Extensive Cervico-Thoracic Necrotizing Fasciitis Secondary to Odontogenic Infection: A Rare Case Report

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Abstract
Cervical necrotizing fasciitis is an infection of polymicrobial origin, causing necrosis of subcutaneous tissue by rapidly spreading along the fascial planes. Various cases of fasciitis have been documented in other areas of the body like abdominal fascia, extremities, perineurium but it is considered a relatively rare incidence in the head and neck region. Odontogenic infections can spread extensively to involve the mediastinum and thorax. Patients with systemic illness and compromised immunity are at a higher risk for such infections. The following is a rare case report of a non-immunocompromised adult patient who developed cervico-thoracic necrotizing fasciitis due to an infection of odontogenic origin.

Keywords: Necrotizing Fasciitis; Cervicothoracic Involvement; Polymicrobial Etiology; Odontogenic Origin; Cervicofascial Infection; Head and Neck Infection

Introduction
Cervical necrotizing fasciitis (CNF) is a deadly polymicrobial infection in the head and neck region spreading along the tissue planes to involve the subcutaneous tissues, fascia and muscles [1,2]. The hallmark of the disease involves a rapid progression and associated systemic toxicity [3]. The incidence of necrotizing fasciitis has been reported commonly in the extremeties, abdominal fascia and the perineurium, but the disease with a mediastinal and extensive thoracic extension has been rarely reported in the literature. In the initial stages, the CNF resembles an abscess or cellulitis, both of which resolve with the administration of antibiotics and surgical drainage. A surgical debridement of the necrotic tissue under adequate anaesthesia is required in cases of CNF. The disease is characterized by a fulminating, devastating and rapid progressing course. The mortality rate in patients with CNF is high if not treated promptly and vigorously.

Hippocrates was the first person to describe about the condition in the 5th century, whereas the term necrotizing fasciitis was coined by Wilson in 1951. Wilson coined the term to describe the tissue death and the associated fascial plane involvement. CNF is often misdiagnosed as erysipelas or cellulitis. The classical signs of the disease include tachycardia, dehydration, difficulty in swallowing due to edema in the neck region and trismus. The overlying skin is often erythematous, tense and may be hypoesthetic or anaesthetic to touch. A fetid odour associated with the disease is suggestive of the presence of dead tissue. Usually it is confined to the subcutaneous spaces, but may spread to the underlying muscles in extensive and advanced cases. Most of the cases have been reported in patients who were systemically or immunologically compromised, but it may occur in completely healthy individuals. The following case report of a 45 year old male patient in CNF that developed due to an odontogenic infection had a severe extension into the thorax not involving the mediastinum [4-8].

Case Report
A 45 years old male patient reported to the Department of Oral and Maxillofacial Surgery, with the chief complaint of pain and mild swelling in the posterior mandibular region, and a draining sinus over the sternum since 3 days. Clinical examination revealed an erythematous, tender swelling with cracking sound over the anterior chest wall, with continuous pus discharge from...
the same (Figure 1). Patient gave a history of tooth ache and swelling over the posterior mandibular region 6 months before he visited us, for which he was given a 5 day course of antibiotics and analgesics. The initial symptoms of the patient reduced, but later increased with the spread of the lesion to the chest region. Patient was then referred to the Department of General Surgery for further management.

After a prophylactic dose of Intravenous antibiotics (Amoxycillin + Clavulanic Acid and Metronidazole), drainage of pus from the anterior chest wall was done through the sinus opening, and sent for a culture and sensitivity testing. The patient was admitted for further monitoring and aggressive antibiotic regimen. On day two, the induration had increased with sloughing and continuous foul smelling pus discharge. On day three, necrosis of the overlying skin was seen with direct exposure of the underlying thoracic cage (Figure 2).

On day four, curettage and debridement was done to remove the necrotic fibro-fatty tissue and drain all the accumulated pus. The large skin flaps were left open in the chest region to allow healing by secondary intention. Povidone iodine based dressings were placed over the area for the next 30 days, until a healthy bed of granulation tissue formation was seen. Fresh pus samples were sent for every 72 hours for antibiotic culture and sensitivity testing. After the cessation of the active phase of infection, the carious teeth 46 and 47 were extracted, that were the causative agents of CNF in this case. Once the healthy granulation tissue bed was seen, the defect was closed with a split thickness skin graft that was harvested from the thigh region (Figure 3). A satisfactory adaptation of the graft was seen at the recipient site postoperatively (Figure 4). Patient was kept on a long term follow up [9-11].
The fulminating nature of CNF is attributed to the symbiotic relationship between aerobic, facultative anaerobic and obligate anaerobic organisms. The primary pathogens in CNF are the B-hemolytic streptococci, staphylococci and bacteroides. Diabetes Mellitus, Hypertension, Obesity, alcohol abuse and HIV infection are considered the risk factors in the development of CNF and their extension into the thorax, although the occurrence of the same cannot be overlooked in a completely healthy individual. The key to an accurate diagnosis and the prompt treatment lie in the ability to diagnose the patient history, signs and symptoms. Necrotizing fasciitis may initially be mistaken for a superficial soft tissue infection such as cellulitis (Klabacha and Stanciewicz, 1982) and accordingly may be undertreated [5].

Spread of the infection in the subcutaneous tissue along the fascial planes give rise to a smooth tense, shiny inflamed skin surface. As the disease progresses, the overlying skin becomes dusky with irregular purple/blue patches. Blisters or bullae eventually appear in the affected area as superficial vessels to the skin thrombose on their way through the involved fascia leading to skin necrosis (Mruthyunjaya, 1981) [5]. Mandibular molars are the usual causative factors in the cases of CNF. If left untreated, the infection may spread to the submandibular, submental, sublingual and parapharyngeal spaces. Ultimately the extension of the infection can occur cephalad to base of skull or into the mediastinum and thoracic cavity. Aggressive surgical debridement should be implemented without delay and all necrotic tissue must be excised. In this case, the patient developed odontogenic infection due to chronic irreversible pulpitis with 46 and 47 which led to an extensive spread to the subcutaneous fascia. All the features of CNF may be not seen on clinical examination, especially cracking sound and emphysema may be difficult to elicit, but a thorough history taking and raised White Blood Cell count on blood examination may give an insight into diagnosis. The patient needs to be typically admitted to intensive care unit for frequent monitoring, wound checks, triple antibiotic therapy, central monitoring to assess fluid status and hemodynamic parameters [3].

Effective management of CNF depends on early diagnosis and immediate aggressive regimen of broad spectrum intravenous antibiotics whilst specimen culture sensitivity is awaited. Intensive monitoring for assessment of systemic toxicity of the patient should be carried out along with the removal of odontogenic etiologic tooth. Future studies on the prevention and effective management of such extensive infections need to be carried out.

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Informed consent was obtained from all individual participants included in the report. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.