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Necrotizing Fasciitis: The "flesh eating" disease

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Introduction

Necrotizing fasciitis, often referred to as the "flesh-eating disease", is a rare bacterial infection with an extremely high mortality rate with symptoms that begin subtle but can quickly ravish the human body.1 While the prevalence of this disease is relatively low, evidence of this disease can be traced back as far as the 5th century BC where it was initially described by Hippocrates.2 It wasn't until 1952 however that Dr. Bob Wilson termed the disease "necrotizing fasciitis".3 The rapid progression of this disease and the acute deterioration it causes in a patient is extremely intriguing. This "flesh-eating disease" can present as an unassuming reddened area and manifest into a serious life threatening condition with a mortality rate close to 70% in a matter of hours if not properly identified and treated.4 The underlying bacteria that cause necrotizing fasciitis in an individual can consume or "eat" up to one inch of flesh every hour.5 Necrotizing fasciitis is reported in 4.3 infections for every 100,000 people worldwide.6 The overall prevalence of necrotizing fasciitis in the United States is also relatively low, with only 650-800 cases being reported each year. The disease has been reported higher in males versus females (2.6:1) and also seen more often in adults versus children.7 Prevalence of this disease however has increased nearly five fold over the past few decades which can most likely be related to a growing older population with increased comorbidities and predisposing risk factors, the most common of which being diabetes mellitus. Other risk factors predisposing an individual to necrotizing fasciitis are immune deficiencies such as AIDS, malignancies and complement C4 deficiency. Intravenous drug users and individuals with dermatological compromises such as psoriasis and skin breakdown are also at increased risk.7

Presentation

Necrotizing fasciitis can be caused by a variety of bacterial infections including Klebsiella, Clostridium, E. coli, Staphylococcus aureus, Aeromonas hydrophila, as well as the most commonly found cause, group A Streptococcus (GAS).1 While necrotizing fasciitis can develop anywhere on the body, development is most typically seen around the rectal, perianal and genital areas. Individuals predisposed to conditions and risk factors such as diabetes mellitus. chronic steroid use and advanced age put the individual at an increased risk.4 The destructive process of necrotizing fasciitis begins once the bacterium enters the subcutaneous tissue of the body. Any and all of these types of bacterium can enter through a variety of ways including a burn, laceration, insect bite, or even a minor scrape.1 The diagnosis of necrotizing fasciitis is often missed because the initial symptoms can be so subtle and is often mistaken for cellulitis.2 The initial diagnosis can also be masked because "the cutaneous manifestations of the disease are often very limited".8 Since the overall mortality of necrotizing fasciitis has been reported as high as 73%, diagnosis is of utmost importance so that immediate treatment may begin.7 Along with dermatological signs such as erythema, diagnosis of necrotizing fasciitis is often aided by

cultures and radiological studies such as computed tomography (CT). Since the presence of gas in the tissue beneath the epidermis is an important diagnostic finding, CT is an important radiological study.7,2

Signs & Symptoms

- Dull pain or soreness at site1
- Localized warmth, redness and/or swelling5
- Flu-like symtoms3
- Severe progressing local pain out of proportion to the size and physical presentation of site3,8
- Hard "wooden" feel to area upon palpitation3
- Leukocytosis with left shift upon CBC³
- Late:
- Formation of bullae and blisters8
- Numbness at site replacing pain indicating destruction of subcutaneous nerves5
- Crepitus as evidence of necrosis8
- Site redness changes to dusky blue-gray color5
- Metabolic acidosis8

Malnutrition

Paraplegia

leukaemia

Childbirth

ulcers

Rums

Advanced age

open fractures

- Anemia caused by large hemorrhagic bullae5
- Tachycardia, hypotension. tachypnea, hyper/hypothermia confusion and other associated

Table I. Predisposing signs of septic shocks ons and factors for necrotizing

Diabetes

Chronic renal failure

Peripheral vascular disease Malignancy

Immunosuppressive therapy

Chronic cardiac and pulmonary disease

Human immunodeficiency virus infection

Non-steroidal antiinflammatory drugs (NSAIDs)

Hypertension

Obesity

Muscle injuries From: Carter and Banwell, 2004; Hasham et al, 2005;

Predisposing conditions and factors for necrotizing fasciitis2

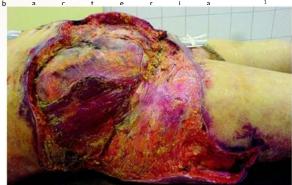




Preoperative findings of typical skin discolorations¹³

Pathophysiology

Once the bacteria have found entry into the body, typically through a break in the skin such as trauma, burn or insect bite, it releases pyrogenic exotoxin A. This exotoxin causes stimulation in the production of cytokines, which leads to extensive deterioration of the endothelial lining. Once the endothelial lining is damaged, fluid begins to permeate into the extravascular space resulting in profound diminished blood flow causing tissue hypoxia and ultimately leading to tissue death. 9 "As vasculitis and thrombosis occur in the adjacent tissues, further necrosis occurs involving the subcutaneous nerves". 10 The formation of thrombosis within small veins and arteries is the primary cause of overwhelming ischemia. Thrombosis must manifest in a significant number of dermal capillary beds before topical skin changes suggestive of widespread ischemia can be seen. 11 The resulting skin ischemia is the primary factor for the topical signs of warmth, redness and pain often seen in these individuals. It is important to note that before these dermal signs present, a significant amount of damage is being done by the infection at the fascia layer. If this disease is not interrupted by early diagnosis and treatment, toxins that are released into the individual's bloodstream lead to septicemia, multiple organ dysfunction syndrome and even death in as little as 24 to 96 hours after initial entry of the



Nursing Implications

Once necrotizing fasciitis in identified, aggressive treatment is required in order to reduce morbidity and mortality. 10 Initial nursing care for the patient presenting with necrotizing fasciitis revolves around treating the manifesting symptoms including pain. tachycardia, hypotension, and hypoxia until the patient can be taken to the operating room for debridement. The assessment of pain by the nurse is essential in evaluating the progression of necrotizing fasciitis. Excruciating pain is seen in the early stages and changes to numbness as the disease progresses to a more critical stage. 12 The goal of resuscitation is to establish oxygen delivery to the tissue and preserve tissue perfusion. Goaldirected therapy around hemodynamic parameters should mimic those suggested for treatment of sepsis by the Surviving Sepsis Campaign.11 Surgical debridement is typically required in most cases of necrotizing fasciitis. In actuality, most patients require multiple surgical interventions and debridement to ensure the necrotic tissue and accompanying infection is fully removed. Antibiotic administration as well as frequent dressing changes and monitoring of the wound are part of the postoperative care by nursing. Wound assessment by the nurse should include observation and documentation of the color, odor, and drainage of the wound. Any noted swelling or crepitus-like feeling could be indicative of gas formation under the subcutaneous tissue and a sign of remaining infection. 12

Conclusion

In conclusion, necrotizing fasciitis is a progressive and often life-threatening diagnosis that can be dated back to the "Father of Medicine" Hippocrates. With mortality as high as 73%, a diagnosis of necrotizing fasciitis can often be a grotesquely fatal one if not identified early and combatted with aggressive treatment.7 Survival from necrotizing fasciitis revolves around immediate diagnosis and aggressive resuscitation as well as antibiotic therapy and surgical debridement followed by continued monitoring and reevaluation by well trained healthcare providers.13

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