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Diagnosis and Management of Necrotizing Fasciitis of the Head and Neck

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Abstract Necrotizing fasciitis is a rapidly progressing and life-threatening soft tissue infection that often affects the abdominal wall, perineum, or extremities following surgery or trauma. It predominantly occurs in elderly and immunocompromised patients. It is rarely seen in the head and neck region. Necrotizing fasciitis of the head and neck carries high rates of morbidity and mortality. Symptoms usually develop quickly and well-timed diagnosis is critical to optimizing outcome. Diagnosis is based on a combination of clinical history, Gram staining and culture, imaging and surgical exploration. Early and aggressive surgical management and urgent parenteral antibiotic therapy are critical to optimizing outcome.

Keywords Necrotizing fasciitis · Head and neck · Diagnosis · Treatment

Introduction

Necrotizing fasciitis (NF) is a progressive, potentially fatal bacterial infection of the skin, the subcutaneous tissue and the underlying fascia. This uncommon disease generally occurs in the lower extremities, abdominal wall and perineum, usually secondary to surgery or trauma [1–3]. It rarely affects the head and neck region [4, 5•]. It may result from a superficial infection associated with a skin injury or folliculitis, or from a deep soft-tissue infection such as pharyngitis, tonsillitis, or a dental infection that spreads along the deep facial planes.

NF is a poly-microbial infection, including Streptococcus and Staphylococcus species, enteric bacilli and anaerobic organisms [6, 7•]. It predominantly occurs in elderly and immunocompromised patients. Diabetes mellitus, burns and malnutrition are common predisposing factors [8].

History

NF was first described during the American Civil War by Joseph Jones, who reported cases of "hospital gangrene" that were characterized by skin discoloration and a loss of superficial and deep fascia [9]. In 1918 Pfanner described a patient with beta hemolytic streptococcal infection, and he named the process necrotizing erysipelase [10]. Meleney accurately described this infection, based on 20 patients, and termed it "hemolytic streptococcus gangrene" [11]. The term NF was first used by Wilson in 1952 in describing cases with similar presentations that were caused by staphylococcal infections [12].

Classification of NF

NF is generally classified into 2 types on the basis of the organisms present in the culture. Type 1 NF is caused by a polymicrobial infection consisting of both aerobic and anaerobic bacteria. Type 2 NF is identified by existence of Streptococcus pyogenes alone or, in combination with

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Staphylococcus species [13]. A more recent classification suggesting 4 major NF types also exists [14•].

Etiology: Microbiology

Polymicrobial infections (Type 1 NF) are composed of non-group-A streptococci, aerobic organisms, anaerobic organisms like Clostridium and Bacteroides and enteric bacteria, including Escherichia coli and Enterobacter cloacae, Klebsiella pneumoniae, Pseudomonas and Vibrio species. However, Type 2 NF is caused by Streptococcus pyogenes alone or, in combination with Staphylococcus species [13, 15]. Benavides reported an uncommon type-I polymicrobial infection with Klebsiella pneumoniae and Acinetobacter baumannii, which developed out of a small pustule on the patient's right cheek within 24 h [16].

Predisposing Conditions

A total of 46% of patients have at least one debilitating condition [17]. The predisposing factors for NF include prior trauma, immunosupression, HIV, diabetes mellitus, arteriosclerosis, alcoholism, chronic renal failure, malignancy, intravenous drug abuse and the postpartum state [5•, 7•]. Besides, two cases of NF associated with radiotherapy have been cited in the literature [18].

Pathophysiology

The pathogenesis of NF has been studied by numerous investigators, but the exact mechanism of this rapidly spreading gangrenous infection has not been established. Once infectious organisms breach the protective barrier of skin, rapid tissue destruction is possible. The release of enzymes, such as hyaluronidase and proteolytic portions of cell membranes, have been shown to be contributing factors in the necrosis. After sensing the release of bacterial chemicals, the immune system goes into overdrive, mounting an exaggerated response. The area blood vessels, which dilate, thus distributing immune components and removing toxins, begin to leak. This decreases the actual flow of blood and oxygen. Lack of oxygen causes cell death. As this process progresses, inducing increasing amounts of ischemia, the vessels develop a thrombosis, leading to a greater increase in damage [19].

