Review Article

Massive infectious soft tissue injury: diagnosis and management of necrotizing fasciitis

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INTRODUCTION

Necrotizing fasciitis is a destructive invasive infection of the skin, subcutaneous tissue and deep fascia with relative sparing of muscle. Streptococcal gangrene, progressive bacterial synergistic gangrene (Meleney's gangrene)[1], Fournier's gangrene and synergistic narcotizing cellulitis all present with extensive skin and soft tissue destruction requiring wide debridement, long term antibiotic therapy and treatment of multi system dysfunction and eventual reconstruction. All these processes have unique characteristics but are commonly considered variations of necrotizing fasciitis. Necrotizing infections, although easily diagnosed, usually require operative debridement in conjunction with antibiotic therapy.

Understanding the natural history and unique characteristics of these processes is essential for effective surgical management and favorable outcome. A comprehensive review of the literature pertaining to this condition is presented outlining the pathophysiology, pattern of presentation and the treatment strategies necessary for successful management of this massive infectious soft tissue disease.

PATHOPHYSIOLOGY

Although the pathogenesis of necrotizing fasciitis is still not completely understood, the rapid and destructive clinical course of necrotizing fasciitis is

believed to be the result of multilateral symbiosis and synergy [2]. Group -A beta hemolytic streptococcus has been considered the common generator of necrotizing fasciitis[3]. These mono-microbial infections usually affect the extremities, with nearly two thirds of cases in the lower extremities[4], although cases involving the face have been described. In most cases an underlying cause such as diabetes, uremia, immunodeficiency or other chronic illness is present. A polymicrobial synergistic pathogenesis has recently suggested being more common[5-8]. Polymicrobial infections are commonly associated with previous surgical procedures, penetrating trauma, decubitus ulcers, perianal abscesses, intravenous drug abuse, and Bartholin's or other vulvovaginal abscesses[4]. Other identified organisms include Staphylococcus aureus, E.coli, Pseudomonas, Enterobacter, klebsiella, Proteus, Bacteroids, Clostridium and Peptostreptococcus. Seal[9] and Kingston[10]previously demonstrated synergism between beta hemolytic streptococci and other organisms such as Staph aureus. This synergistic action of multiple bacteria is well known in experimental and natural infections, and it is likely that this process plays a role in the pathogenesis[8].

CLINICAL PRESENTATION

Early, intermediate, and late presentations may be present, and may evolve over a period of several hours to days. Early presentation is one of high fever, tachycardia, stable blood pressure and normal sensorium. The involved skin reveals erythema, blisters and intense pain on palpation. The intermediate presentation is similar but involves a worsening clinical picture with a larger area of skin involvement, increasing number and size of skin blisters and mild disturbance in sensorium. Patients presenting late have high fever, white blood cell counts greater than 25,000, classic skin findings of necrotizing fasciitis, systemic sepsis, shock, multisystem failure and unconsciousness. A patient may progress through these stages with alarming rapidity. Rectal and perineal examinations are mandatory. Appropriate laboratory work up includes blood counts with differential, blood chemistries, arterial blood gases and tissue and blood cultures. Radiographic studies may be done to identify soft tissue air. Computerized tomography scans

may have utility for difficult areas such as the abdominal wall, perineum and neck[9]. The " Finger test" and rapid frozen section biopsy examinations have been used as complementary steps in the work up of patients presenting in the early or intermediate stages[9]. The finger test is performed in the following manner: The area of suspected involvement is infiltrated with local anesthesia. A two cm incision is made in the skin down to the deep fascia. Lack of bleeding is an ominous sign of a necrotizing process. A gentle probing maneuver with the index finger is performed at the level of the deep fascia. If the tissues dissect with minimal resistance, the finger test is positive. Tissue biopsies can be sent for frozen section. If the finger test or the rapid frozen section is positive or if the patient has progressive clinical findings consistent with necrotizing fasciitis, the patient should be resuscitated and taken to the operating room emergently for debridement.

