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CASE REPORT

Necrotizing fasciitis of the face: the flesh-eating catastrophic malady

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Necrotizing fasciitis (NF) is a rare, rapidly progressing, life-threatening infection involving the superficial fat, fascial layers with necrosis of skin. It is usually seen in the extremities, abdominal wall, and perineum and predominantly seen in elderly and immunocompromised patients. NF of the face and cervical area is usually very rare and if it occurs it is characterized by its fulminating, devastating and rapidly progressive course. Untreated facial NF may lead to complications such as airway obstruction, vascular thrombosis, mediastinitis, pleural empyema, large vessel thrombosis and septic shock. Management of NF requires a prompt and accurate diagnosis, emergency airway management, aggressive surgical debridement, intravenous antibiotics and nutritional support. We have described a rare case of necrotizing fasciitis of the face which has high morbidity and mortality rates, posing challenging reconstructive problems and a brief review of literature.

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Introduction

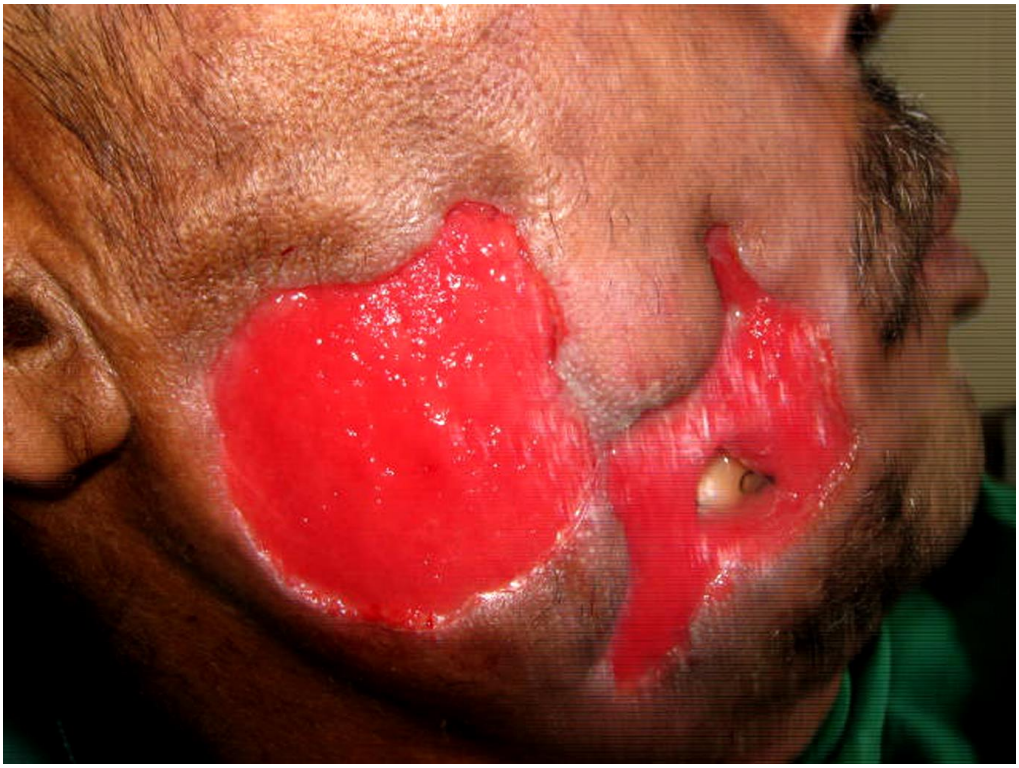
Necrotizing fasciitis (NF) was first described in 5th century BC by Hippocrates who depicted this as a complication of erysipelas.[1-2] In 1871, Joseph Jones, a confederate army surgeon observed cases of necrotizing fasciitis during the American civil war and termed it as hospital gangrene.[1-3] Pfanner in 1918 described this condition as necrotizing erysipelas[1] but the term NF was coined by Wilson in 1952.[1-3] NF is a rare rapidly progressing & potentially fatal infection of subcutaneous soft tissue characterized by extensive necrosis of fascial planes and the overlying skin.[2-5]

NF in the head and neck region is a rare occurrence especially in the facial region and in an immunocompetent patient.[4,6] Overall incidence is estimated in 3.5 cases per 100,000 persons, with a mortality rate as high as 80% without an early medical or surgical intervention.[2] NF is most commonly encountered in the extremities, abdominal wall, and perineum and is predominantly in the elderly and immunocompromised.[2-4,7] Successful management of facial NF requires early diagnosis, prompt institution of intravenous broad spectrum antibiotics, aggressive surgical debridement to control the infection, and reconstruction of the resultant soft tissue defects.