Clinical Presentation

NF is rarely seen in the head and neck region. Head and neck NF is further classified into: Craniofacial NF (above the lower rim of the mandible) and Cervical NF (below the lower rim of the mandible [5•]. The most common cause of NF of the head and neck is dental infection from the mandibular molar teeth. Other frequent causes reported in the literature are trauma, peritonsillar abscess, laceration, abrasion, surgery, burns and insect bites [20•, 21•]. The infection process usually begins 48 to 96 h after an initial insult. The first symptom of NF is characteristically intense local pain, although the affected area may initially have a benign appearance. Initial symptoms may also include flu-like symptoms, thirst, diarrhea and gastric distress. At first, the skin becomes inflamed, smooth, tense and shiny with no demarcation line between normal and infected skin. Pain can be greater than expected from clinical findings. Crepitation may be elicited at this early stage although it is usually a late clinical sign. As the disease progresses the skin becomes dusky, appearing as a small, purplish-blue patch with irregular and ill-defined borders, which is characteristic of NF. At the same time, blisters or bullae may appear. Skin necrosis is secondary to thrombosis of nutrient vessels passing through the necrotic fascia. Low-grade fever, leukocystosis, anemia, and/or jaundice and tissue crepitation secondary to gas formation may be the accompanying clinical findings. The patient becomes systemically unwell, develops regional lymphadenopathy and may develop shock. Metabolic changes such as hyponatremia, hypoproteinemia, hypocalcemia, and dehydration may also be seen in severe cases [22•, 23•].

The progression of NF is usually rapid. It may even spread to the mediastinum and chest wall with a poor prognosis [23•]. Potential complications include airway obstruction, arterial erosion and occlusion, jugular vein thrombophlebilitis, mediastinitis, pneumonia, septic shock, lung abscess, mandible necrosis, pleural and pericardial effusion [20•, 24]. Systemic illnesses, such as diabetes, severe kidney, hearth and lung diseases, HIV, vascular insufficiency, malnutrition and obesity, suppress the host immunity and can thus predispose to this fulminant infection [25].

Diagnosis

Delayed diagnosis in NF is directly associated with increased morbidity and mortality. Therefore, a presumptive diagnosis and rapid treatment is necessary for optimal outcomes. Early signs and symptoms may be confusing, and the disease is often misdiagnosed as cellulitis or erysipelas. A variety of symptoms should alert the clinician, such as the rapid progression of the infection, abnormal gas accumulation in the soft tissues, and the occurrence of systemic toxicity, especially in patients with contributing factors [26, 27•].

Laboratory tests usually show a leukocytosis with left shift and an increased C-reactive protein level as expression of inflammation. Further abnormal laboratory findings may be hypoalbuminemia, elevation of liver enzymes and blood urea nitrogen levels. Hypocalcemia may occur as a consequence of calcium precipitation in areas of extensive fat necrosis [7•].

Computed tomography (CT) and magnetic resonance imaging (MRI) can play important role in the diagnosis of NF and help to differentiate from cellulitis [7•]. Becker et al. investigated 14 cases of NF of the head and neck and identified specific findings on CT such as diffuse thickening and enhancement of subcutaneous fat, cervical fascia and chronic infection in muscles [28]. They reported that gas and fluid within the soft tissues were present in 9 (64%) of their patients. The treatment should be based on both the clinical findings and imaging.

A delay in obtaining the imaging studies should not postpone surgical exploration and debridement especially if the infection is rapidly progressive. In this case, an incisional biopsy should be considered. At the bedside, suspected area is locally anaesthetized and a 1 to 2 cm incision is made through the skin and platysma to the level of deep cervical fascia. If no bleeding is present or if a murky dishwater discharge is observed, NF should be highly suspected. When a finger is inserted and pressure is applied to the subcutaneous tissue, if minimal resistance is felt and easy separation of tissue planes is observed, the test is positive and NF is indicated [29].

