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REVIEW

Necrotizing cervical fasciitis: a case report and review of literature

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Abstract Necrotizing cervical fasciitis is a rare, fulminating infection causing extensive necrosis of the subcutaneous tissue and fascial planes with resultant skin gangrene. To reduce the morbitity and mortality from this condition, it requires early recognition and aggressive surgical debridment with appropriate antibiotic therapy. The origin is generally odontogenic and presents more often in immunocompromised patients. This article presents a case with typical clinical features and appropriate management of this condition. An review of literature was carried out for microbiology, pathogenesis, clinical features, diagnosis, management and prognosis of this condition.

Keywords Cervical fasciitis · Fulminating infection · Immunocompromised patients

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Introduction

Typically, necrotizing fasciitis occurs in abdomen/perineum or lower limbs after trauma or surgery. About (1–10%), are reported in neck region [1]. Antibiotics have reduced the prevalence and improved the outcome of deep neck infection; however deep neck infection continues to be associated with severe illness and death. Mortality rate have been reported upto 40%. Poor dental hygiene and IV drug abuse are now the current causes of deep neck space infection in adults. Necrotizing cervical fasciitis is a fulminant infection with necrosis of connective tissue spread along facial planes with high mortality. It is usually polymicrobial and odontogenic and occurs more frequently in immunocompromised and postoperative patients.

Historically necrotising fasciitis was described in the late eighteenth century by Claude Pouteau in 1783 [2]. The infection was more common in military hospitals by name of 'Hospital gangrene' or 'phagedena gangraenosa', among civilians it was less common and was thought to be confined to prostitutes, where disease was aggressive and involved in its ravages the vagina, perineum and anus [3,4].

Melaney isolated a Hemolytic Streptococcus from the wound and the disease was termed as 'Melaney's disease' [5]. Wilson in 1952 termed it as 'Necrotizing Fasciitis'. Currently, it occurs as unexpected isolated attacks.

Clinical presentation

A 50-year-old female reported to the casualty for neck pain and swelling for the last 4 days. Patient was not a known case of diabetes mellitus and was not on any medication for systemic conditions. History of tooth pain from last 8 days was noted. On evaluation vitals were stable and the patient was febrile with reduced mouth opening and with history of dysphagia for 2 days. Local examination revealed diffuse swelling in the bilateral submandibular region, extending anteriorly on the neck. The overlying skin was erythematous in submandibular region, whereas in anterior neck region the overlying skin was anesthetic, dark and dusky. A draining sinus was present with the right submandibular region. With complete blood count and electrolytes examination, emergency Incision and Drainage (I&D) under general anesthesia was done.

Bilateral submandibular spaces and submental spaces were drained, superficial neck spaces were drained with an anterior midline incision. Serosanginious discharge with a foul odor was noted, offending root fragments in bilateral molar region were extracted, drains were placed in the spaces, and the patient was kept intubated on spontaneous respiration in surgical ICU. Frequent dressings were done and IV antibiotics were administered. Extubation was done on third day. Serial debridement was carried out; necrotic tissue was excised revealing a large raw wound 3cms x 5cms revealing the strap muscles of the neck, surgical wound was packed with antimicrobial-soaked dressing. The surgical wound granulated and healed over a period of 3 weeks with a split thickness skin graft. With improved mouth opening and general health patient was discharged subsequently.

Discussion

Microbiology

Melaney was the first to associate group A streptococcal infection with severe necrotizing fasciitis. Various bacterial strains may dominate different wounds, it is clear that most necrotizing wounds sustain a mixture of bacteria working synergistically [5]. Various bacterial strains may dominate different wounds, but essentially necrotizing fasciitis may be categorized into three types according to the causative organism. (1) In cool and

temperate climates it tends to be associated with group A beta hemolytic streptococci (Streptococcus pyogenes) alone or with Staphylococcus aureus [6]. (2) In upto 60% cases the necrotizing fasciitis may be polymicrobial, including one or more obligate anaerobes (Peptostreptococcus, Provetella) [7–9]. (3) In tropical climates, the condition can be caused by members of family Vibrionacae, which are seawater origin. The pathogenesis of the polymicrobial form of the infection is unclear, though it is well known that various strains of bacteria work together to evade the host defenses and cause tissue damage. Host factors may predispose to the rapid spread of some infection [9,10].

Pathogenesis

Few studies have described about the pathogenesis of cervical necrotizing fasciitis. Literature describing pathogenesis of necrotizing fasciitis suggests that there is progressive liquefaction of the subcutaneous fat and the connective tissue below a relatively normal looking skin surface. The facial planes disintegrate, and with the ensuing necrosis come edema and the release of the tissue fluid. Early in the development of the disease the veins that transverse the liquefying subdermal fat become inflamed and start to thrombose, which gives the skin first a red and then a mottled color. Later arterial supply is also jeopardized and the skin becomes pale, which leads to necrosis and wet gangrene. Local inflammatory response within the dermis that is characterized by an intense polymorphonuclear infiltrate, focal necrosis, and micro abscesses formation. The histological picture is one of the arteriolar and venous thrombosis of the subcutaneous fat, whereas the adjoining muscles shows comparatively little inflammation [11–13].

