Case Report

Cervical necrotizing fasciitis caused by dental infection: A review and case report

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ABSTRACT

Necrotizing fasciitis of the head and neck is an uncommon, potentially fatal , soft tissue infection characterized by extensive necrosis and gas formation in the subcutaneous tissue and fascia. The purpose of this report is to heighten the awareness of this infection. The article also outlines an appropriate management strategy for use in the treatment of these patients and reviews the literature along with a report of a case which was successfully managed.

Key words: Cervical necrotizing fasciitis, odontogenic cause

Introduction

Necrotizing fasciitis (NF) is a rapidly spreading, soft tissue infection characterized by diffuse necrosis of fasciae and subcutaneous tissues. Necrotizing fasciitis has a potentially fatal outcome. It occurs in all age groups but most patients are below 40 years of age. There is no sex or race predilection. Joseph Jones, an American army surgeon, described this entity in 1871 during the civil war. He named it "hospital gangrene." In 1924, Melany reviewed 20 cases of ''streptococcal gangrene.'' He was the one to note that subcutaneous necrosis is the hallmark of necrotizing fasciitis. It predominantly affects the tissues of the abdominal wall, the perineum and the extremities, but can be seen in the maxillofacial region also.[1]

The term ''necrotizing fasciitis'' was coined by Wilson in 1952 and has gained wide acceptance. Necrotizing fasciitis of the neck is rare and usually occurs secondary to dental infection, gingivitis, or pulpitis. However, any infection that can cause deep neck infection can cause necrotizing fasciitis. It is most often a mixed synergistic infection involving both aerobes and obligate anaerobes. This mixed bacterial infection spreads rapidly through the fascial planes of the head and neck to involve the subcutaneous tissues, skin, fasciae and even muscles.[2]

Misdiagnosis and delayed treatment can result in severe systemic toxicity, carotid artery erosion, jugular vein thrombobhlebitis, aspiration pneumonia, meningitis and mediastinitis. The mortality rate is 15%–40%. The foundation for successful treatment of this life-threatening condition consists of early diagnosis and aggressive surgical intervention combined with supportive therapy such as appropriate antibiotics, airway maintenance and adjunctive hyperbaric oxygen therapy. Computed tomography (CT) is a very useful tool for early diagnosis because it detects gas bubbles which can be difficult to see on plain radiographs.

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Case Report

A 60-year-old female patient presented to our department with chief complaints of toothache in the lower left posterior region since 10 days, a swelling on the lower third of the face since 3 days and difficulty in breathing since 1 day. She also gave a history of hot

fomentation. She had no history of systemic disease and of substance abuse.

On examination: There was a bilateral, tender, diffuse, edematous swelling on lower third of the face and neck region. The temperature over the swelling was raised. Patient had fever of 100 °F. Intraoral examination revealed a carious 37 and 31. Trismus was present. An initial diagnosis of bilateral submandibular, sublingual and submental space infection was made. The patient was admitted in the ICU and intravenous line was secured and carefully monitored.

Laboratory investigations revealed leukocytosis (25,000/mm^3), hemoglobin (9.9 gm%). The patient weighed 45 kg. Pus was collected from the submandibular region and sent for culture sensitivity and empirical broad spectrum antibiotics were started, viz., IV amoxicillin 500 mg, 8 hourly, IV metronidazole 500 mg, 8 hourly, IV gentamycin 80 mg, 12 hourly, injection diclofenac sodium 50 mg. IM 12 hourly, IV fluids were also administered. The culture and sensitivity report showed growth of gram positive cocci and gram negative rods.

Twenty-four hours after admission—incision and drainage was done in the submental region and bilaterally in the submandibular region, under i.v. sedation, 37 and 31 were extracted and the patient was monitored in the ICU constantly. Copious irrigation was done with povidone iodine, normal saline and H_2O_2 four hourly. There was some improvement in the symptoms but on the 7th day necrosis and sinus formation occurred in the chest region and the serum urea was 50 mg% (n = 8–21 mg%). Twenty-four hours later the necrotic area increased and dirty yellow necrotic foul smelling fascia was visible on removing the blackened overlying skin [Figure 1]. The diagnosis was changed to necrotizing fasciitis. The necrosed skin and fascia was excised and dressing with sofratulle and chloramphenicol powder was done. Antibiotics were changed to inj. cefocef 1 g, 12 hourly and inj. amikacin 500 mg, 12 hourly. However, the necrosis spread superiorly and the pus was draining from the neck and chest wounds and both the wounds communicated with each other. Left buccal space also got involved by the 10th day [Figure 2]. The space was explored and pus was drained out. A second culture and sensitivity report showed diptheroids sensitive to ampicillin and ofloxacin. Antibiotics were changed again to oflox 200 mg, 12 hourly and dressings were continued and the necrosed fascia over the chest was further excised on the 14th day, after which there was no further spread of the lesion. The margins started granulating and the patient's condition improved [Figure 3]. By the 23rd day, the neck part of the wound was freshened and approximated with 3-0 silk and 1 week later skin graft taken from the right medial thigh was used to cover the chest wound [Figure 4]. The graft took up only intermittently and the wound continued to heal and the patient was discharged by the 40th day [Figure 5]. By then her weight had come down to 30 kg, but she was healthy.

