CHAPTER **13**

NECROTIZING FASCIITIS

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Necrotizing fasciitis is a rapidly progressive disease entity that, although relatively uncommon, is extremely important for podiatric surgeons to recognize as the majority of these infections affect the lower extremities. The process is characterized by necrosis of the skin and its associated structures including subcutaneous tissues and the underlying fascia. A life threatening infection that has been documented for centuries, necrotizing fasciitis has been known under many names. During the Civil War it was known as "Hospital Gangrene",[1] it is also known as phagedena, phagedena gangrenosum, progressive bacterial synergistic gangrene (Meleney's gangrene), Fournier's gangrene, necrotizing erysipelas, gangrenous erysipelas, and hemolytic streptococcal gangrene. In 1952 Wilson's term Necrotizing Fasciitis was finally adopted as it fully describes the features of the disease.[2]

Cases of necrotizing fasciitis in the United States number 500 – 1500 annually.[3] The disease progresses rapidly and can mimic more common soft tissue infections. The lack of early cutaneous signs and symptoms can make for a challenging diagnosis. Prognosis is dependant upon the accurate diagnosis and timely initiation of appropriate treatment modalities.

PREDISPOSING FACTORS

This process often is precipitated by trauma, including surgery and other soft tissue injury. Portals of entry vary and may be distant to the actual expression of the disease process. Pre-existing skin conditions such as psoriasis, decubitus ulcerations or dermatitis may also provide the nidus for the spread of this soft tissue condition.[4]

Immunosuppression caused by diabetes mellitus, AIDS or complement deficiency may be predisposing conditions but the degree to which they contribute is not well defined.[5]

Smoking, peripheral vascular disease and the use of illicit drugs, specifically IV drug abuse and "skin popping" are also factors in necrotizing fasciitis. Recent Varicella infection and advanced age also appear to be factorial.[6]

NSAIDS have been implicated in several cases of group A streptococcal severe invasive infections.[7] They impair granulocyte function, augment inflammatory cytokine release, and they inhibit renal prostaglandin release. Suppression of fever and pain may also mask the manifestations of the disease process. One animal model study refutes this and suggests that the administration of NSAIDs (diclofenac) does not increase the extent of tissue damage.[8]

CLINICAL PRESENTATION

Patients usually present to the emergency department acutely ill. Physical findings include increases pain over the affected area and skin changes that include blistering, non-pitting edema, induration beyond the area of erythema, crepitus, and usually the absence of lymphangitis. Myofascial planes are usually not discernable by palpation secondary to the induration in contrast to cellulitis.

Presentations can be categorized as early, intermediate and late.[9] Early presentation exhibits high fever, tachycardia, stable blood pressure and normal sensation. Cutaneous signs and symptoms include erythema, blisters, indurated shiny skin that extends beyond the area of erythema and severe pain on palpation. Intermediate presentation appears similar with worsening clinical findings: larger area of skin involvement, larger and more numerous blisters/bullae and mild loss of sensation. These bullae often resemble fracture blisters and are filled with a reddish-brown fluid.[10] Late presenters have high fever extreme elevations in white blood cell counts (greater than 25,000) and the classic skin presentation of necrotizing fasciitis: edema with central patches of dusky blue discoloration, weeping blisters and border cellulitis with advanced cutaneous anesthesia, the hallmark of the disease.[11] They also will be showing the signs of systemic sepsis, shock, multisystem failure, and possible unconsciousness.[9]

Laboratory tests on admission often indicated the extreme systemic consequences of the disease. Along with the elevated white cell count and the associated left shift will come significant anemia secondary to the large amounts of blood loss from small vessel thrombosis, subcutaneous bleeding and hyperemia. Acute jaundice may occur as a result of hemolysis, hypocalcemia,

hypergammaglobulinemia, hypoalbuminemia and acidosis may also present in the late stages.[12]

In a study by Wong et al peripheral locations of necrotizing infections accounted for 80% of reported cases. Of those, the lower extremity was involved in 70% of the cases. In light of these figures it is essential that, when presented with lower extremity infections, necrotizing fasciitis be high on the list of differential diagnoses.

