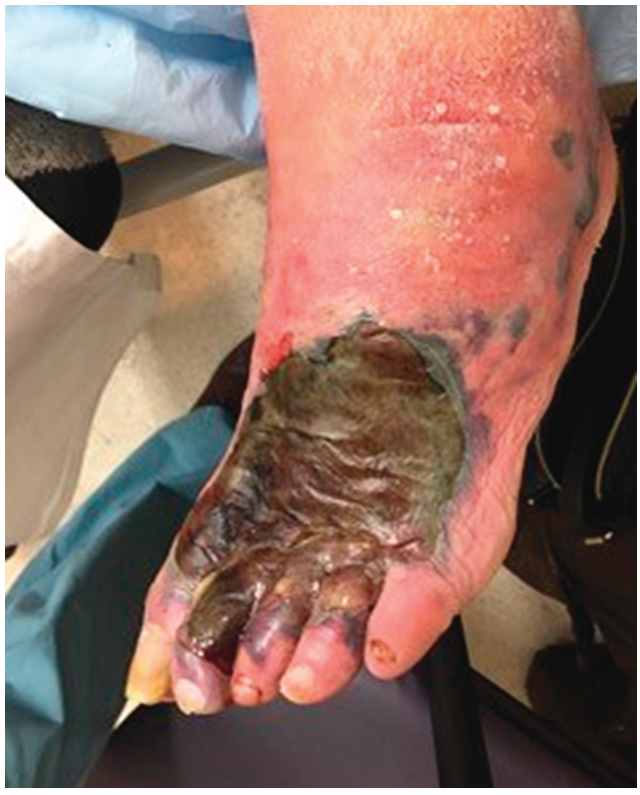
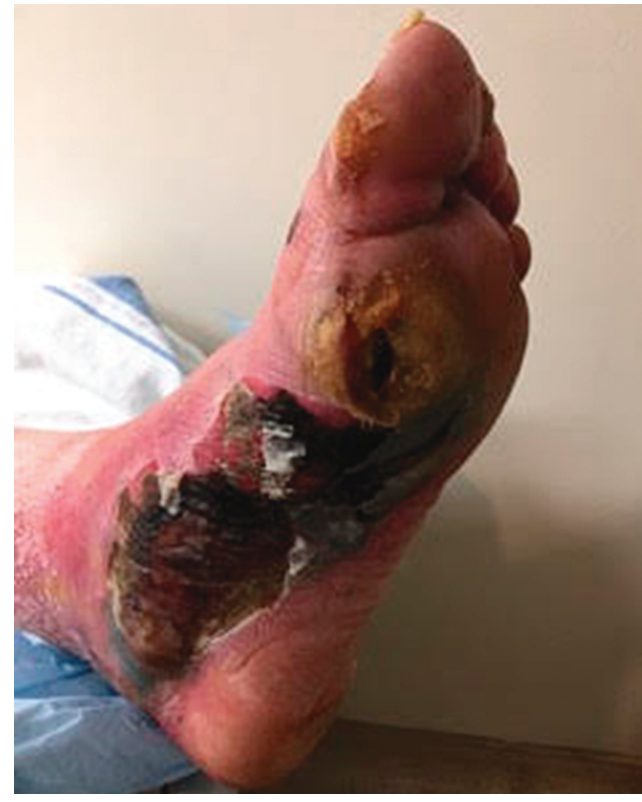


Figure 4. CASE 2 CLINICAL PRESENTATION, PLANTAR VIEW



PRACTICE PEARLS

- Necrotizing fasciitis is a rapidly progressive soft-tissue infection that can lead to death and disability.
- Risk factors for NF include older age, male sex, immunosuppression, and a history of trauma or surgery to an affected site.
- A high index of suspicion is needed to diagnose NF based on its nonspecific clinical presentation.
- Early surgical debridement of the infected site is essential in cases of NF to improve outcomes.

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