Discussion

The most frequent initiating factor reported, for necrotizing fasciitis, in the head and neck region is a primary odontogenic infection or post extraction infection.[3-5] Tonsillar infections, salivary gland infections, otogenic and dermatologic infections are other rare causes. Patients often have underlying systemic disease such as diabetes mellitus, alcoholism, malnutrition, or chronic renal failure.[1,2,6,7] In early stages, necrotizing fasciitis can be misdiagnosed as a soft tissue infection such as cellulitis or erysipelas with a benign superficial appearance. The relative mild external clinical signs can mask the severe underlying necrosis. This may lead to delay in necessary aggressive surgical intervention, which can be life threatening.[8]

Umeda *et al.* [7] did a review of literature and found 125 cases whose age ranged from 12 to 82 years (mean 45.2 years). Male predominance was seen (male:female = 3:1). The origin of infection was periapical infection from mandibular molars. Fifty percent of the patients had associated diseases; diabetes mellitus being the most common, followed by alcohol abuse, drug abuse, HIV infection, heart disease, liver cirrhosis, renal insufficiency and schizophrenia. However, 34% had no systemic problems. Of the 125 patients, 19.2% reportedly died despite aggressive therapy. The relationship among various clinical factors and the prognosis was studied. The mortality rate of the patients with associated diseases was 24.3% which was much higher than that of those who did not have systemic disease (9.3%).[7]

Necrotizing fasciitis is a polymicrobial infection of aerobic, anaerobic, gram positive and gram negative bacteria. Up to 11 organisms have been isolated from a single case.[6] Streptococcal species is the most common organism, but enterobacter, fusobacterium, bacteroides, staphylococci and diptheroids have all been isolated from these wounds. The clinical relevance of this is that initial empiric antibiotic therapy should be a high dose broad spectrum antibiotic.[9]

Since the clinical features of necrotizing fasciitis in the early stages are not specific, it may not be correctly diagnosed. Patients with oral and maxillofacial infections who had extraordinary clinical symptoms such as extensive swelling, redness, fever, crepitations or a marked

