Necrotizing fasciitis: Frightening disease, potentially grim prognosis

This infection can be devastating, but the survival rate is improving.

By Lydia Meyers, BSN, RN, CWCN

Necrotizing fasciitis (NF) results from an infection that attacks the fascia and subcutaneous tissues. The primary bacterial etiology is group A streptococcus, a facultative anaerobic bacterium. However, other bacteria may contribute. Sometimes called the "flesh-eating" disease because of the potentially devastating effect on the afflicted patient, NF can be monomicrobial or polymicrobial.

The four typical settings for NF are:

- surgical bowel or abdominal trauma surgery
- pressure ulcer and perianal abscess
- injection sites (especially in drug users)
- Bartholin abscess or minor vulvovaginal infection.

Because of the rapid course and ravaging nature of acute NF, clinicians must maintain a high index of suspicion if the patient has suggestive signs and symptoms. In 1990, puppeteer Jim Henson (best known for creating the Muppets) died from NF. At that time, little was known about the progression of group A streptococcal infection.

The disease can quickly cause death, so starting immediate treatment is even more crucial than confirming the diagnosis. Once the disease is suspected, antibiotics must be given immediately and the patient must be prepared for surgery at once. NF spreads rapidly, capable of progressing from a small lesion to death in days to weeks. Thus, delayed diagnosis increases the risk of death. Lack of knowledge about the disease and inability to recognize it promptly are the main reasons many victims die. This article can improve your knowledge base.

Overview

NF was discovered in 1871 by Joseph Jones, a Confederate Army surgeon. At that time, it was called hemolytic streptococcal gangrene, nonclostridial gas gangrene, nonclostridial crepitant cellulitis, necrotizing or gangrenous erysipelas, necrotizing cellulitis, bacterial synergistic gangrene, or synergistic necrotizing cellulitis.

NF involves the fascia, muscle compartments, or both. It can affect not only the muscle fascia but the superficial fascia. NF and cellulitis differ in the amount of tissue involved and extent of tissue involvement.

The most common areas of infection are the abdominal wall, perineum, and extremities. When NF affects the perineum and scrotum, it's called Fournier gangrene, after the French dermatologist and virologist Alfred Jean Fournier.

The most common causes are trauma, surgery, and insect bites. The disease can affect persons of any age. Such comorbidities as diabetes, chronic renal failure, immunosuppressive therapy, hypertension, obesity, and malnutrition increase susceptibility.

Pathophysiology

NF falls into four classifications based on wound microbiology. *Type 1*, the most common, involves polymicrobial bacteria. *Type 2* results from trauma and is associated with comorbidities. *Type 3*, rare in this country, stems from gram-negative marine bacteria. *Type 4* is a fungal infection occurring mostly in immunocompromised persons. (See *Comparing types of necrotizing fasciitis*.)

Disease progression

The four types of NF progress in a similar way. Bacteria secrete pyrogenic exotoxin A, which stimulates cytokines. These cytokines damage the endothelial lining; fluid then leaks into the extravascular space. M proteins in streptococci and β-hemolytic streptococci exacerbate the immune reaction by inhibiting phagocytosis of polymorphonuclear leukocytes and normal neutrophil chemotaxis. As the immune reaction increases, blood vessels dilate, allowing toxins to leak through vessel walls, which in turn decreases blood flow. As the cascade continues, hypoxic conditions cause facultative aerobic organisms to grow and become anaerobic. These bacteria exacerbate destruction of surrounding cells and lead to release of carbon dioxide, water, hydrogen, nitrogen, hydrogen sulfide, and methane. As the infection continues to progress, toxins spread throughout the bloodstream and the patient becomes septic.

> In all NF types, patients commonly present with a small, painful area.

Assessment

Obtain the patient's medical history and description of the wound. Determine when the changes first appeared and whether the affected area seemed to get worse recently.

In all NF types, patients commonly present with a small, painful area (possibly with entry marks) but no other signs or symptoms. The wound may appear as a bulla, cellulitis, or dermatitis, representing an infection developing in underlying tissues. The skin may have a wooden-hard feel as the infection progresses to the subcutaneous space and causes necrosis. The wound becomes discolored and necrotic; drainage is rare until surgical debridement begins. The patient quickly develops fever, chills, nausea, and vomiting. As NF progresses, bullae become dark purple with darkened edges; the patient grows disoriented and lethargic, and organ failure and respiratory failure ensue. Without treatment, the patient dies.

Diagnosis

Diagnostic tests usually include magnetic resonance imaging, complete blood count with differential, comprehensive metabolic panel, and cultures. (See *Diagnostic findings in necrotizing fasciitis*.)

