AJMS
(A US National Library of Medicine enlisted journal)
Al Ameen J Med Sci (2011)4(3):290-294
ISSN 0974-1143
CODEN: AAJMBG

CASE REPORT

Necrotizing Fasciitis of Odontogenic Origin in a Non-Immunocompromised Patient- A Rare Case Report

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Abstract: Necrotizing fasciitis is a rapidly spreading infection involving the superficial fat, fascial layers with necrosis of skin and is a disfiguring condition that is fatal. Head and neck is an unusual site which is rarely affected. It is characterized by its fulminating, devastating and rapid progressive course. It usually occurs in patients with systemic conditions such as diabetes mellitus, renal disease, cardiovascular disease, HIV infections etc. A case of cervical necrotizing fasciitis of odontogenic origin occurring in a non-immunocompromised patient is reported here who was treated successfully by surgical debridement and antibiotic therapy.
Key words: Cervical necrotizing fasciitis, odontogenic, surgical debridement

Introduction

Necrotizing fasciitis is a rare and rapidly spreading superficial infection along fascial planes causing necrosis of fascia, superficial fat, overlying skin and blood vessels with characteristic sparing of muscles and bone [1].

Necrotizing fasciitis was actually first described by Hippocrates in 5th century B.C, who described this as a complication of erysipelas. In late 18th century Sir Gilbert Blane, Thomas Trotter and Leonard Gillespie British naval surgeons described necrotizing fasciitis in detail. In 1871 Jones called it “hospital gangrene” which was the first description of Necrotizing fasciitis in US, in 1918 Pfanner described “necrotizing erysipelas”, but the name “Necrotizing fasciitis was given by Wilson in 1952 to describe tissue death and associated fascial plane involvement characteristic of the disease [2].

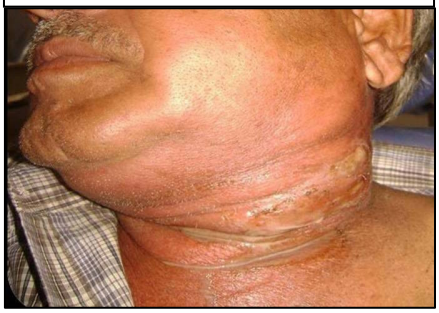
Necrotizing fasciitis is caused by polymicrobial or mixed aerobic and anaerobic microorganisms resulting in massive tissue destruction and toxic shock syndrome. Cases affecting the head and neck regions are unusual and rare [3-4].

However in contrast to an abscess or cellulites which resolves after antibiotic therapy or incision and drainage, treatment of necrotizing fasciitis requires thorough surgical debridement of necrotic tissue [5]. Without aggressive medical and surgical intervention, the patient usually becomes toxic, frequently requiring critical care support, with the potential for severe cosmetic deformity and death if not treated appropriately in a timely manner [3].

Case History

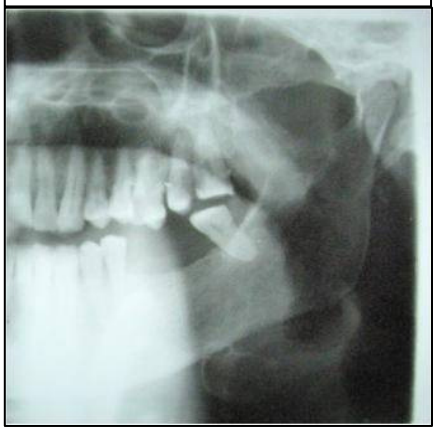
A 53 year old male patient reported to the Dental Hospital with the complaint of swelling and discharge in the left lower one third of the face and the upper part of neck for the past two days. He had been seen previously by a dentist who drained abscess giving extraoral incision that yielded pus and the patient was put on a course of routine antibiotics and analgesics. But the patient was not relieved of symptoms. A day later patient developed swelling in the upper part of the neck associated with severe pain, fever and dysphagia. It was at this stage that patient reported to the out patient dental department. There was no relevant medical history. Patient's physical examination was unremarkable and his vital signs were as follows, BP-110/70 mm Hg, pulse-94 b/min, respiratory rate-16 cycles/min and patient was febrile with temperature-102^{o} F.

