Cervicofacial Necrotizing Fasciitis

1FM Debta, 2Priyanka Debta, 3Abijet Deoghare, 4Rajkumar Diwan
1Associate Professor, Department of Oral Medicine and Radiology, Chhattisgarh Dental College and Research Institute Rajnandgaon, Chhattisgarh, India
2Lecturer, Department of Oral Pathology, Chhattisgarh Dental College and Research Institute, Rajnandgaon, Chhattisgarh, India
3Lecturer, Department of Oral Medicine and Radiology, Chhattisgarh Dental College and Research Institute Rajnandgaon, Chhattisgarh, India
4Postgraduate Student, Department of Oral Medicine and Radiology, Chhattisgarh Dental College and Research Institute, Rajnandgaon, Chhattisgarh, India

Correspondence: FM Debta, Associate Professor, Department of Oral Medicine and Radiology, Q No-8/16, Staff Quater, Chhattisgarh Dental College and Research Institute, Rajnandgaon, Chhattisgarh, India, e-mail: fm_debta@rediffmail.com

ABSTRACT

Necrotizing fasciitis is rapidly spreading soft tissue infection involving the subcutaneous tissues. Cervicofacial necrotizing fasciitis (CNF) is rare complication from dental infection that can lead to the involvement of the neck, mediastinum and chest wall. The case discussed below is of a patient with an odontogenic infection, leading to spread of infection along the cervical fascial planes due to immunocompromised state of patient causing necrosis and wound in neck region, diagnosed as cervicofacial necrotizing fasciitis. The author found a successful outcome in the patient after he underwent surgical debridement and antibiotic coverage.

Keywords: Cervicofacial necrotizing fasciitis, Immunocompromised, Fascial planes, Odontogenic infection.

INTRODUCTION

Necrotizing fasciitis is a rare and rapidly spreading superficial infection along the fascial planes causing necrosis of fascia, superficial fat, overlying skin and blood vessels with characteristic sparing of muscles and bone. It is less common in head and neck because of the rarity and higher vascularity in the region. The commonest cause of cervical necrotizing fasciitis is dental infections (Chan et al 1997). Necrotizing fasciitis is caused by polymicrobial or mixed aerobic and anaerobic microorganisms resulting in massive tissue destruction and toxic shock syndrome. The importance of early intervention had been consistently emphasized.

PATHOPHYSIOLOGY

The key pathological process resulting from this uncontrolled proliferation of bacteria is angiothrombotic microbial invasion and liquefactive necrosis of the superficial fascia. As this process progresses, occlusion of perforating nutrient vessels to the skin causes progressive skin ischemia. This is the underlying event that is responsible for the cutaneous manifestations of necrotizing fasciitis as the disease evolves. Initially a horizontal phase predominates with rapid spread through the fascia with extensive undermining of the apparently normal looking skin. As the condition evolves, ischemic necrosis of the skin ensues with gangrene of the subcutaneous fat, dermis and epidermis, manifesting progressively as bullae formation, ulceration and skin necrosis.

CASE REPORT

A 50-year-old male patient, farmer by profession reported to the department of oral medicine and radiology with complain of toothache 6 months back. There has been pain and swelling since last 3 weeks in left side of neck and soft tissue defect of skin since 3 to 4 days (Fig. 1). Patient medical history revealed known case of tuberculosis with symptoms of recurrent cough since 6 months and is under medication for same.

Present history of patient revealed that he was apparently alright 6 months ago then he had on and off pain. Then he noticed swelling in upper half of left side of neck, which suddenly increases in size and painful in nature. The swelling ruptured 3 to 4 days back forming surface defect over the skin with

Fig. 1: Necrotizing nonhealing ulcer

656
swelling persisting in lower half of wound. The defect started increasing in size with foul odor and pus discharge. The defect and surrounding swelling was painful causing discomfort to patient, which became intense and severe last week and patient presented to the department in the present state.

Extraoral examination on inspection revealed size of the ulcer approximately 4 × 5 cm in upper half of left side of neck extending anterioposteriorly from parasymphseal region to the body of mandible, superior-inferiorly from line extending lower border of lip up to 2 to 3 cm below lower border of mandible. Maximum portion of ulcer margins found to be everted and surrounding skin of posterior part of ulcer with necrosed black color skin. Floor of the ulcer having slough along with discharging pus and tense inflammatory swelling persist in lower half. Submandibular lymph node palpation of the left side was not possible following large surface defect with persistent swelling in same region, while right side submandibular lymph nodes were two in number enlarge and tender on palpation. Intraoral examination revealed poor oral hygiene, carious 36 and 37. Palpatory finding confirming size of ulcer 4.3 × 5.2 cm. Swelling surrounding the ulcer was tender on palpation was soft fluctuant in consistency. The temperature over skin was raised. Margins raised but nonindurated, base of the ulcer was soft nonindurated and tender, slough on removal shows raw bleeding.

Based on history of patient and clinical findings of the patient our provisional diagnosis comes to be in favor of necrotizing ulcer and the patient was further investigated for other radiographic and laboratory investigations. Aspiration of pus oozing out of the wound was sent for culture sensitivity. The result of the culture obtained after 72 hours shows polymicrobial mixed aerobic and anaerobic infection.

Panoramic radiograph showed proximal radiolucency with periapical rarefaction with 37 suggestive of periapical rarefying osteitis (Fig. 2). The differential diagnosis of ulcer comes to be cold abscesses with tuberculous ulcer—the clinical picture of inflammatory signs with pain and tenderness does not support the diagnosis for cold abscess. Absence of undermined edges, deep involvement of tissues and nonindurated base rules out the diagnosis of cold abscess with tuberculous ulcer. Malignant ulcer—the absence of induration on base as well as margin which is one of the prominent feature of malignant ulcer. For further confirmation and diagnosis, patient was advised incisional biopsy.