Case Report

A 34-year-old male patient reported to the Department of Oral and Maxillofacial surgery, SDM Craniofacial surgery and research Centre, Dharwad, Karnataka, India with a chief complaint of pain in the right back region of the jaw for one month. He did not have any relevant medical and drug history. Systemic examination revealed that he was poorly built and nourished and was anemic. Extra oral examination revealed complete skin loss on the right side of the lower face with exposure of the mandible and discharging pus (Fig 1). The submandibular lymph nodes were palpable, soft and tender on the right side. There was complete restriction of mouth opening. He was diagnosed to have necrotizing fasciitis of the face. He underwent drainage of the pus and debridement under general anesthesia with Intravenous empirical antibiotics such as Amoxycillin and clavulanic acid 1.2gm twice a day, Metronidazole 500mg three times a day and adequate hydration. The culture and sensitivity report which was obtained after 72 hours revealed Enterococcus species which was sensitive to Injection Linezolid 600gm and hence given accordingly two times per day. He also received non-steroidal anti-inflammatory drugs for pain relief and dressing of the wound was done twice per day. The patient was planned for secondary reconstruction of the face after the infection subsided. He denied further treatment due to financial constraints and was discharged against medical advice. He has been lost for any further follow up.

Figure 1 Extra oral Photograph showing complete skin loss with exposure of the mandible.



Discussion

NF is a rapidly advancing, suppurative infection that causes extensive necrosis of the fascia and subcutaneous tissues, often associated with vascular thrombosis and necrosis of the overlying skin.[2-3] A variety of synonyms have been used for NF including streptococcal gangrene, gangrenous erysipelas, necrotizing cellulites' and Meleney gangrene.[3] The most common site of facial NF is the periorbital area. The infection involves the superficial fascial planes of the head and neck, i.e., the superficial musculoaponeurotic system.[2]

The predisposing factors for NF include advanced age, blunt or penetrating trauma, burns, chronic alcohol abuse, diabetes mellitus, human immunodeficiency virus infection, intravenous drug abuse, malnutrition, obesity, organ failure, peripheral vascular disease, severe liver disease and patients with underlying malignancy.[6-7] NF was previously thought to be monomicrobial in its etiology, the main causative agent being Group A Beta hemolytic streptococci and hence was called as streptococcal gangrene.[4-5,8] Now NF is proved to be a polymicrobial infection involving anaerobes, gram negative bacilli and enterococci species.[1,4-5] The pathophysiological features include the formation of micro thrombi and vasculitis with eventual intravascular coagulation and spreading of necrosis.[8] The most common clinical presentations are painful edema, erythema, warmth, tenderness, crepitation, tissue necrosis, bullae, putrid discharge, gas production, rapid spread through the fascial planes and the presence of the classic tissue inflammatory signs[2,6,8] Systemic findings can include fever, tachycardia and hypotension.[2]

The diagnosis of NF of the head and neck is often a clinical one while imaging techniques such as soft

tissue radiography, CT scan and magnetic resonance imaging (MRI) will reveal the extent of the infection and the anatomic structures involved, identify any vascular thrombosis or vessel erosion.[2] Relevant laboratory tests include a complete blood test, an electrolyte panel, a coagulation profile, blood and tissue cultures, urinalysis and arterial blood gases. These often reveal leukocytosis, anemia, acidosis and hypocalcemia secondary to the deposition of calcium in necrotic tissues.[2,4]

NF is regarded as a surgical emergency and the cornerstone of treatment is surgical debridement. *The* necrotic tissue must be removed until fresh viable tissue growth is seen.[1,8] Empirical antimicrobial treatment should be instituted early and changed once the results from cultures and the antimicrobial susceptibility tests are obtained in order to enhance the patient's clinic response and improve their outcomes.[2] Patients should be resuscitated according to their clinical state and evidence of hemodynamic instability demands intensive care support with immediate resuscitation and nutritional support in order to replace lost fluids and proteins from large wounds.[2] Other adjunctive approaches to treatment that are still controversial include hyperbaric oxygen (HBO) therapy and intravenous immunoglobulin (IVIG). IVIGG can neutralize super antigens and down regulate the production of tumor necrosis factor.[2,4] HBO increases free radicals, which helps neutrophil mediated killing of some common bacteria and also acts as a bactericide for certain anaerobes.[7]

Reconstructive procedures should be planned only after complete resolution of the disease and once the recipient bed is healthy.[8] The defect can initially be covered with a split thickness skin graft and reconstructed secondarily by advancement flaps or revascularized free flaps if necessary.[1] The main complications of facial NF are airway obstruction, vascular thrombosis, mediastinitis, pleural empyema, large vessel thrombosis and septic shock.[2,6] Even with adequate surgical debridement and intravenous antibiotic therapy, the mortality rate associated with NF is 20% to 40%.[4,5]

CONCLUSION

NF of the head and neck is a rare but potentially fatal disease. Hence every medical professional should be aware of its etiology, epidemiology, risk factors and initial clinical manifestations to arrive at an accurate, prompt diagnosis and to offer adequate treatment. A delay in diagnosis would result in disastrous morbidity and mortality. An early diagnosis with Circulation, Breathing and Airway management, empirical intravenous antibiotic administration, aggressive surgical debridement followed by reconstruction of the resultant soft tissue defects, will play a pivotal role in achieving a successful outcome.

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