Fig.-1: swelling showing shiny unusually erythematous with orange peel appearance



Examination of the site revealed a diffuse swelling on the left lower third of the face extending from ala-tragal line to 4 cm below the lower border of the mandible towards the upper part of the neck. Skin over the swelling was slightly stretched, shiny generalized erythematous appearance (Fig.1) with foul smelling purulent discharge from the incised area. Few blackish dusky spots were present. On palpation the swelling was tender with rise of local temperature. Submandibular and upper group of cervical lymph nodes were palpable and tender.

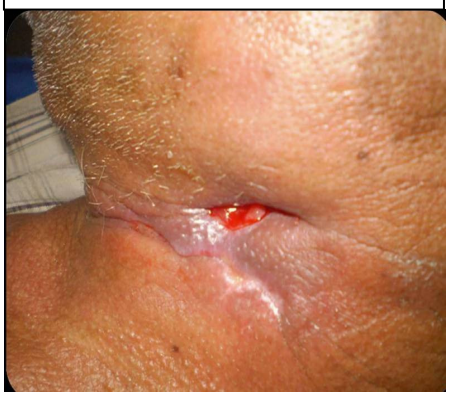
Fig.-2: OPG showing carious teeth



Intraoral examination showed 12mm mouth opening, vestibular obliteration with 27, 28 region and relevant teeth were tender on percussion. Provisionally diagnosis of buccal and submandibular space infection secondary to carious teeth 27, 28 were made. OPG was taken which showed carious teeth 27, 28(Fig.2). Then the patient was subjected to routine investigative procedures like complete blood count, RBS, HIV, and HbsAg. All the reports were within the normal limits except for the increased neutrophil count. Patient's physical fitness data was obtained from the physician. The patient was admitted for surgical debridement. Empirically the patient was put on Amoxicillin + Clavulinic acid 1.2gm intravenously every 8th hourly, amikacin 500mg intravenously every 12th hourly, metranidazole 500mg infusion every 8th hourly along with diclofenac sodium 75mg intramuscularly every 8th hourly.

Then the patient was shifted to the operating room. Under general anesthesia he underwent aggressive incision and drainage. A copious quantity of pus was released. Massive necrosis of the subcutaneous fat, fascia and skin was appreciated and surgically the diagnosis of cervical necrotizing fasciitis was made. 27, 28 were extracted and the specimen was sent for culture and antibiotic sensitivity testing. All necrotic tissue was debrided thoroughly along with fascectomy and involved areas were irrigated with tetracycline 400mg capsule mixed with normal saline. Later on corrugated rubber drain was placed for the residual pus. The patient was receiving the same drug regime as above for the next 7 days postoperatively. After 48 hours rubber drain was removed and patient was returned to the operating room for additional irrigation and debridement of the wound and open dressing was given regularly till the time he was discharged. No growth was appreciated on the culture.

Fig.-3: 12th postoperative day



On the 4th postoperative day, the patient's condition had improved remarkably. Much of the edema had resolved. On the 8th postoperative day shrinkage in the size of the wound along with healing sloppy edges were appreciated. Patient was then discharged from the hospital with one week course of oral antibiotics and analgesics were prescribed (Amoxicillin + Clavulinic acid 625mg every 8th hourly, amikacin 500mg every 8th hourly, metranidazole 400mg every 8th hourly along with diclofenac sodium 50mg every 8th hourly). Follow up after 12 days patient improved dramatically (Fig. 3).

Discussion

There are only about 68 reported cases in world literature of cervical necrotizing fasciitis [6]. Immunocompromised patients are at increased risk of developing necrotizing fasciitis [3, 5, 7]. However in the present case report the patient was non-immunocompromised.

