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Necrotizing Fasciitis

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Necrotizing Fasciitis

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Introduction

Necrotizing fasciitis also known as necrotizing soft tissue infections (NSTIs) is a rare life-threatening infection that involves the skin and soft tissue. A rapid and accurate diagnosis of NSTIs must be identified by healthcare providers to diminish morbidity and mortality. NSTIs are characterized by progressive necrosis of subcutaneous tissue and fascia involving large areas of tissue (Lin, Chang, Lai, Lin, & Chen, 2013).

Hippocrates (500 BC) described necrotizing fasciitis as "diffuse erysipelas caused by trivial accidents [where] flesh, sinews, and bones fell away in large quantities, [leading to] death in many cases" (Lancerotho, Tocco, Salmaso, Vindigni & Bassetto, 2012). In recent years the bacteria which causes this infection has been described by the media as "flesh eating", a term that remains synonymous with necrotizing fasciitis.

Pathophysiology

Pathogenesis

The pathogenesis of NSTIs is comprised of several micro-organisms including aerobic, anaerobic and mixed flora (Lin et al., 2013). Group A Streptococcus (GAS; Streptococcus pyogenes) is a primary contributor responsible for necrotizing fasciitis (Lin et al., 2013).

Category Types

Necrotizing soft tissue infections cases tend to occur as one of two broad clinical categories known as Type I and Type II with each type characterized by certain patient populations, clinical histories and presentations, and microbial etiologies (Shiroff, Herlitz & Gracias, 2012).

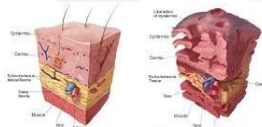
Type II NSTIs patients are usually young, generally healthy with a history of skin compromise often associated with extremity trauma (Shiroff, Herlitz & Gracias, 2012). Group A Beta-hemolytic streptococcus (Streptococcus pyogenes) is the most prevalent monomicrobial cause of Type II NSTIs (Shiroff, et al., 2011). A combination of Staphylococcus aureus or methicillin-resistant Staphylococcus aureus may be present with Streptococcus pyogenes in some cases (Shiroff et al., 2012).

Type I NSTIs patients demonstrate a significant contrast from Type II patients, as they are sicker and older with multiple medical problems, no obvious history of trauma, and a polymicrobial infection of the trunk or perineum (Shiroff et al., 2012). The pathophysiology of Type I NSTIs are polymicrobial with combinations of gram-negative bacilli, gram-positive cocci and anaerobes in most cases (Shiroff et al., 2012).

A more recent addition to the NSTIs categorization is the Type III NSTIs which are rare, exclusively gram-negative Vibrio species and confined to warm-coastal water regions in Asia, South and Central America (Shiroff, et al., 2012). Clinical presentation includes early and fulminant systemic shock and cardiovascular collapse which often precede the evolution of cutaneous signs and symptoms (Shiroff et al., 2012).



Group A Beta-hemolytic streptococcus (Imgarcade, 2014)



Normal Skin Necrotising Fasciitis (Microbewiki, 2010)

Case Study

Initial Presentation

An 84 year old male resident of an Extended Care Facility (ECF) arrived at a local Emergency Department (ED) with a complaint of a sore near the end of her nose. The area looked like a pimple with the surrounding tissue slightly swollen and black in color. Due to the area becoming more edematous and black, the patient was transferred to a second ED.

Evolving Clinical Presentation & Initial Treatment

Upon arrival, the ED staff was noted she was hypotensive and tachycardic, fluid resuscitation was initiated with normal saline. The "pimple-like" sore on her nose was becoming larger, more edematous and was now involving her face and neck. Triple antibiotic coverage was initiated for a possible diagnosis of necrotizing fasciitis. The patient was transported by helicopter to a tertiary care facility and admitted to the Intensive Care Unit (ICU).