To ascertain the definitive diagnosis, biopsies must be taken during surgery. Histologically, early lesions show superficial epidermal hyaline necrosis, dermal edema and hemorrhage, but no inflammatory cells, bacteria or tissue necrosis. Advanced lesions of NF include tissue necrosis, collagen fragmentation, intravascular thrombosis of the vessels coursing through the fascia, inflammatory granulocytic infiltration of the deep dermis and fascia as well as bacterial invasion with gram-positive cocci [30, 31].

The tissue samples obtained during the surgery should be send for microbiological analysis and histology. To diagnose the microbiologic etiology adequate cultures should be taken from the body fluid samples such as blood and pus [5•]. Samples should be sent using transport media adequate for recovery of anaerobic bacteria. Organisms are best identified by taking cultures obtained at the edges of the involved area, not from the center with necrosis [33]. During the course of treatment, samples of the drained fluid should be periodically sent for microbiological analysis and the antibiotic therapy is modified accordingly.

Management

Management of NF is based on 5 basic principles: early diagnosis and debridement, broad spectrum antibiotics, aggressive resuscitation, repeated reevaluation and aggressive nutritional support [23•, 27•, 32]. The management should involve a multidisciplinary team comprising the otorhinolaryngologist, anaesthetist, microbiologist, plastic surgeon and cardiothoracic surgeon as appropriate to the case.

The initial surgical excision and debridement helps in halting the spread of the necrotizing process and thus stop the release of inflammatory mediators responsible for systemic complications [23•]. A delay in appropriately aggressive surgery has been associated with a high mortality rate [21•]. Immediate airway control is essential when there is extensive cervical involvement. The authors suggested strong consideration of early tracheotomy in patients with extensive disease [7•, 27•].

The principle of using antimicrobials effective against aerobic and anaerobic bacteria is the mainstay of antimicrobial choice. Antimicrobial coverage for MRSA may be needed in instances where this organism is present or suspected.

The use of antibiotics in NF is not standardized. It is prudent to use an aggressive antibiotic therapy to prevent complications such as septic shock, even though surgical therapy is of primary importance. The selection of antibiotics varies from country to country and according to the presence of resistant strains [5•]. A broad spectrum antibiotic treatment must consist of penicillin 6 x 5 millions IE, clindamycin 3 x 600 mg and metronidazol 2 x 500 mg [33]. However, some authors start empirical treatment with a combination of third generation cephalosporin, penicillin or cloxacillin or prefer to start with imipenem [5•, 34]. After microbiological analysis, antibiotics should be prescribed according to culture and sensitivity. The duration of hospitalization of the patient with NF is relatively long. Patients with NF must undergo antibiotic treatment during the hospitalization. Duration of antiobiotic treatment is approximately 20–25 days long [21•, 34].

Rigorous daily wound care is essential after surgical debridements to facilitate further removal of compromised tissue. A twice-daily regimen of wound irrigation and wet to dry dressing changes are recommended [27•]. We prefer rifampicine and nitrofurazone soaked gauze for dressing [22•]. Reconstruction should be delayed until infection resolution is well established.

There are other adjunctive therapy modalities including HBO therapy and intravenous poly-specific immunoglobulin therapy in addition to surgical and antibiotic therapy. Addition of intravenous immunoglobulin has reduced the mortality in patients with severe Group A Streptococci infections [35]. The effect of immunoglobulin is probably an inhibition of the super antigen activity related to exotoxins secreted by Group A Streptococci. HBO is another clinically useful adjunctive treatment for NF. HBO reinstates the defense against infection by increasing free

radicals, which helps neutrophil-mediated killing of some common bacteria [36]. In addition, HBO therapy acts as a bactericide for certain anaerobes. HBO reduces the mortality, shortens the duration of hospitalization and decreases the required number of surgical debridements [37, 38].

Conclusions

NF of the head and neck is a rare but potentially fatal disease. Early diagnosis with prompt and aggressive treatment is critical to reducing the associated morbidity and mortality. Surgical debridement and appropriate antibiotic therapy with supportive therapy must be started immediately. A high mortality rate is still noted, despite aggressive management.

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