Clinical features

Necrotizing cervical fasciitis is a fulminant infection with necrosis of connective tissue spread along the facial planes and high mortality rate. It is usually polymicrobial and odontogenic in nature, with high incidence in immunocompromised patients. Patient of cervical necrotizing fasciitis is usually severely ill with high fever. The overlying skin may be tender, edematous, erythematous and soft tissue crepitation from gas are diagnostic. As the necrosis progresses the overlying skin becomes pale and anesthetic, and then dusky. The presence of gas is neither a reliable nor discriminatory sign for clostridial infections because it can be absent in gas gangrene and present in various nonclostridial infection. The incidence of this disease increases with age (median age of 57 years) and most adult cases (70%) occur in patient with atleast one underlying chronic illness (immunosuppression, diabetes, alcohol/drug abuse, malignancy, or chronic systemic disease). Occasionally the disease afflicts apparently healthy individuals. In two third of cases, the necrosis followed either a skin lesion or trauma. The condition even has been reported after routine dental surgery or dental sepsis. The variable clinical picture means that delay in diagnosis and management is common. The rapid progression of the disease is a distinguishing feature [14,9].

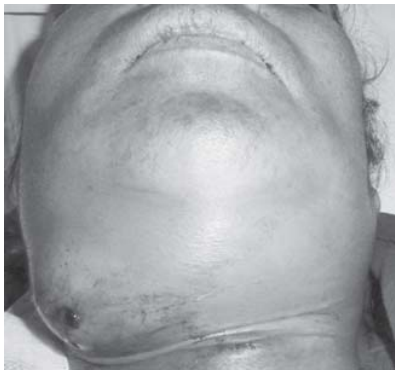

Fig. 1 Preoperative submental

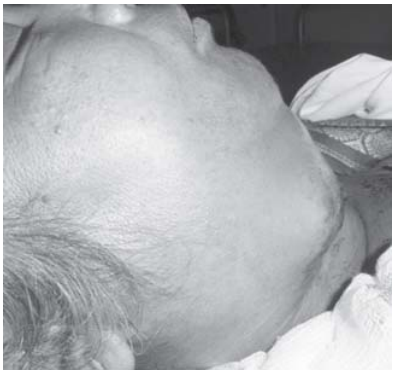

Fig. 2 Preoperative profile

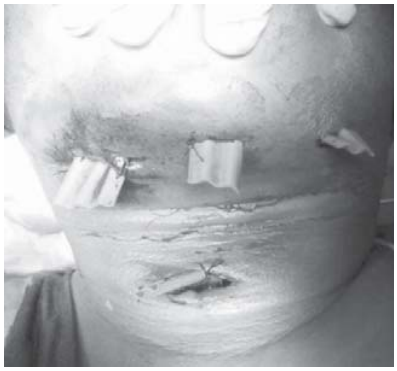

Fig. 3 Immediate postoperative

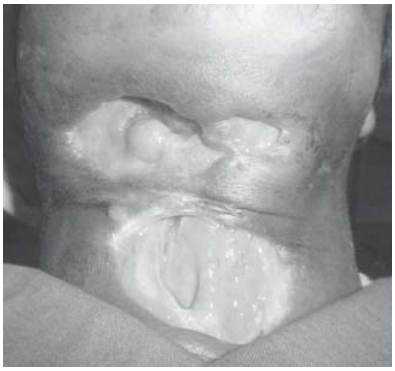

Fig. 4 3 weeks postoperatively

Diagnosis

If the clinical features are suggestive of necrotizing fasciitis, the diagnosis should not wait till the availability results of bacterial culture. Clinical inspection of the wound demonstrates that the subcutaneous fat has no structural integrity and offers little resistance to the exploring finger.

The skin is widely undermined by the progressing infection. Histologic criteria have been described for the early diagnosis of necrotizing fasciitis by frozen section and the typical pattern of a dense polymorphonuclear infiltrate in the dermal layers of the skin clinches the diagnosis [15].

Management

Time is of the essence at this stage, because mortality is associated with delayed intervention. Even if it is recognized promptly, if significant necrosis has taken place then excision is undertaken. More than one debridement may be necessary and it is considered prudent to make a second operative inspection of the wound after 24 to 36 hours.

Management is similar to that of an extensive burn. The wounds should be washed (hydrogen peroxide is useful for debridement) and packed regularly (every 4 hours) (Fig. 4), a procedure best done personally by the attending surgeon. Gradually the slough clears and shiny granulation tissue emerges from beneath

the yellow slime. The undermined skin at the edge of the wound reattaches to the underlying granulation tissue and the packing can be withdrawn slowly on daily basis. Regular dressing still should be maintained at 8 hour intervals, which demands nursing commitment. Eventually, antibiotic therapy is dictated by the cultures, but intravenous penicillin is the initial drug of choice. If the Gram's stain shows a mixed flora, a broad-spectrum antibiotic also should be used (gentamycin), and it can be supplemented as appropriate information is obtained. Hyperbaric oxygen has been suggested as a supportive measure, but there is no definitive evidence of efficacy and there are logistic problems if this technology is contemplated.

Prognosis

Despite proper management of necrotizing fasciitis, mortality remains high. In a multivariate analysis, age, hypotension, and bacteremia were independent variables that predicted mortality.

Patients can die from systemic problems some days or weeks after the infection, and in historical texts there are reports of fatal arterial bleeds occurring approximately 10 days after surgery, just as the infective process is settling [8,15].

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