Necrotizing Fasciitis: The “flesh eating” disease

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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 ravage the human body. While the disease is relatively unknown, relatively little evidence of this disease can be treated back to as far as the 5th century BC where it was initially described by Hippocrates. It was first described in 1892 by Dr. Bob Wilson termed the disease “necrotizing fasciitis.” The rapid progression of this disease and the resulting mortality 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 sideline nears to 70% in a matter of hours if not properly identified and treated. The underlying bacteria that cause necrotizing fasciitis in an individual can consume or eat up to one inch of flesh very fast.[3]

Necrotizing fasciitis is reported in 4.2 infections for every 100,000 people worldwide.[4] The overall prevalence of necrotizing fasciitis in the United States is also relatively low. Between 1973 and 1985, only 68 cases were being reported each year.[5] The disease has been reported higher in males with a risk factor of 67% of developing complications versus 33% in females. Males are 3 times more often in adults versus children. The etiology of this disease however has increased nearly fourfold since the late 1970s, yet 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 C1 deficiency. Infectious drug users and individuals with dermatological compromising such as portwine and skin breakdown are also at increased risk.[6]

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). 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, cirrhosis due to end-stage and advanced age put the individual at an increased risk.[7]

The destructive process of necrotizing fasciitis begins once the bacteria enter the subcutaneous space of the body. Any and all of these types of bacteria can enter through a variety of ways including a burn, laceration, insect bite, or even a minor scrape.[8]

The diagnosis of necrotizing fasciitis is often missed because the initial symptoms can be so subtle and is often mistaken for cellulitis. The initial diagnosis can also be masked because the “cutaneous manifestations of the disease are often very limited.” Since the overall mortality of necrotizing fasciitis has been reported as high as 77%, diagnosis of is almost important so that immediate treatment may begin. The initial signs such as erythema, diagnosis of necrotizing fasciitis is often aided by sampling excised tissue from the affected area followed by Gram stain and culture.[9]

Table 1: Predisposing factors for necrotizing fasciitis

| Diabetes | Chronic renal failure | Alcohol abuse | Perianal/rectal disease | Malignancy | Immunosuppressive therapy | Chronic/boil infected or purulent disease | Prolonged immunosuppression | Wound infection, surgical site infection | Virus infection | Post-traumatic/inflicted/inflammatory drugs (opioids) | Hypertension | Obstructive jaundice | Burns | Fusospirochetal infections | Predisposing conditions and factors for necrotizing fasciitis |

Postmortem view after aggressive skin debridement

Necrotizing fasciitis located in deep fascia

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 simulation 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.[10] As vasculitis and thrombosis occur in the affected tissue, further serious occurs involving the subcutaneous layers.[11] The formation of thrombosis within small vessels and arteries is the primary cause of overwhelming ischemia. Thrombosis manifest in a significant number of different cell death pathway before topical skin changes suggestive of widespread ischemia can be seen.[12] 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 bacteria.[13]

Preoperative findings of typical skin discoloration[14]

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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 70%, a diagnosis of necrotizing fasciitis can often be a rapidly fatal if not identified early and combated with aggressive treatment.[15] Survival from necrotizing fasciitis revolves around immediate diagnosis and aggressive resuscitation as well as antibiotic therapy and surgical debridement followed by continued monitoring and rehabilitation to help heal trained healthcare providers.[16]