DIFFERENTIAL DIAGNOSIS

Due to the high degree of mortality associated with necrotizing fasciitis, rapid and accurate diagnosis are essential. Mortality rates have been improved since the discovery of the disease, however the reported range of mortality rates is still a cumulative 34% (range 6% to 76%).[13] The 12 to 48 hours after admission are the most critical. The majority of reported deaths occurred within this time period. Differentiation of necrotizing fasciitis from less aggressive disease entities is imperative.

The criteria for diagnosis of necrotizing fasciitis include: 1) extensive necrosis of the superficial fascia with undermining of surrounding tissues; 2) moderate to severe systemic toxic reaction with altered mental status; 3) absence of muscle involvement; 4) absence of clostridia as the predominant bacterium in cultures; 5) absence of major vascular occlusion; and 6) pathologic examination of debrided tissue, showing intense leukocytic infiltration, thrombosis of the microvasculature, and focal necrosis of fascia and surrounding tissues.[9, 10]

Due to the paucity of signs and symptoms in the early stages of the disease, diagnostic techniques that will aid in the rapid diagnosis are paramount. Direct needle aspiration for gram stain and culture has been espoused[14] as well as the "Finger Test" for the work-up of early and intermediate presenting patients. This test is performed in the following manner: the area of suspected involvement is infiltrated with local anesthesia. A 2 cm incision is made in the skin down to the deep fascia. Lack of bleeding is an ominous sign of necrotizing processes. "Murky dishwater fluid" has been noted in the wound, as well as gray stringy subcutaneous tissues. Gentle probing with the index finger is performed along the deep fascia. Tissue dissection with minimal to no resistance is a positive finger test.[9] Frozen section biopsy is also useful for early diagnosis and may be employed at the time of the finger test.[15]

MICROBIOLOGY

A wide variety of causative organisms have been reported throughout the literature. Hemolytic streptococci and staphylococci are found in most cases and may be commonly mixed with enterococci. Pessa et al in 1985 showed 75 organisms isolated from 16 patients with necrotizing fasciitis.[16] Anaerobic organisms are also recovered from these wounds. A classification system based on culture results was presented by Guiliano et al Type I patients are those with anaerobic and facultative anaerobic bacteria in combination, whereas type II patients exhibit group A streptococci alone or in combination with staphylococci and no anaerobes.[10] As with all infections, cultures from deep tissues are the most reliable as superficial cultures may be contaminated and/or negative.

TREATMENT

Stabilization of the patient's medical condition is imperative when treating necrotizing fasciitis. Until incision and drainage is performed this may prove to be a difficult task, requiring fluid and electrolyte repletion and possible transfusion of blood products. There is no substitute for swift decompression of the affected areas as this has been shown to dramatically reduce the mortality rate of this disease.[12]

Surgical approach should include multiple linear incisions as needed to evacuate and debride the necrotic tissues. Gentle probing as is done in the finger test should be performed to elicit the extent of the infection/necrosis. Necrosis may be extensive and extend beyond the skin manifestations. The entire circumference of the limb may be affected. Incisions need to be lengthened until healthy tissues are encountered. All necrotic tissues need be debrided thoroughly and copious irrigation should be employed (Figures 1-3). Wounds should be packed open and inspected within 12 to 24 hours. If inadequate debridement occurs, repeat OR debridement will be required to eradicate all infected and necrotic tissues.

A treatment regimen was developed by Sudarsky et al. which is comprised of the following steps: 1) antibiotics, 2) surgical debridement, 3) re-exploration 24 hours after surgery, 4) nutritional support, and 5) early soft tissue coverage as needed.[12]

The massive tissue loss from the radical debridements may result in the need for creative means of soft tissue coverage. Skin grafting over granulating tissues is an appropriate means of coverage, however in cases involving

