Necrotizing Soft Tissue Infections: A Review of Diagnosis, Management, and Implications for NP Practice

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ABSTRACT

Skin and soft tissue infections are among the most common diagnoses seen by nurse practitioners practicing in all settings. These infections range from mild, uncomplicated cellulitis to the more severe, complicated diagnosis of necrotizing soft tissue infections (NSTI). For patients presenting with symptoms of skin and soft tissue infections, differentiating NSTIs from less insidious infections is of paramount importance. NSTIs can be difficult to diagnose because the early presentation may be misleadingly straightforward; however, it is essential that NPs carefully consider NSTIs when a patient presents with the following cardinal skin signs: erythema, swelling, and warmth.

Keywords: cellulitis, necrotizing fasciitis, necrotizing soft tissue infection, skin and soft tissue infection, skin infections

In the past several years, visits for skin and soft tissue infections (SSTIs) have grown significantly. Researchers identified 56,527 weighted necrotizing soft tissue infection (NSTI) admissions from the Nationwide Inpatient Sample from 1998 to 2010. This study found an incidence of NSTI ranging from 3,800 to 5,800 cases annually.[1] A retrospective study conducted by Pallin et al[2] found a 3-fold increase in the number of patients presenting to the emergency department (ED) for SSTIs, from 1.2 million in 1993 to 3.4 million in 2005. Hsiao et al[3] conducted a retrospective cohort study of patients admitted via the ED with a discharge diagnosis of necrotizing fasciitis. This study examined 128 patient cases and determined that only 49 patients (38.3%) were diagnosed or suspected to have necrotizing fasciitis in the ED. Because of the high mortality associated with NSTIs, it is imperative that nurse practitioners (NPs) consider this diagnosis in all patients presenting with cardinal skin signs and act quickly when this diagnosis is suspected.

NSTIs are a group of rare but complex infections. These infections are characterized by rapidly advancing tissue necrosis. Other common names for these infections include gas gangrene, streptococcal gangrene, gangrenous cellulitis, necrotizing cellulitis, necrotizing fasciitis, and Fournier gangrene.[3] NSTIs are highly lethal conditions requiring early recognition, diagnosis, and aggressive intervention to preserve life and limb.[4]

The data on the incidence and prevalence of NSTIs are contentious because many statistics only support NSTIs caused by group A *Streptococcus* (GAS). Although GAS is one of the important pathogens responsible for NSTIs, several other microorganisms contribute to the pathogenesis of these infections.[5,6] The Centers for Disease Control and Prevention tracks specific GAS infections cumulatively, including necrotizing skin infections, strep throat, impetigo, and streptococcal toxic shock syndrome.[7] The Center for Disease Control and Prevention reports approximately 9,000 to 11,500 cases of invasive GAS infections each year, with SSTIs and necrotizing fasciitis comprising about 6% to 7% of these cases.[7] An ED-based study revealed an occurrence of 0.17 cases of GAS necrotizing fasciitis per 1,000 ED visits.[8]

Although still an uncommon diagnosis, the incidence of this life-threatening condition has increased in the past several decades.[5,9–11] It is suggested that the frequency of these infections has been on the rise

because of an increase in conditions that may result in immunocompromise, such as diabetes mellitus, cancer, alcoholism, obesity, advancing age, vascular insufficiencies, organ transplants, human immunodefi ciency virus, or neutropenia.[12]

CLASSIFICATION

NSTIs are classified based on anatomy, depth of infection, or microbial source of infection. These classification systems are extremely useful because they provide a common language for research and treatment. Additionally, the depth of the primary site of infection correlates directly with mortality.[4] Necrotizing adipositis is the most superficial and is the most common type of NSTI. Necrotizing fasciitis is characterized by pathological findings at the level of the subcutaneous fat and deep fascia, whereas necrotizing myositis is associated with the highest mortality.

Type I NSTIs are the most common type, accounting for 55% to 75% of infections. Most commonly, they are polymicrobial, including gram-positive cocci, gram-negative bacilli, and anaerobes. They are associated with multiple predisposing factors, including surgical procedures, diabetes, and peripheral vascular disease.[13] Type I infections tend to occur in the perineal and trunk areas. Additional risk factors include obesity, chronic renal failure, human immunodeficiency virus, alcohol abuse, abscess, intravenous drug use, blunt or penetrating trauma, insect bites, surgical incisions, indwelling catheters, chicken pox, vesicles, and, rarely, perforation of the gastrointestinal tract. Despite this exhaustive list of risk factors, approximately half of the patients diagnosed with type I NSTIs cannot recall a specific inciting event.[4]

Type II NSTIs are less common and occur in young, healthy individuals, typically on the extremities, although lesions on the trunk have been reported. Type II NSTIs are caused by group A beta hemolytic streptococci, with or without *Staphylococcus aureus*. Since 2004, there has been an increasing incidence of community-acquired methicillin-resistant *S aureus* (MRSA) reported.[4] MRSA is cultured in up to 40% of necrotic wounds, and, most commonly, those at risk include intravenous drug users, athletes, and institutionalized groups.[4]

Type III NSTI is the most fulminant type of NSTI, leading to multisystem organ failure within 24 hours if not properly recognized and treated. The offending organism in type III is not universally agreed upon. Some resources classify the bacteria as *Vibrio vulnificus*, whereas others refer to the *Clostridium* species.[4,13]

PATHOPHYSIOLOGY

Once the offending organism invades the subcutaneous tissues, the bacteria rapidly track subcutaneously, producing endo- and exotoxins, causing tissue ischemia, liquefactive necrosis, and systemic illness. Infection can spread as fast as 1 inch per hour with little overlying skin change.[4] Additionally, toxins cause endothelial damage, resulting in increased tissue edema, impaired capillary blood flow, phagocytosis, and neutrophil infiltration at the site of the infection and activation of the coagulation cascade, thus leading to vascular thrombosis and worsened tissue ischemia.[13]

CLINICAL PRESENTATION

NSTIs are difficult to recognize in the early stages. Early manifestations include swelling, erythema, and warmth, similar to those found in cellulitis. Pain that is out of proportion to physical examination and tenderness to palpation extending beyond and proximal to the area of skin involvement point toward the diagnosis of an NSTI. A clinician should maintain a high index of suspicion for NSTIs with these findings. Late signs of NSTIs include bullae, crepitus, or skin necrosis, which are associated with increased morbidity and mortality because of the delay in diagnosis (Figure).[13,14]

Earlier in the infection, NSTIs can be easily misdiagnosed as a muscle strain or a viral illness. Severe pain precedes skin changes by 24 to 48 hours. Patients also appear pale, lethargic, and febrile. Despite severe pain and ill appearance, some patients only present with mild erythema, cellulitis, or swelling overlying the affected area.[14]

DIAGNOSTIC STRATEGIES

A thorough history is paramount to the correct diagnosis of NSTI. In addition to the basic questions relating to skin infections (eg, minor trauma, soft tissue injury, insect or human bites, skin infections,