Necrotizing Fasciitis of Neck due to Third Molar Infection: Case Report

Abstract

Abstracts background: Necrotizing fasciitis (NF) is a severe soft tissue infection characterized by rapidly progressing necrosis, involving fascia and subcutaneous tissues. This rare condition carries high mortality rate and require prompt diagnosis and urgent treatment with radical debridement and antibiotics.

Method: We prescribe a case of 61 years old Chadian male with history of Diabetes Mellitus who presented to emergency of KFHJ. He reported dental infection and swelling related to lower right jaw one week before. Which developing necrosis, ill-defined black border and gas formation in the neck region.

Result: patient underwent extensive debridement within 24 hours and second debridement after another 24 hours with multiple antibiotics.

Objective: The purpose of this article is to review the NF of neck infection secondary to lower third molar tooth infection.

Conclusion: The surgeon should suspect necrotizing fasciitis when dental abscess is resistance to the conventional therapy. Satisfactory outcomes can be achieved with early diagnosis and aggressive surgical therapy in concert with empirical antimicrobial therapy.

Keywords: Necrotizing fasciitis; Dental infection; Third molar

Introduction

Necrotizing fasciitis (NF) is a severe soft tissue infection characterized by rapidly progressing necrosis, involving fascia and subcutaneous tissues [1]. Cervical necrotizing fasciitis (NF) was first clinically defined by Joseph Jones in 1871, during the US civil war. In 1918, Pfanner diagnosed a patient with a beta-hemolytic streptococcal infection and designated it "necrotizing erysipelas". Wilson was the first person to use the term NF in 1952. Cervical NF is a fulminant infection that spreads with high mortality and necrosis of connective tissue that spreads along the facial plane [2]. In 1977, Giuliano et al. [3] classified NF into two subtypes:

Type I: A polymicrobial infection (usually caused by a combination of Gram-positive cocci, Gram-negative rods, and anaerobes), and

Type II: A monomicrobial infection (caused by Streptococcus and/or Staphylococcus aureus). A polymicrobial infection (type I) is often diagnosed in immune compromised patients and usually occurs in the perineum and Trunk area.

In general, the disease is limited only with the subcutaneous tissue and muscle involvement is rare. The fact that necrotizing fasciitis causes tissue necrosis and spreads rapidly along the facial plane is due to its being polybacterial and the synergistic effect of enzymes formed by the bacteria. The most common factor is pathogen streptococcal. While involvement in the head and neck area is rare, it is more common in extremities, the genital region, and the abdomen [4]. The main reasons for the disease are odontogenic infections and trauma. The disease frequently develops in individuals that have an insufficient and low immune system. It is commonly seen in middle-aged individuals. The early stage of the disease looks like abscess and cellulitis. The covering skin is usually red and taut. Hyperesthesia or anesthesia can be identified by touch. The benign nature of the disease is the most important reason for late diagnosis. The diagnosis can be made by subcutaneous gas formation [5]. The present of immunocompromising conditions predisposes to CNF as well as increase morbidity and mortality. Satisfactory outcomes can be achieved with early diagnosis and aggressive surgical therapy in concert with empirical antimicrobial therapy [6].

Case Report

A 61 year-old Chadian male with uncontrolled DM was referred to emergency department of KFHJ complaining of rapidly progressive large laceration covered with black necrotic skin extended from the right side of the lower border of the mandible to the thyroid cartilage in the neck. He reported dental infection and swelling relation to lower right jaw one week ago. Clinically the patient was fully conscious, oriented, dehydrated and fatigue. Head and neck region examination revealed ill-defined border of black necrotic skin associated with bad odour, pus discharge and crepitating on palpation due to gas formation.
According to the oral examination, there was edema in the floor of the mouth, badly decayed teeth relation to 48, and the state of general oral hygiene was bad. His vital signs during arrival arterial PB was 145/80 Hg, pulse was 60 beat/minute, their respiratory rate was 22, body temperature was 36.7°C. Laboratory investigation: WBCs 40, HB 12.8 gr/dL, HCT 39.7 L/L, glucose 337 mg/dL, and . Head and Na 161 MMOL/L, K 3.2 MMOL/L, head and neck CT showed widespread subcutaneous air, edema in tissues, micro abscess focuses that hold together deep tissues (Figure 1).

