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Non-odontogenic necrotising fascitis caused by acute sialadenitis- a rare case report

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Abstract

Necrotizing fasciitis (NF) is a rapidly progressive inflammatory infection of the fascia with secondary necrosis of the subcutaneous tissues. Here we present a rare case of a 72-year-old male patient with severe pain and swelling in the left parotid region for the past 15 days. The swelling was tense and brawny. Ultrasonography and contrast-enhanced computed tomography confirmed necrotic areas in the subcutaneous tissues. Antibiotics such as Vancomycin and Ceftazidine, as well as multiple debridement and fasciotomy, followed by split skin grafting, were used. The mortality rate for NF is around 9.8%, which indicates the need for aggressive surgical and medical management. Close monitoring and serial debridements and dressings are the golden rule for a better prognosis.

Keywords: Necrotising fascitis, Debridement, Immunocompromised, Split skin graft, Antibiotics, Hyper baric oxygen therapy.

Case Study

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1. Introduction

Necrotizing fasciitis (NF) is a rapidly progressive inflammatory infection of the fascia with secondary necrosis of the subcutaneous tissues. Hippocrates in the 5th century BC described NF as a complication of erysipelas. Jones et al., in 1871, called NF "hospital gangrene." Finally, Wilson coined the term "necrotizing fasciitis" in 1952, which refers to tissue death involving the facial plane. He also stated that the speed of the disease's spread is directly proportional to the thickness of the subcutaneous layer [1]. NF can be classified into four major types, namely type I is polymicrobial, type II haemolytic group A streptococcus, methicillin resistant streptococci, type III by the clostridium bacterias causing gas gangrene, type IV by marine and fungal infections [2]. The most common is type II that is caused by streptococci. The streptococcal bacteria produce M proteins and exotoxins. M proteins initiate inflammatory response with numerous cytokines IL1, 6, TNF alfa. Exotoxins destroy the neutrophils permitting bacterial growth and destroying tissues [3]. Dental infection is the commonest cause of NF in the head and neck region. NF of odontogenic origin is characterised by rapidly progressive bacterial infection in multiple fascial planes, leading to vascular compromise, thrombosis, and necrosis of adipose, cutaneous, and subcutaneous tissues. It could be as a result of polymicrobial or single organisms.Nonspecific symptoms of odontogenic infections such as cellulitis and periapical or cervical

abscess include swelling, pain, and trismus. Odontogenic infections with systemic comorbidities are liable to be victims of necrotizing infections. Early diagnosis of this entity can be cured better due to high vascularity in the head and neck region [4]. Patients suffering from various immunocompromised conditions are at higher risk of developing NF [5]. Systemic diseases like diabetes, renal failure, HIV infection, and malignancy cause impaired immunity. Impaired or diminished immunity permits various pathognomic bacteria to invade the host system. They manifest various fatal conditions, like NF. Here we present a case of NF in a patient with poorly controlled diabetes and the challenges faced in diagnosing and treating it.

2. Case report

A 72-year-old male patient came to the OPD with a complaint of pain and swelling in the left cheek region for the past 15 days. Initially, the swelling was small and persistent. The patient had taken medications from a nearby pharmacy for the previous 7 days before arriving at our OPD. The patient is unaware of the nature of the medications taken. Pain and swelling regressed with the medicines. Again, after 3 days, the pain became more severe and the swelling rapidly progressed to attain the present size. patient complained of difficulty swallowing and chewing. After reviewing his medical history, We discovered that he had been a known diabetic for the previous ten years without receiving proper treatment. According to his personal history, the patient has

been smoking 15 cigarettes per day and drinking 180 mL of alcohol per day for the past 30 years. Extraoral examination revealed a single, solitary swelling measuring approximately 5 X5 cm extending supero-inferiorly from the infraorbital rim to inferiorly 1 cm below the inferior border of the mandible. Antero-posteriorly, 1 cm away from the ala of the nose and 1.5 cm posterior to the earlobe. Earlobe appears to have been raised. With no other secondary changes or crepitus, the skin over the swelling appears tense and brawny. An intraoral examination showed no abnormality other than generalized chronic periodontitis. A Palpation of the entire oral cavity confirmed the same on hard tissue examination. The salivary flow was less and slightly yellow in color in comparison to the right side, where the flow was continuous and clear. From these findings, acute sialadenitis of the left parotid gland was considered a provisional diagnosis. [FigureA, B]. Laboratory investigations show WBC of 6500/mcL and an erythrocyte sedimentation rate of 65 mm after one hour. The patient was instructed to undergo contrast-enhanced computed tomography and seek physician consultation for emergency diabetes management. He was also prescribed Cefixime-CV, metronidazole, and analgesics. The next day, the patient reported even more swelling on the left side, extending into the submandibular and submental regions but not crossing the midline. On intraoral examination, the mouth opening was restricted to 3 cm. An irregularly blanched surface of about 1 x 1.5 cm was evident in the retromolar region at the conjunction of the maxillomandibular arch. On palpation, finger pressure applied to the blanched surface created a breach. Blood and a whitish purulent discharge started to ooze out [Figures C,D,E]. As the pus came out, the patient felt relieved, so more attempts were made to remove it nearly completely. After removal of the pus, a betadine mouth rinse was given, and the wound was left open for further draining and secondary healing. [Figures C,D,E].