DIAGNOSIS

The presumptive diagnosis must be made clinically. Rapid diagnosis and treatment have led to increased survival in many studies[1,10,11,12,14]. In many cases, an identifiable antecedent trauma is present. The initial lesion is often trivial such as a minor abrasion, insect bite, injection site or boil. It is important to note however that this is not an absolutely necessary element for diagnosis. Particular historical predisposing factors for acquiring necrotizing fasciitis such as smoking, diabetes, peripheral vascular disease, immunologic compromise and intravenous drug abuse should be noted[9,10]. Particularly important physical findings are increased pain over an area with associated skin changes. Other signs include edema extending beyond the area of erythema, skin vesicles, crepitus and absence of lymphangitis. In most cases the subcutaneous tissues will demonstrate a wooden hard feel, which may help to distinguish the infection from simple cellulitis. In fasciitis, the facial planes and muscle groups cannot be discerned by palpation as they can in cellulitis [4].

MANAGEMENT

Necrotizing fasciitis is a surgical emergency .The large amount of necrotic tissue fuels a persistent septic state. When possible, aggressive resuscitative efforts should be made. One may not be able to completely stabilize the patient before surgery because the delay may lead to a fatal outcome. Surgery is the primary treatment, wide debridement of all obviously necrotic and poorly perfused tissues leads to more rapid overall clinical improvement. Fillet type incisions have been recommended in the past [4] and there has been controversy regarding how much tissue should be initially resected because the skin may often appear normal. Normal appearing tissues examined microscopically revealed extensive early vascular thrombosis and vasculitis, suggesting a high potential for full thickness loss. Therefore, wide extensive debridement of all tissues that can be easily elevated off of the deep fascia with gentle finger dissection is recommended. The wound must be inspected closely after the initial debridement. Hemodynamic instability usually persists postoperatively and progressive skin necrosis may occur from infectious spread or hypoperfusion. Affected patients must have further debridement [1,13]. The Allograft or xenograft skin can be used for wound closure until the patient is satisfactorily stabilized for reconstruction. Temporary closure helps to minimize the fluid and protein loss, catabolism and infection associated with massive open wounds.

Broad-spectrum antibiotic coverage is recommended due to high incidence of polymicrobial infection. A combination of Pencillin G, an aminoglycoside (if renal function permits) and clindamycin to cover streptococci, staphylococci, gram negative bacilli and anerobes is a reasonable choice.

Hyperbaric oxygen in the treatment of necrotizing fasciitis has been debated [16, 17], however, studies show a trend toward increasing survival. The standard treatment protocol is 20 treatments of 90 minutes each at 2 atm of total pressure. Hyperoxemia allows for more efficient leukocyte function by providing more substrate for formation of free radicals and by augmenting respiratory burst. The effects of hyperbaric oxygen also include increased fibroblast growth, inhibition of

bacterial toxin formation, increased red cell pliability and reduction of tissue edema. Hyperoxia also causes increased neovascularisation, which may lead to improved delivery of antibiotics to the infected areas[19]. The literature seems to support the use of hyperbaric oxygen as an adjunctive treatment measure.

Nutritional support is also an integral part of treatment. Supplementation should begin as soon as hemodynamic stability is achieved and enteral feedings should be established to offset the catabolism associated with large open wounds. The administration of excess protein and calories is necessary to promote healing[20].

Once all affected tissues have been debrided and the patient has been stabilized, soft tissue reconstruction of patients can be considered. Traditionally reconstruction has been performed with skin grafts and flaps. In cases with large amounts of soft tissue involvement, auto graft reconstruction maybe limited by donor site availability or questionable underlying wound bed viability. Integra artificial skin is an alternative to standard skin graft. This product is a bilayered membrane consisting of an inner dermal substitute composed of bovine tendon and glycosaminoglycan plus an outer silicone epidermal layer. Infiltration of host cells and capillary growth occurs within the dermal matrix, forming a "neodermis". In 14 – 21 days the patient is returned to the operating room for removal of the silicone layer and auto grafting. AlloDerm is a dermal transplant obtained from allograft skin and processed into a cellular matrix, which retains normal extra cellular ultra structure. Autograft epidermis can be placed over this matrix, either immediately or in a delayed fashion. These products may allow for earlier wound closure and reduced catabolic insult. Necrotizing fasciitis is a devastating but survivable illness. An aggressive surgical approach is necessary in the treatment and reconstruction.

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