The causative organism may be a single agent, commonly Group A β-hemolytic streptococci or staphylococcus aureus or may be a polymicrobial involving gram positive and gram negative aerobic, anaerobic bacteria and fungi. However many authors have found culture reports to be negative in the cases they have reported [6]. Initially there is cellulites which leads to invasion of the deeper tissues. Clinically at this stage skin changes of erythema and edema are seen. Progressive tissue necrosis causes an invasion by the normal flora. Continuous bacterial overgrowth and synergy, causes a decrease in oxygen tension and development of local ischemia and proliferation of anaerobic organisms. In 4-5 days gangrene is evident and after 8-10 days necrotic tissue separates from the underlying ischemic but viable tissue [6].

As the disease progresses, pathognamonic signs of Necrotizing fasciitis appear "…a dusky discoloration of skin appearing as small purplish patches with ill defined borders. Concomitantly blisters or bullae of few millimeters in diameter appear on the skin of the involved area. The skin beneath blisters becomes necrotic and blue in color. Localized necrosis of skin is secondary to thrombosis of nutrient vessels as they pass through the zone of involved fascia" [5].

The cervical necrotizing fasciitis of odontogenic origin involves mandibular second and third molar frequently as the apices of these teeth extend below mylohyoid insertion. Infective process originating from these teeth easily traverse into the submandibular space. In our case maxillary second molar was culprit which was consistent with findings of many authors [5].

In necrotizing fasciitis of odontogenic origin disease takes the usual path of spread, which invades deep tissue planes in early stage. It is only when the infective process gets to superficial fascia, spread becomes rapid and typical features of necrotizing fasciitis begin to show. This mode of presentation can be misleading to unsuspecting clinician and makes the early diagnosis difficult [8].

However some findings may present clinically when one is suspicious of cervical necrotizing fasciitis 1) Odontogenic infection that spreads to neck and anterior chest 2) Abnormal accumulation of gas in the tissue 3) Very rapid progression of infection 4) Orange peel appearance of involved skin which change to dusky discoloration as the disease progresses. Owing to the edema of the overlying tissue or the absence of gas, subcutaneous crepitus may be absent [4] which were also seen in our case.

The key to accurate and prompt diagnosis of necrotizing fasciitis versus a typical odontogenic infection lies in the clinician's ability to fully appreciate the patient's history, presenting condition and accurately progressing disease process [5]. If there is high index of suspicion clinical diagnosis of necrotizing fasciitis can be made by prompting early surgical exploration to confirm the diagnosis and perform appropriate debridement [2].

The corner stone of treatment is surgical debridement. All necrotic tissue must be removed until healthy bleeding tissue is encountered. Reluctance to debride fascial soft tissues aggressively and avoid unsightly disfigurement often leads to under treatment of the disease early in its course. Multiple surgical debridements in the operating room are usually needed [9]. After surgical debridement, wounds are left open and packed with povidine-moistuned gauze, which is changed frequently. It is important to prevent pooling of secretions in the wound that may provide a culture medium for further bacterial growth. Along with debridement, appropriate antibiotic coverage is imperative [9].

Hyperbaric oxygen therapy has been gaining support as an adjunctive treatment for Necrotizing fasciitis. Recently published reports cite a 50% reduction in mortality when hyperbaric oxygen therapy is used along with surgery to treat necrotizing fasciitis [5], but its role in necrotizing fasciitis as mentioned by other authors needs further evaluation [1].

Once the infection has been resolved, the defect can initially be covered with a split thickness skin graft and reconstructed secondarily by advancement flaps or revascularised free flaps if necessary [9].

Conclusion

Early clinical diagnosis has to be made as there is direct relationship between morbidity, mortality and time lapse before the start of appropriate treatment. Necrotizing fasciitis of head and neck is rare but potentially fatal disease that all dentists should be aware of as prompt diagnosis and recognition are the first and most important steps in its management. A delay in diagnosis would result in further disastrous morbidity and mortality.

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