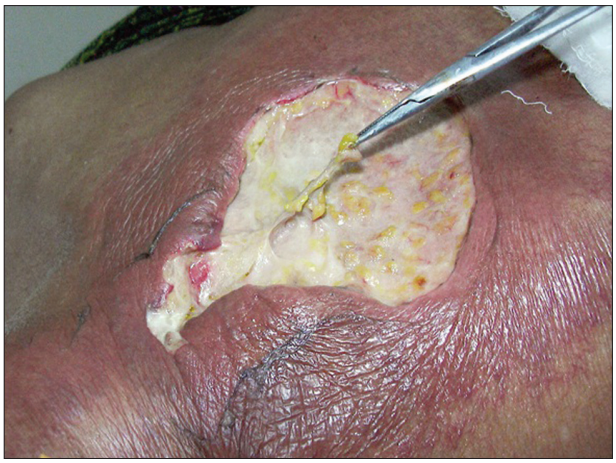

Figure 1: Necrotizing fasciitis of chest

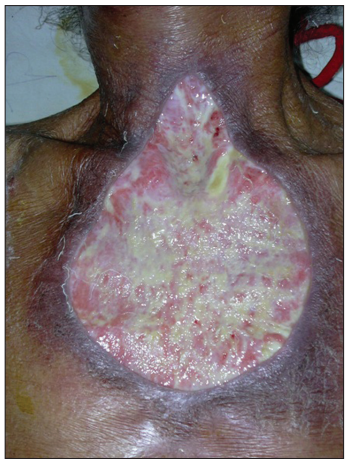

Figure 2: Lesion increased in size on day 10

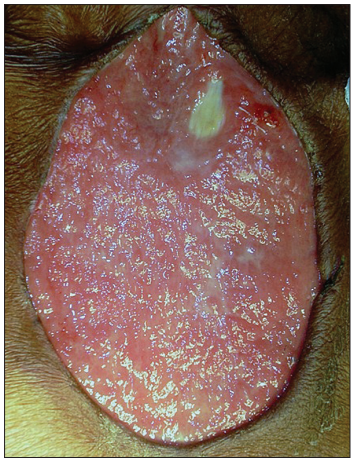

Figure 3: Granulating wound

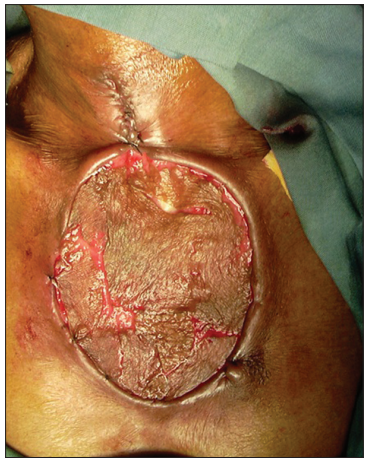

Figure 4: Skin graft and primary closure at the neck

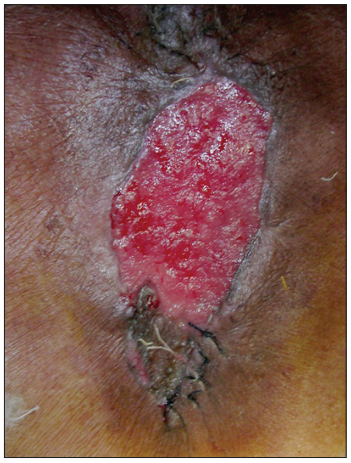

Figure 5: Healing wound

increase in serum CRP (C-reactive protein) should be strongly suspected of having necrotizing fasciitis. The necrotizing fasciitis infections are poorly localized and are characterized by inflammation and necrosis, extending deep to what is normal appearing skin. Skin changes are seen later as erythema, edema and hyperesthesia. Fever is prominent and features of systemic inflammatory response syndrome (SIRS) are seen (high fever, tachycardia, apathy, weakness and nausea).

Thus, a necrotizing soft tissue infection should be suspected whenever a small or clean wound is followed within 12–36 h by prominent systemic signs of sepsis. Crepitus and blistering are late features which indicate presence of gas in the tissues. CT is more sensitive than palpation in detecting gas in the tissues.

Necrotizing soft tissue infections often result in a loss of significant amount of fluid and there is a marked hemodynamic response. The response may be minimal to profound septic shock. In every case, the potential for profound cardiovascular and respiratory failure exists. Renal failure is a frequently associated complication. Therefore, careful evaluation, resuscitation and appropriate monitoring are mandatory. An intensive unit level of care is the minimum requirement, with continuous pulse oximetry, hourly monitoring of urine output and frequent recording of the hemodynamic values and respiratory rate.[8]

The principles of management of necrotizing fasciitis are:

1. Begin high dose empirical broad spectrum antibiotic therapy.
   - IV Benzyl penicillin 2.4 g, 4 hourly + flucloxacillin 1 g, 6 hourly + metronidazole 500 mg, 8 hourly or
   - IV cefotaxim 2 gm, 8 hourly + metronidazole 500 mg, 8 hourly or clindamycin 900 mg, 8 hourly or
   - IV imipenem/cilastatin 500 mg, 6 hourly or add penicillin 20 million units(if gram negative cocci present).
2. At least two blood culture and sensitivity specimens should be taken 20 min apart as well as specimens from the wound at a point away from any open wound to rule out contamination.
3. A CT scan will be helpful in detecting gas in the tissues and blood serum C-reactive protein (CRP) will be raised (11.7–33.7 mg/dl) and leukocytosis present (11,80038,700/mm^3)
4. ICU care and constant monitoring of all vital parameters and nutritional support and care of the systemic diseases like diabetes mellitus.
5. Tracheostomy if necessary to maintain the airway.
6. Surgical debridement will be required minimum twice or more times. Excision of all necrotic tissue is done till normal appearing tissue appears which bleeds freely on incising. Extension of the infection is easily overlooked at the first procedure, so a second procedure is required after 24 h. Wound should be left open and insert penrose drains into deeper fascial planes.
7. Irrigation with NaCl 0.9% and 0.5% H_2O_2 is done as often as possible.
8. Hyperbaric oxygen therapy (1.5 h at 2.5 atm for 15 days) has been used as an adjunct in the treatment of necrotizing fasciitis and can be used if available.
9. When the wound is seen to be granulating healthily, a skin graft can be placed over the site or an attempt at primary closure can be made.

It takes about 20–40 days for a patient to recover completely from this infection.

Necrotizing fasciitis of the head and neck is a rare disease, but dentists may encounter it because dental infection is the main cause of this disease. The reduction in mortality of this disease depends upon its early detection and adequate surgical treatment.

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