Swap culture was obtained and sent to the microbiological lab for culture and sensitivity; specimens of the necrotic tissue were collected and sent for histopathology. All the sloughed tissue was removed with the irrigation of the wound by saline solution and dressing of chlorhexidine with Vaseline gauze was applied. Taking into consideration the aggressive nature of disease decision was made to perform second aggressive debridement of necrotic tissue and purulent materials (Figure 4 & 5).

The initial working diagnosis was Necrotizing fasciitis of Neck Secondary to Infection in Lower Third Molar. In the second day patient was taken to operative room, debridement of overlying necrotic skin and subcutaneous tissue until vital vascularized tissue was encountered and extraction of teeth (Figure 2&3).

Figure 1: On clinical examination, the patient showed swollen ill-defined border of black necrotic skin associated with bad odour, in the submandibular, sub mental, and upper neck areas.

Figure 2: CT scan of head and neck showing air and pus collections in bilateral submandibular, sub mental and cervical region.

Figure 3: 1st & 2nd Operation: Debridement of overlying necrotic skin and subcutaneous tissue until vital vascularized tissue was encountered and extraction of teeth #48.

Figure 4: 1st & 2nd Operation: Debridement of overlying necrotic skin and subcutaneous tissue until vital vascularized tissue was encountered and extraction of teeth #48.

Figure 5: 3rd Operation: Reconstruction by right supraclavicular pedicle flap and split thickness skin grafting from the thigh was done on the 12th postoperative days.
The patient was taken to the ward for management of his poorly controlled diabetes. Meropenem was initially prescribed (1g intravenously every 8 hours) for broad spectrum coverage until the results for culture and sensitivity were obtained. He remained stable systemically and responded well to the treatment as evidenced by normalizing inflammatory markers. The Antibiotic coverage was changed to Tazocin (IV 4.5 g every 6 hours) by infectious disease department after consultation according to developing hypernatremia and hypokalemia.

The culture and histopathology report was extensive necrotic of fascia, subcutaneous, muscle and overlying epithelium invaded by mixed groups of gram positive cocci and gram negative bacilli.

Patient was followed routinely, blood glucose was properly managed. The wound continues on twice daily irrigation by normal saline and dressing until signs of recovery tissue present.

The patient improved dramatically. Healthy granulation tissue appeared and reconstruction by right supraclavicular pedicle flap and split thickness skin grafting from the thigh was done on the 12th postoperative days (Figure 6&7).

After oral intubation and administration of general anesthesia, preparation and draping the patient in a sterile surgical fashion, recipient site preparation (done by team A) where curettage until vital vascularized tissue was encountered together with copious saline solution irrigation was done.

At the same time the donor site preparation (done by team B) where the skin incision to right supraclavicular area and dissection was done for the flap. The supraclavicular flap sutured to the skin of right side of submandibular and neck region. Also, we made split thickness skin graft harvested from both side of thigh utilizing the Dermatome. The harvested skin graft meshed and cut in tow pieces for maximum coverage and sutured to the left side of cervical skin defect and flap donor site.

Figure 6: 3rd Operation Reconstruction by right supraclavicular pedicle flap and split thickness skin grafting from the thigh was done on the 12th postoperative days.

Figure 7: 3rd Operation Reconstruction by right supraclavicular pedicle flap and split thickness skin grafting from the thigh was done on the 12th postoperative days.

Figure 8: 3rd Operation Reconstruction by right supraclavicular pedicle flap and split thickness skin grafting from the thigh was done on the 12th postoperative days.

Figure 9: After 1 month (POST OP).

