Necrotizing Skin and Soft Tissue Infections

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KEYWORDS

• Necrotizing skin and soft tissue infection (NSSTI) • Necrotizing fasciitis
• Gas gangrene • Fournier's gangrene • Wide local debridement

KEY POINTS

- Necrotizing skin and soft tissue infections (NSSTIs) are caused by aggressive and often toxin-secreting bacteria.
- NSSTIs may be caused by single agents such as clostridia or streptococci (type I), but are often polymicrobial (type II).
- Immunosuppressed individuals are especially susceptible to NSSTIs.
- Systemic signs of infection are ubiquitous, with patients often becoming septic and showing signs of multiorgan dysfunction syndrome.
- The Laboratory Risk Indicator for Necrotizing Fasciitis has a high positive and negative predictive value for NSSTIs.
- Computed tomography might be helpful in diagnosing NSSTIs, but is often not sufficient to rule it out.
- Prompt wide surgical debridement and broad spectrum antibiotics are the key elements needed for successful management and prevention of the high morbidity and mortality associated with NSSTIs.

INTRODUCTION

Necrotizing skin and soft tissue infections (NSSTIs) are severe infections resulting in life-threatening soft tissue destruction and necrosis and resulting from toxin-secreting bacteria. Extensive, rapid, and widespread progression of the infection and necrosis along soft tissue planes is the essential characteristics of the disease.

Epidemiology

The epidemiology of NSSTIs is not very well established; reliable data on its incidence are largely absent from medical literature, and most published studies reflect

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individual institutional experiences. Using the Centers for Disease Control and Prevention data, O'Loughlin and colleagues[1] report an incidence of 3.5 cases in 100,000 persons of NSSTIs specifically caused by group A streptococcus in the United States and a case-fatality rate of 13.7%. Viewing the wide variation in reporting the clinical specifics, the treatment modalities, the outcome, and eventual prognosis of NSSTI patients, there is a definite need for large national multi-institutional registries for NSSTIs, similar to those existing for trauma or oncology patients.

History

One of the earlier medical references to NSSTIs was in 1871, whereby more than 2600 cases were reported during the American Civil War by a Confederate Army surgeon, Joseph Jones.[2] At that time and continuing until the early to mid 20th century, the disease entity was popularized in media as being caused by "flesh-eating bacteria," obviously a misnomer. NSSTI is also referred to in medical literature as gas gangrene, necrotizing skin and skin structure infections, necrotizing fasciitis, Fournier gangrene (when affecting the groin, genital organs, and perineum), and Ludwig angina (when affecting the submandibular floor of the mouth, usually in relationship with serious dental infections).

PATHOPHYSIOLOGY

Occasionally, an infection entry point such as a subtle wound is present on physical examination. The history of an insect bite or minor laceration can also be occasionally elicited. Many other times, such history or evidence of a wound break is not present.

Microbiology

There are 2 types of NSSTIs. Type I infections are monomicrobial and are most commonly caused by anaerobic bacteria such as clostridia, streptococci, or bacteroides species. Type II infections are polymicrobial in cause. Most causative pathogens, especially *Clostridium perfringens*, secrete exotoxins (eg, hemolysins, collagenases, lecithinases, proteases) that lead to fast evolution of deep tissue necrosis along fascial and muscle layers. Concomitant small-vessel thrombosis results in the grayish skin hue that is often noted on careful examination of these patients. Due to both the small vessel thrombosis and the progression of infection along deeper soft tissue layers, the infection is often more widespread than apparent on physical examination of the underlying skin. Systemically, the secreted exotoxins lead to increased cytokine production, T-cell proliferation, as well as interleukin (IL) and lymphokine secretion (IL-1, IL-2 IL-6, tumor necrosis factor-α and -β), clinically leading to severe sepsis/septic shock, pronounced systemic inflammatory response syndrome, and not uncommonly, multi-organ dysfunction syndrome.

Risk factors

Immunocompromised patients, including patients with diabetes mellitus, human immunodeficiency virus, malnutrition, peripheral vascular disease or malignancy, and intravenous drug abuse patients, are at an especially increased risk of developing NSSTIs.

CLINICAL MANIFESTATION

Early suspicion and prompt diagnosis of NSSTIs are critical, as fast progression to systemic shock and lethal septic shock are definite if aggressive control of the infection is not obtained.