Treatment

Immediate surgical debridement and broad-spectrum antibiotics are needed to stop the immune response to infection. Clindamycin, gentamicin, penicillin, or metronidazole may be given alone or in combination until culture results are available. Supportive care includes total parenteral nutrition for nutritional support,

Comparing types of necrotizing fasciitis

Type	Microbe(s) involved	Features
Type 1	Polymicrobial, including group A hemolytic streptococci, staphylococci, anaerobic and aerobic *Staphylococcus pyogenes, Staphylococcus aureus, Vibrio vulnificus*, and *Aeromonas hydrophila*	• Rapid necrosis • Systemic signs and symptoms
Type 2	Group A streptococci, with or without staphylococci	• Often associated with trauma • Skin lesion present • Develops over days
Type 3	Gram-negative, marine-related organisms (*Vibrio* species, *Mycobacterium ulcerans*)	• Incubation period of 2 months to 2 years • Presents as ulcerated, edematous lesion
Type 4	Fungi (commonly *Mucor, Rhizopus*, or *Absidia* species of the Mucorales order)	• Occurs mainly in immunocompromised persons • High mortality (70% to 100%) • Progresses rapidly once lesions appear • May result from contaminated dressing or splints applied to skin • Also called mucormycosis, zygomycosis, or phycomysosis

Type 1 NF typically is associated with multiple bacteria, both aerobic and anaerobic (including facultative bacteria). This rapidly progressing, severe form causes systemic sepsis. Facultative bacteria can change from anaerobic to aerobic, depending on the environment.

Type 2 NF can occur at any age, and may or may not be linked to other illnesses. The main bacterium involved is group A β-hemolytic streptococcus, a facultative aerobic bacterium.

Type 3 NF is associated with gram-negative, marine-related organisms and is more common near coastal waters. It commonly appears as a painful edematous lesion, which may indicate bacteria are becoming active and starting a devastating infection. Signs and symptoms arise 3 to 7 days after exposure related to trauma. Patients need emergency care with immediate antibiotic administration and surgical debridement.

Type 4 NF is a rare fungal infection occasionally seen in immunocompromised persons. It may become necrotizing and spread quickly. The typical cause is introduction of bacteria from contaminated dressings, splints, and foreign bodies. The causative fungi are found in soil, decaying wood, and other organic matter.

I.V. fluids, and oxygen. Limb amputation should be done only as a last resort.

Surgical debridement involves penetrating deep into the fascia and removing all necrotic tissue. After the first debridement, release of "dishwater fluid" may occur. Administering hyperbaric oxygen therapy (HBOT) after the first debridement increases tissue oxygenation, thus reducing tissue destruction by anaerobic bacteria. During HBOT (usually given as a 90-minute treatment), the patient breathes 100% oxygen in an environment of increasing atmospheric pressure.

HBOT should be given in conjunction with surgical debridement (usually after each debridement) and should continue until necrotic tissue ceases and cell destruction stops. HBOT also promotes collagen synthesis and neoangiogenesis (new

Diagnostic findings in necrotizing fasciitis

Diagnostic test	Findings in NF
Magnetic resonance imaging	• Underlying pockets of gas and subcutaneous air spaces
Complete blood cell count with differential	• White blood cell count > 14,000/mm^3 • Hemoglobin < 10 g/dL • Thrombocytopenia (platelet count < 150,000/µL)
Comprehensive metabolic panel	• Blood urea nitrogen > 15 mg/dL • Creatinine > 2 mg/dL • Sodium < 135 mEq/L • Increased blood glucose and lactate levels • Decreased calcium, protein, albumin, and cholesterol levels
Cultures	• Vary with bacteria present

blood vessel growth), which boosts blood supply and oxygen to tissues.

Adverse effects of HBOT include ear pain, oxygen toxicity, and seizures. Ear pain can be minimized by swallowing or yawning. If the patient continues to have ear pain, ear tubes may be inserted by an otolaryngologist. During HBOT, air breaks (intervals of breathing room air) are important in controlling oxygen toxicity (the main cause of seizures).

Throughout the HBOT treatment period, wound dressings must be simple. Well-moistened gauze dressings and an abdominal pad provide good support. Once necrotic destruction occurs, dressings depend on wound size and the need to fill cavities. The patient may require a diverting colostomy, depending on wound location and the amount of uncontrolled diarrhea. Blood glucose levels must be monitored before and after HBOT, as this treatment affects blood glucose.

Supportive care and follow-up treatment

During initial treatment, patients need supportive care and monitoring. Once they're out of danger, begin teaching them how to prevent NF recurrences. Advise them to control blood glucose levels, keeping the glycated hemoglobin (HbA1c) level to 7% or less. Caution patients to keep needles capped until use and not to reuse needles. Instruct them to clean the skin thoroughly before blood glucose testing or insulin injection, and to use alcohol pads to clean the area afterward.

Before discharge, help arrange the patient's aftercare, including home health care for wound management and teaching, social services to promote adjustment to lifestyle changes and financial concerns, and physical therapy to help rebuild strength and promote the return to optimal physical health. One helpful patient resource is the **National Necrotizing Fasciitis Foundation**. The Centers for Disease Control and Prevention section on necrotizing fasciitis includes "**Common sense and great wound care are the best ways to prevent a bacterial skin infection**."