Clinical Diagnosis

A diagnosis of necrotizing fasciitis was confirmed with an immediate surgical consultation to evaluate the patient for emergent surgical debridement of the infected tissue. Preparation for hyperbaric therapy was initiated. However, the extensive involvement of the infection was significant and the patient became too hemodynamically unstable to utilize hyperbaric therapy.

Outcome

The patient arrested from profound sepsis less than 24 hours after initial admission to the local ED. Resuscitation efforts were unsuccessful.

Clinical Presentation & Management

Signs & Symptoms

Signs and symptoms of NSTIs include severe pain, edema, blisters and erythema and a systemic inflammation syndrome (Friederichs et al., 2013). The histopathology findings include features of necrotizing fasciitis, vasculitis, and thrombosis of perforating veins (Friederichs et al., 2013). These infections can be polymicrobial or monomicrobial and caused by a variety of anaerobic and aerobic organisms (Shiroff et al., 2012). In 50%-80% of the cases the extremities are the involved site with the trunk and perineal region accounting for the rest of the potential locations (Friederichs et al., 2013).

Diagnostic Considerations

Patients presenting with NSTIs are considered some of the most difficult disease processes a healthcare provider is likely to encounter in the clinical setting (Shiroff et al., 2012). It is extremely challenging to determine the extent and severity of severe soft tissue infections (SSTIs) in the absence of obvious physical findings (Schwartz et al., 2013). It is even more difficult to identify which of the SSTIs will develop into NSTIs requiring immediate surgical intervention (Schwartz et al., 2013).

Physical signs such as crepitus, skin eschar, and/or bullae with evidence of soft tissue gas on radiologic study have a significant probability of NSTIs (Schwartz et al., 2013). Unfortunately, these findings are present in less than 50% of patients with a biopsy-proven NSTIs (Schwartz et al., 2013).

The difficulty associated with accurate diagnosis of NSTIs has led to multiple studies focused on physical, laboratory, and radiologic predictors in an attempt to improve diagnostic accuracy of NSTIs and therefore, diminish errors in the identification of the NSTIs that require immediate surgical intervention (Schwartz et al., 2013).

Treatment Considerations

Management of NSTIs requires rapid diagnosis and treatment (Schwartz et al., 2013). Patients diagnosed with NSTIs must receive immediate interventions focused on critical care support, antibiotic therapy, and aggressive surgical treatment (Friederichs et al., 2013). The physician and nurse practitioner (NP) are met with a difficult challenge in the diagnosis and treatment of NSTIs.

The differential diagnosis between a SSTI and a NSTI must be met with caution due to the lack of clear physical and diagnostic indicators to assist in the differentiation of these infections. NSTIs can be difficult to identify due to a misleading early presentation of the infection (Fodel & Smith, 2014).

Cardinal skin signs including erythema, edema and warmth require the physician and NP to consider NSTIs (Fodel & Smith, 2014). Determination of when to manage these patients medically versus surgically remains a dilemma for practitioners. The implications of an incorrect diagnosis can be devastating and life-threatening for the patient.

Conclusion

The Center for Disease Control and Prevention estimates between 500-1000 new cases of GAS necrotizing fasciitis occur annually in the United States and accounts for 6%-7% of all invasive GAS infections (Lin et al., 2013).

More than 2000 years have passed since Hippocrates first identified necrotizing fasciitis. However, mortality remains high (25%-35%) despite recent medical advancements (Wilson & Schneir, 2013). It is evident that the elusive challenge related to the prompt diagnosis and aggressive management of NSTIs continues to evade clinicians.

Further research is required to identify definitive indicators of NSTIs to improve the diagnosis and management of NSTIs and diminish the mortality and morbidity of this life-threatening disease process.



Post-surgical wound debridement (Medscape, 2010)



Pre-surgical wound debridement (JAMA, Dermatology, 2014)



Post-surgical wound debridement (John Hopkins University, 2010)



Post-surgical wound debridement (Deadly Microbes, 2014)

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