Ultrasonography revealed enlargement of the left parotid gland. Diffuse hypoechoic areas with well-defined margins are seen within the parotid gland and surrounding areas of the masseteric space. Ductal dilations and calcifications were ruled out. A wide-bore needle was used to aspirate transcutaneously with ultrasound guidance. A cheesy white purulent aspirate was collected and cultured. The culture showed the presence of streptococcal strains. Contrast Enhanced computed tomography showed hypodense areas in the left parotid gland. The gland is enlarged, showing hypodense areas with highlighted walls following the contrast. a breach in the continuity seen in the posterolateral wall of the left parotid gland. Hypodense regions extend into the subcutaneous tissues via the masseteric space, involving the sternocleidomastoid muscle. [Figure F,G]. Patient was immediately admitted & started with I.V. Vancomycin & Ceftazidine, repeated debridement was done. Patient slowly recovered after multiple debridement and fasciotomy followed by split skin grafting [Figure H]. Follow up of 6 months was done with no remission.

3. Discussion

NF is the most common type of necrotizing soft tissue infection. These kinds of infections involving the head and neck region can be further classified into two subtypes. Type I involves the eyelids and scalp, where trauma could be the probable etiology. Types II are comparatively rare and involve the head and neck region, mostly from odontogenic infections. There can be other causes like peritonsillar abscess, osteoradionecrosis, pharyngitis, trauma, foreign bodies, cervical lymphadenitis, surgical wounds, tumors and various salivary gland pathologies. NF does not present any variations with age, gender, or race [6]. Clinical manifestations range from fever, erythema, pus formation, and sloughing to systemic toxicity. Beta-haemolytic streptococci produce enzymes and toxins from the subcutaneous tissue [1]. The toxins from the subcutaneous tissue travel through the superficial and deep planes and infect the facial plane, causing myonecrosis. Hence the patient presents with dusky, tense, insensate, crepitant, or mottled skin [7,8,9]. Patients presenting with rapidly progressive swelling should be kept in high suspicion for NF. Vigorous spreading swelling, erythema, fever, and crepitus on palpation are likely symptoms. Though the absence of crepitus does not rule out the diagnosis, Marking the extent of the swelling on the first visit and comparing it to subsequent visits would be useful in determining the speed and extent of the infection's spread. CT or CECT at an early stage may show soft tissue gas formation and also ascertain the extent of the anatomical sites involved in the infection. It is also useful in identifying vascular thrombosis [3, 7]. Secondary deposition of calcium in the necrotic tissues may lead to hypocalcemia. Leucopenia or leucocytosis could be other laboratory findings. However, the diagnosis of cervicofacial NF is mostly based on peculiar clinical behaviour.

Cervicofacial NF has significant morbidity and mortality due to four factors. Host immunity, polymicrobial etiology, delay in diagnosis, and inappropriate treatment planning. retrospective study found immunocompromised patients with NF have double the chance of an in-hospital mortality rate [10,11,12]. Aggressive treatment modalities are mandatory for the management of NF. Broad-spectrum IV antibiotics are the drug of choice for targeting the microorganisms. The commonly used antibiotics are metronidazole, penicillin, clindamycin, and ceftriaxone. Along with antibiotic therapy, repeated surgical debridements are done until fresh bleeds from the viable tissues are achieved. Close monitoring and serial debridements with dressings are the golden rule for a better prognosis^[8,9]. The mortality rate for NF is around 9.8%, which indicates the need for aggressive surgical and medical management. In addition to Medi-Surge combo management, hyperbaric oxygen therapy has proven to be significantly effective. In patients with resultant skin or soft tissue loss physical rehabilitation and reconstructive procedures may also be needed. Concurrent management of diabetes or any other immunosuppressive conditions are also warranted [13, 14, 15].

















Figure 1. A & B shows swelling in the parotid region extending below the inferior border of mandible, C,D,E: Intraoral sinus opening seen in the retromolar region (C), extra oral sinus opening (D), Orthopantomograph shows no relavant dental findings(E), F, G: Axial (F) and coronal section showing enlarged parotid gland in the left side (G), H: postoperative picture after skin graft

4. Conclusion

The main strategy for managing NF is to make an early diagnosis. The lack of understanding among dental practitioners, the rarity of this ailment, and patients who present with symptoms are obstacles to an early correct diagnosis and effective treatment ^[9]. Since odontogenic and salivary gland infections can spread quickly, doctors should report such cases and think about NF as a potential differential diagnosis.

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