The life-threatening nature of NF, scarring caused by the disease, and in some cases the need for limb amputation can alter the patient's attitude and viewpoint, so be sure to take a holistic approach when dealing with the patient and family. Today, NF has a much better survival rate than 2 decades ago when Jim Henson

(continued on page 19)

tissue when the dressing is removed.

Ostomy paste products can serve as effective filler. These pliable products can be spread into position to obtain a secure seal under the transparent drape in hard-to-seal areas, such as the perineum. Pastes remain flexible and can be removed without residue. Temporarily increasing NPWT pressure to a higher setting may help locate a subtle leak or provide enough negative pressure to self-seal the leak. Once the leak resolves, remember to return the pressure to the ordered setting.

Knowledge optimizes healing

It's important to be aware of potential complications of NPWT (See *Take care with NPWT*). However, when applied correctly, NPWT is an effective option for managing complex wounds. Recognizing and managing potential complications at the wound site, ensuring periwound protection, minimizing epibole formation, and preventing wound infection can result in a better-prepared wound bed and promote optimal healing. ■

View: NPWT case study

Review a step-by-step process for preparing a wound for negative pressure wound therapy (NPWT)

Selected references

Baranoski S, Ayello EA. (2012). *Wound Care Essentials: Practice Principles*. 3rd ed. Springhouse, PA; Lippincott Williams & Wilkins.

Bovill E, Banwell PE, Teot L, et al. Topical negative pressure wound therapy: a review of its role and guidelines for its use in the management of acute wounds. *Int Wound J*. 2008;5:511-529.

Sussman C, Bates-Jensen B. *Wound Care: A Collaborative Practice Manual for Health Professionals*. 4th ed. Baltimore, MD; Lippincott Williams & Wilkins; 2011.

Ronald Rock is an Adult Health Clinical Nurse Specialist in the Digestive Disease Institute at the Cleveland Clinic in Cleveland, Ohio.

Necrotizing fasciitis

(continued from page 10)

died. In my practice, I've seen four NF cases. Thanks to early identification, good wound care, and HBOT, these patients suffered only minimal damage. ■

Selected references

Boyer A, Vargas F, Coste F, et al. Influence of surgical treatment timing on mortality from necrotizing soft tissue infections requiring intensive care management. *Intensive Care Med*. 2009;35(5):847-853. doi:10.1007/s00134-008-1373-4.

Cain S. Necrotizing fasciitis: recognition and care. *Practice Nurs*. 2010;21(6):297-302.

Centers for Disease Control and Prevention. Notes from the field: fatal fungal soft-tissue infections after a tornado—Joplin, Missouri, 2011. *MMWR*. 2011;60(29):992.

Chamber AC, Leaper DJ. Role of oxygen in wound healing: a review of evidence. *J Wound Care*. 2011; 20(4):160-164.

Christophoros K, Achilleas K, Vasilia D, et al. Postraumatic zygomycotic necrotizing abdominal wall fasciitis with intraabdominal invasion in a non immunosuppressed patient. *Internet J Surg*. 2007;11(1). doi:10.5580/17a8.

Ecker K-W, Baars A, Topfer J, Frank J. Necrotizing fasciitis of the perineum and the abdominal wall-surgical approach. *Europ J Trauma Emerg Surg*. 2008; 34(3):219-228. doi:10.1007/s00068-008-8072-2.

Hunter J, Quarterman C, Waseem M, Wills A. Diagnosis and management of necrotizing fasciitis. *Br J Hosp Med*. 2011;72(7):391-395.

Magel DC. The nurse's role in managing necrotizing fasciitis. *AORN J*. 2008;88(6):977-982.

Phanzu MD, Bafende AE, Imposo BB, Meyers WM, Portaels F. Under treated necrotizing fasciitis masquerading as ulcerated edematous Mycobacterium ulcerans infection (Buruli ulcer). *Am J Trop Med Hyg*. 2012;82(3):478-481.

Ruth-Sahd LA, Gonzales M. Multiple dimensions of caring for a patient with acute necrotizing fasciitis. *Dimens Crit Care Nurs*. 2006;25(1):15-21.

Stevens DL, Bisno AL, Chambers HF, et al; Infectious Diseases Society of America. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. *Clin Infect Dis*. 2005;41(10):1373-1406.

Su YC, Chen HW, Hong YC, Chen CT, et al. Laboratory risk indicator for necrotizing fasciitis score and the outcomes. *ANZ J Surg*. 2008;78(11):968-972.

Taviloglu K, Yanar H. Necrotizing fasciitis: strategies for diagnosis and management. *World J Emerg Surg*. 2007;2:19.

Lydia Meyers is a medical reviewer for National Government Services in Castleton, Indiana, and a clinical liaison at CTI Nutrition in Indianapolis. She has 11 years of wound care experience in nursing homes, wound clinics, and home health.