Figure 10: After 1 month (POST OP).
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Discussion

Cervical NF is an infection that rapidly progresses on the facial plane. It is more common in males. The mortality rate is from 19% to 40% [4]. Necrotizing fasciitis may affect any part of the body; commonly involved are the extremities, abdominal wall and perineum and it's less common in the head and neck region [7]. The commonest cause of cervical necrotizing fasciitis is dental infections. The mandibular second and third molar teeth are the common reason behind odontogenic-based infections. The fact that these teeth reach the region where the mylohyoid muscle sticks to the mandible enable infections caused by these teeth to reach the submandibular region. The infection may progress to the base of skull in the upper region and the thorax and mediastinum in the lower region [8]. Immunocompromised patients and those suffering from systemic illnesses such as diabetes mellitus are at an increased risk of developing this infection. It may also affect previously healthy individuals (13-31%) [4,9]. Different terms and classifications have been used to describe necrotizing infections of the skin and subcutaneous tissue. These include necrotizing fasciitis, synergistic necrotizing cellulitis, streptococcal myonecrosis, and gas gangrene. This variety of classifications and terminology has been based on affected anatomy, microbial cause, and depth of infection. Awareness of this helps to reduce confusion.

Terms like necrotizing fasciitis, myonecrosis, and necrotizing adipositis refer to classification by depth of infection. Necrotising fasciitis can be divided into 5 types:

1. Necrotising fasciitis Type I: Polymicrobial, it is caused by non Group A streptococci and anaerobes and / or facultative anaerobes. Usually seen after trauma or surgery. There is involvement of subcutaneous fat and fascia with sparing of muscle. Gas formation is common.
2. Necrotising fasciitis Type II: Group A B hemolytic streptococcus. It is caused by Streptococcus pyogenus alone or with Staphylococcus. Usually associated with Streptococcal toxic shock syndrome. Predisposing factors are trauma, surgery or varicella infections.

3. Clostridial myonecrosis - Gas gangrene. It is characterized by its fulminant onset. The predominant features are muscle necrosis and gas production. The commonest causative organism is Clostridium perfringens.

4. Fournier's gangrene (J.A. Fournier, 1883) - necrotising fasciitis of the scrotum.


This evolving emergency state requires prompt diagnosis (both clinical and radiological), prompt implementation of pharmacological measures (broad spectrum IV generation antibiotic therapy based on blood and wound cultures), and emergent surgery [5,9]. Our patient suffered from necrotizing fasciitis caused a necrosis, which spread to the submandibular region after an infected third lower molar tooth and progressed over a broad region of the neck. The infection causes necrosis in submandibular tissue as a result of intensive lymphocytic infiltration, vascular thrombosis and edema as it spreads along the muscle layers. The layer covering the skin is sensitive, red and has edema. Subcutaneous tissue is dull and suffers from edema.

Suitable radiological examinations should be requested as soon as possible in order to determine the extent of the disease, and to correctly evaluate the airway of the patient. Standard radiographic assessments and CT examinations fall among these examinations. The CT enables us to see the formation of subcutaneous gas and abscess. The CT of our case helps us identify subcutaneous gas and micro-abscesses. Even though the CT image does not illustrate significant pathology, it helps guide surgical intervention [10,11].

The first step of treatment is checking the airway. Medical treatment requires a broad range of antibiotics together with fluid and electrolyte replacements. Surgical treatment requires a wide fasciotomy incision, exploring facial planes together with drainage, and excision of all necrosis tissues. Wound care should be carried out using local antibiotic pomades. Once culture results are obtained, antibiotic treatment should be modified to be effective on the effective organisms [5,12,13].

Major complications from NF are mediastinal involvement, septic shock, pleural effusion, lung empyema, airway obstruction, rupture of major vessels, brain abscesses, disseminated intravascular coagulation (DIC), sepsis, acute renal failure, and respiratory failure. Usually, such complications in immunocompromised patients, such as diabetes mellitus and cancer patients, lead to death [14,15]. Most deaths occur in patients in whom the infection is complicated by mediastinitis. Banerjee et al [16]. Found that 44% of the cervical NF cases in their series had mediastinal involvement, 38% of whom died of the disease. Infection can propagate from the head and neck to the mediastinum through the retropharyngeal or prevertebral space or can descend along the carotid sheath. Direct downward extension of fasciitis involving the pretracheal fascia can also result in mediastinal or pleural involvement [16].

**Conclusion**

NF is a disease with a high probability of mortality, which is compounded by several additional risk factors. The surgeon should suspect necrotizing fasciitis when dental abscess is resistance to the conventional therapy. Satisfactory outcomes can be achieved with early diagnosis and aggressive surgical therapy in concert with empirical antimicrobial therapy.

**References**