Routine hematological investigations were done: Hb—10.4 gm%, RBCs—3.9 × 10⁶/mm³, WBC—6.9 × 10⁹/mm³, neutrophil 78%, lymphocytes 25%, eosinophills 03%, monocytes 02% and basophills 00% and ESR of 40 mm/hr (Westergren). Other biochemical and hematological test findings are: Serum sodium conc—132 µmol/l, serum potassium—4.5 mg/dl, serum creatinine—146 µmol/l, C-reactive protein—152 mg/l, platelet count 150 × 10⁹/mm³, with other parameters like blood sugar, etc. were within normal range. After routine laboratory investigation, we categorize our patient on basis of LRINEC (laboratory risk indicator for necrotizing fasciitis) as: High-risk score—8 (Table 1). As per LRINEC score patient is evaluated on basis of total white cell count, serum sodium, serum glucose, serum creatinine and C-reactive protein. LRINEC score is robust capable of detecting early cases of necrotizing fasciitis and differentiate NF from other soft tissue infections. Although modalities, such as CT, MRI and frozen section biopsy have been shown to be useful in the early recognition of NF, routine application of these modalities in the evaluation soft tissue infection has been limited by cost and availability.

Table 1: Laboratory risk indicator for necrotizing fasciitis score³

<table>
<thead>
<tr>
<th>C-reactive protein, mg/l</th>
<th>0</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 150</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>≥ 150</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Total white cell count, per mm³</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>&lt; 15</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>15-25</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&gt; 25</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin, gm/dl</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>&gt; 13.5</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>11-13.5</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&lt; 11</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Sodium, mmol/l</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>≥ 135</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>&lt; 135</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Creatinine, µmol/l</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>≤ 141</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>&gt; 141</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Glucose, mmol/l</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>≤ 10</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>&gt; 10</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

LRINEC score ≤ 5 (Low risk)
LRINEC score 6-7 (Moderate risk)
LRINEC score ≥ 8 (High risk)

The maximum score is 13; a score of ≥ 6 should be raised the suspicion of necrotizing fasciitis and score of ≥ 8 is strongly predictive of this disease.

Patient’s history, clinical presentation with radiographic indicate odontogenic infection. Further laboratory findings and biopsy report (Fig. 3) indicates microbial infiltration and necrosis of superficial fascia, inflammatory infiltrate, thus ruling out other differential diagnosis and confirming the diagnosis of cervicofacial necrotizing fasciitis.

Fig. 2: Periapical rarefying osteitis with 37
MANAGEMENT

The patient was scheduled for surgical treatment, under local anesthesia. Copious irrigation with saline and betadine was done. The offending 36, 37 molar was also extracted. The patient was started on intravenous fluids and crystalline penicillin 2 mega units 6 hourly, and metronidazole 500 mg infused as 100 ml of a 5 mg/ml solution at 5 ml/min eight hourly, for first 48 hours. For pain relief, he was placed on ibuprofen 50 mg intramuscularly 6 hourly for the first 48 hours.

Four weeks following surgical debridement the resultant wound had granulated well and started healing properly. In the mean time the patient refused for skin grafting and allowed the wound to heal by secondary intention, i.e. contracture with moderate scarring of the tissues involved.

DISCUSSION

Necrotizing fasciitis (NF) as a disease entity, has been described under different names including hospital gangrene, suppurrative fasciitis, Meleney’s gangrene, streptococcal gangrene and Fournier’s gangrene. First described in 1871 by Joseph Jones. In 1952, Wilson found this infection to be primarily caused by *Staphylococcal* species. However, recent literature has revealed a polymicrobial etiology including streptococci and anaerobes. The causative organism may be a single agent, commonly group A beta hemolytic streptococci or *Staphylococcus aureus* or may be a polymicrobial infections. Necrotizing fasciitis is most common in the extremities, perineum and abdominal wall and predominantly occurs in elderly and immunocompromised patients though rare, but few reports can be found in the literature involving the head and neck region and most of these follow an odontogenic or oropharyngeal infection while in cases involving the face or scalp regions, trauma has been the predisposing factor. In the early stages of the disease, the signs and symptoms of NF are said to be nonspecific with the skin presenting as red-hot, smooth, tense and tender without demarcation between the involved and normal skin.

Predisposing factor for necrotizing fasciitis are: Immunosuppression, diabetes mellitus, alcoholism, malignancy, severe malnutrition, severe peripheral vascular disease, intravenous drug use, renal failure, radiotherapy, obesity. In our case, positive history of tuberculosis suggesting immunocompromised state of the patient as a predisposing factor for NF, also patient had history of chronic alcoholism. Our patient presented with history of toothache, was investigated for intraoral examination was found carious 36, 37 which on further radiographic investigation was diagnosed as periapical abscess. Whereas the signs of an acute expanding swelling and necrotizing defect suggestive of NF through the odontogenic route further exacerbated due to the immunocompromised state (tuberculosis positive) of the patient.

The role of bacterial necrotoxins in advancing the spread of the disease with ensuing gangrenous necrosis along the fascial planes has been known. The extensive involvement in our patient indicates the body’s acute reactions to these toxins. LRINEC score of ≥6 should raise the suspiscious of necrotizing fasciitis, and a score of ≥8 is strongly predictive of this disease. The LRINEC score is however, very useful diagnostic adjunct in the management of soft tissue infections to stratify these patients into low, moderate and high risk categories for necrotizing fasciitis for further evaluation. In our case score 8 was noted, thus patient comes under high risk category for necrotizing fasciitis. The succesful management of NF requires early accurate diagnosis, aggressive surgical debridement. Recently published reports cite a 50% reduction in mortality when 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.

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.

REFERENCES