Necrotizing Fasciitis

Even with optimal treatment, the mortality rate is 40%.

Few phrases can make one's skin crawl like the words "flesh-eating bacteria." The term was coined by the British tabloids, whose reports on necrotizing fasciitis suggested that this infection would imminently become epidemic—a prediction that remains unfounded. The myths surrounding this very real, very deadly disease evoke terror, spurred on by graphic and sensational coverage in the media.

Necrotizing fasciitis is a relatively infrequent infection of the subcutaneous tissue that destroys fascia; it may lead to loss of limb or life. However, the disease remains frightening because it progresses rapidly and can cause death within 24 hours.

WHO IS AT RISK?
Immunocompromised patients, those with intravenous drug or alcohol problems, and those with systemic illnesses such as diabetes mellitus, peripheral vascular disease, malignancy, or atherosclerosis have an increased risk of developing necrotizing fasciitis. Postoperative patients and patients with burns or traumatic wounds also have an elevated risk. However, previously healthy persons can also develop the disease.

The disease is classified into two types. Type I is usually caused by mixed aerobic or anaerobic bacteria, such as group A streptococcus, Bacteroides fragilis, Staphylococcus aureus, species of Clostridium, Pseudomonas aeruginosa, Enterobacteriaceae, and others. It's seen in postoperative patients and patients with diabetes or other immunosuppressive conditions. Type II is caused by group A streptococcus or, rarely, by group B streptococcus. It's seen in any age group, commonly after minor trauma, although it may develop even in the absence of any underlying medical condition.

The majority of cultures from patients with necrotizing fasciitis reveal mixed aerobic and anaerobic bacteria, with group A streptococcus the most common causative organism. The Centers for Disease Control and Prevention estimates that 500 to 1,500 cases occur annually in the United States.

HOW IT'S TRANSMITTED
From 15% to 30% of the population carry group A streptococci in the throat or on the skin without symptoms and, consequently, can transmit the organism to others via coughing or sneezing or through direct contact with secretions. Entry via the skin can occur through a surgical wound, a paper cut, or any other opening in the skin. Contaminated hands can be an important means of transmission in the hospital.

Transmission of group A streptococci is a frequent occurrence, but rarely does it cause necrotizing fasciitis. Patients may develop a less invasive infection or no infection at all. S. pyogenes, the most notable member of group A streptococci, is a frequent cause of pharyngitis, tonsillitis, scarlet fever, cellulitis, and impetigo.

But in those who do develop necrotizing fasciitis, these invasive bacteria wreak havoc by spreading rapidly along subcutaneous tissues and fascial planes, causing necrosis of fascia, overlying skin, and vasculature. The disease progresses to septicemia and culminates in multisystem organ failure.

About 50% of patients with necrotizing fasciitis have clinical features overlapping those of streptococcal toxic shock syndrome, a disease of shock and multiorgan failure associated with the release of streptococcal pyrogenic exotoxins and evolving from skin and soft-tissue infection.

Even if treated early and aggressively, necrotizing fasciitis has a 30% to 40% mortality rate. However, if left untreated, necrotizing fasciitis is even more deadly than the highly publicized Ebola virus, producing a 100% mortality rate.

IDENTIFYING NECROTIZING FASCIITIS
Necrotizing fasciitis most commonly affects the extremities, abdominal wall, or perineum. The presenting symptomatology is characterized by three stages: early, advanced, and critical.

Early findings, within the first 24 hours, include flulike symptoms, a wound site that is essentially unremarkable (if present at all), localized pain, erythema, and swelling. At this stage, because of the benign nature of the symptoms, the disease is often misdiagnosed, typically as cellulitis or the flu.

However, in the early stages of necrotizing fasciitis, a key identifying factor is pain far beyond what is
expected for a “minor injury.”

As the disease progresses to the advanced stage over two to four days, the skin becomes tense, swollen, shiny, and purplish, and develops hemorrhagic fluid-filled blisters. The skin takes on a tissue paperlike appearance with overlying skin anesthesia (a clue that this is not simple cellulitis), because of the destruction of cutaneous nerves. Subcutaneous emphysema or crepitation can be palpated, if gas-forming bacteria are involved.

With progression to the critical stage, at four to five days, the purple and blue areas become gangrenous. Necrosis with skin sloughing is plainly visible. If left untreated, death may ensue within hours.

Septic shock, manifested by hypotension, delirium, hepatic dysfunction, and acute renal failure, may develop as early as 24 hours after the onset of the process from the release of bacterial toxins. Even with aggressive management, septic shock, neurologic deterioration, and eventual death occur at an alarmingly high rate.

Laboratory studies typically reveal signs of infection (leukocytosis with a predominance of neutrophils), muscle damage (elevated creatine phosphokinase levels), hepatic or renal insufficiency or failure (elevated liver enzyme, blood urea nitrogen, and creatinine levels), electrolyte imbalances, and anemia. Radiographs or CT scans, often used to rule out other injuries, can show soft tissue swelling and, occasionally, “bubbles” of subcutaneous air formed by gas-producing organisms such as _Clostridium_ (a cause of type I necrotizing fasciitis), as seen in gas gangrene.

Gram staining and culturing the tissue and fluid can reveal the etiology. Early, accurate diagnosis can also be obtained with bedside biopsy of tissue and immediate frozen section evaluation.

**TREATMENT**

Unlike most emerging infections, necrotizing fasciitis is primarily treated surgically. Shock and multisystem organ failure are treated with fluid resuscitation, antibiotics, and vasopressors (such as dopamine).

The definitive emergency treatment is immediate and repeated surgical debridement to remove all necrotic tissue. Sometimes amputation of a limb is required. A “second-look” surgery is typically done 12 to 24 hours after the initial debridement. Patients with necrotizing fasciitis may require anywhere from five to 40 operative debridements; one study found an average of 33 operative debridements and grafting procedures.

In addition to surgical management, therapies such as intravenous immunoglobulin and hypobaric oxygen therapy—the use of oxygen at increased pressure to increase oxygen saturation in the wound—can be used. The efficacy of hypobaric oxygen therapy remains unproven; it's been shown to be efficacious only in the treatment of infections due to _Clostridium_. Attention to nutritional support with enteral or total parenteral nutrition is also essential. Patients with necrotizing fasciitis are ideally managed in a burn center or surgical ICU setting. Standard precautions, cleaning of equipment, and contact precautions are required in the care of these patients.

Despite optimal care, early diagnosis, and aggressive treatment, necrotizing fasciitis carries a high mortality rate. For those who recover, extensive and continued rehabilitation is essential to managing the psychological and physical sequelae of this disease.

**THE NURSE'S ROLE**

Caring for the “tiniest of traumas” and practicing basic hygiene measures are the best ways to minimize the chances of spreading the bacteria most associated with the “flesh-eating disease.” Since contaminated hands are an important means of bacterial transmission, be sure to wash them frequently and between every patient contact.

Delays in the diagnosis of necrotizing fasciitis or in treatment via surgical debridement are associated with greater morbidity and mortality. Nurses who can promptly recognize the possibility of necrotizing fasciitis can save a patient’s life.

**REFERENCES**