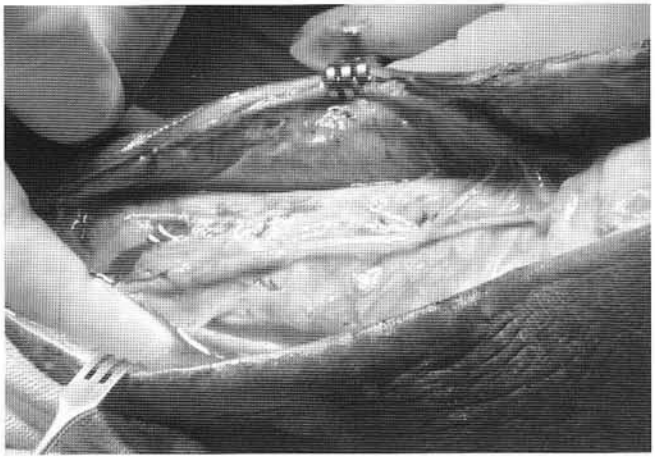

Figure 1. Anterior right leg dissection to deep fascia, exposing superficial peroneal nerve (proximal-left, distal-right

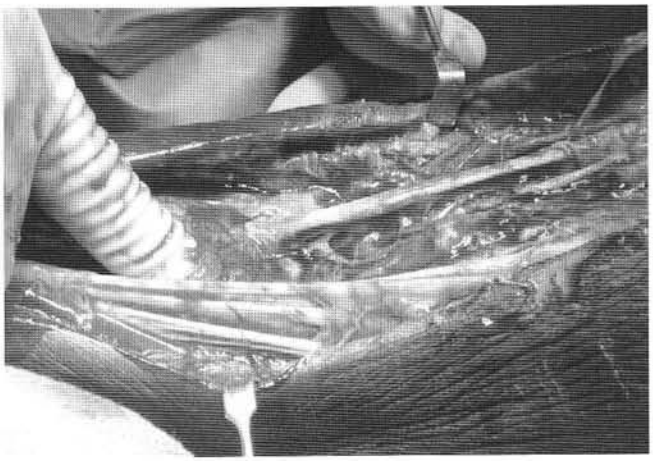

Figure 2. Dissection deep to deep fascia dorsum right foot.

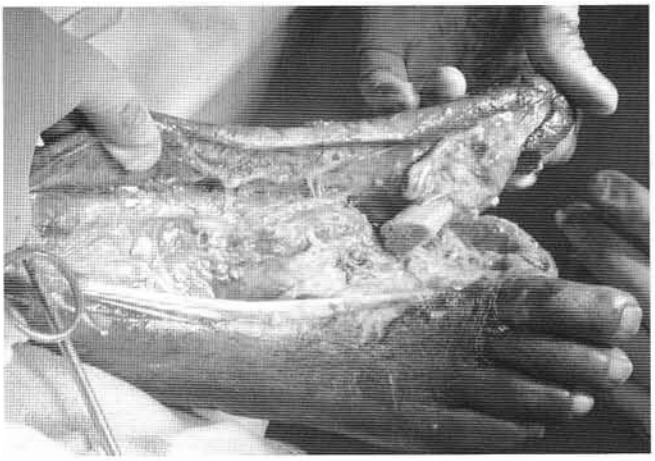

Figure 3. Further debridement right foot, including partial resection of first ray where plantar ulceration introduced the necrotizing infection.

large areas of tissue loss grafting may not be sufficient. A technique described by Mahan et al utilizes abdominal closure strips to gradually achieve re-approximation of the skin margins. This allows for delayed primary closure rather than skin grafting for wound closure. Non-reactive monofilament suture is recommended as well as closed suction drainage of the wound for the first 24-48 hours post closure.

Antibiotic coverage is imperative and should be broad spectrum to cover all possible infectious organisms until definitive OR culture results are available. Multiple agent approaches have been advocated in the literature and usually include an aminoglycoside, clindamycin, and a penicillin/cephalosporin.[16] Modifications to antibiotic coverage can be made after susceptibilities are performed.

Hyperbaric oxygen therapy has also been advocated as an adjunctive therapy in the treatment of necrotizing fasciitis. It has only been shown to be beneficial for early wound closure.[17]

SUMMARY

Necrotizing fasciitis, although uncommon, is associated with a high rate of mortality and significant morbidity due to its' rapidly progressive nature. Timely diagnosis and aggressive treatment and are essential for optimal outcomes for these patients. As many of these infections occur in the lower extremities it becomes imperative that Podiatric physicians have the diagnostic tools and a high index of suspicion for necrotizing fasciitis when presented with the hallmark signs and symptoms as described herein.

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