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Case Report

Cervical Necrotizing Fasciitis: A Case Report

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Abstract

Necrotizing fasciitis is an infrequent infection characterized by rapid progression and potential fatal affecting subcutaneous tissue and fascia. The underlying predisposing systemic conditions are diabetes mellitus, old age, chronic renal failure, peripheral vascular disease. Patients associated with these infections often have history of trauma like insect bite, abrasion, burns and it slightly occurs more in male patients. It is one of the most challenging infections encountered by the surgeon. Due to difficulty in diagnosis as it is polymicrobial infection and its late management makes this condition more fatal.

Keywords: Necrotizing, polymicrobial, predisposing.

INTRODUCTION

Cervical necrotizing fasciitis is a rare but very severe infection that affects the soft tissues of the cephalic extremity. In ancient time they were described by Hippocrates and Galen, also by Avicenna and Renaissance surgeon Pare. In 1952, Wilson first used term necrotizing fasciitis to describe the feature of infection, necrosis of the subcutaneous tissue, fascia [1]. It is commonly known as flesh eating disease also referred as haemolytic streptococcal gangrene, meleney ulcer, acute dermal gangrene, hospital gangrene, suppurative fasciitis and synergistic necrotizing cellulitis [2]. It is a very rare multimicrobial disease commonly caused by streptococcal and staphylococcal species [3] which usually don't cause infection unless they enter the body through a cut or other break in the skin. It may lead to organ failure and death of patient [4].

Case Report

A female patient, 40 years of age admitted with a chief complaint of pain and swelling in right side of the face and unable to open her mouth since 12 days. Patient was apparently alright one month back then she felt pain and swelling in her lower right back teeth region. Pain was dull, localized, continuous in nature and aggravates while eating and relieved after taking analgesics. Swelling was initially small, soft localized no pain on digital palpation with no any discharge. Swelling subsides after taking medications (which patient was not known). Patient visited to some quack for the same. Then 12 days back she again felt pain in the same region which was sharp, radiating (to the right ear), continuous in nature, aggravates while eating, then she visited to same quack who treated her earlier for the same. He gave her some medications and ask for the rest. As the patient condition got worsen then she visited to hospital. At admission, she was febrile (Temperature 100° F. Fever on and off for 12 days, not associated with chills or rigor), hypotensive (BP 100/70 mm HG), Pulse (118 beats/ minutes) dehydrated and severe anemic (Hemoglobin 4 gram/dl). Relevant laboratory values were white blood cells count 19,800/cmm, neutrophilis 78%, eosinophilis 13%, lymphocytes 7%, random blood glucose 130 mg/dl. There was severe sloughing of skin and tenderness extending from right cheek region to chest wall of same side arising from a dental infection with fever, tender neck, chest swelling, dyspnoea and chest pain on inspiration. She also presented with tender, erythematous, crepitant swelling over the neck extending from the right submandibular region to the neck. The examination of oral cavity manifested foul,

putrid odour with multiple carious teeth and severe trismus. Teeth involved revealed deep carious lesion with tender on percussion. After initial establishment and antibiotics administration by parentral route(Amoxycillin 500 mg every 6 hours and Metronidazole 500 mg every 8 hours), DNS and blood transfusion of 8 units (During the hospital stay). Four hours after admission patient was taken to operating theatre. Under all aseptic condition painting (savlon and betadine) and draping was done, 2% lignocaine hydrochloride was administered at the surgical site. Aggressive surgical debridement of all involved tissues with a large amount (about 50 cc) of purulent fluid was drained before obtaining the aerobic and anerobic cultures. During the next 6 days she went to or two additional times for surgical debridement, drainage and washout of neck wounds. Case was treated successfully by early diagnosis, prompt surgical management and aggressive antibiotic therapy (Figure 1-5).

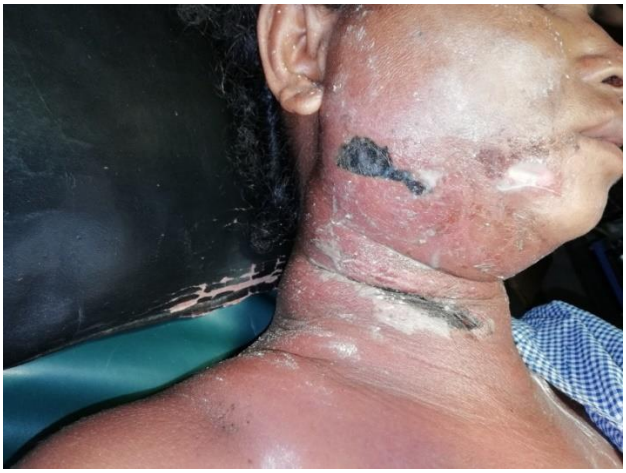

Fig-1: Pre-operative clinical picture

Fig-2: Intra-operative clinical picture showing deep lesion

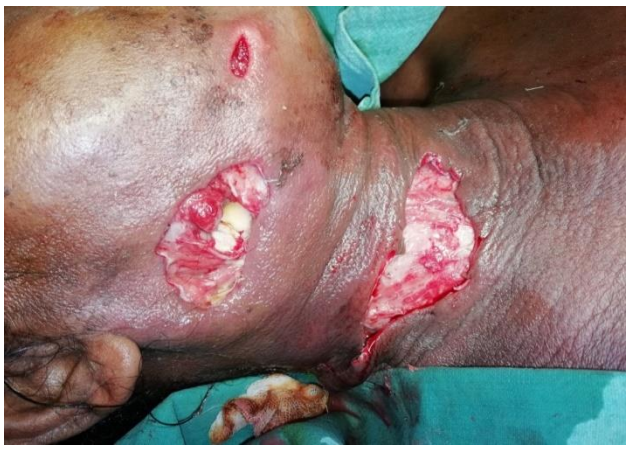

Fig-3: Intra-operative clinical picture showing superficial tissue

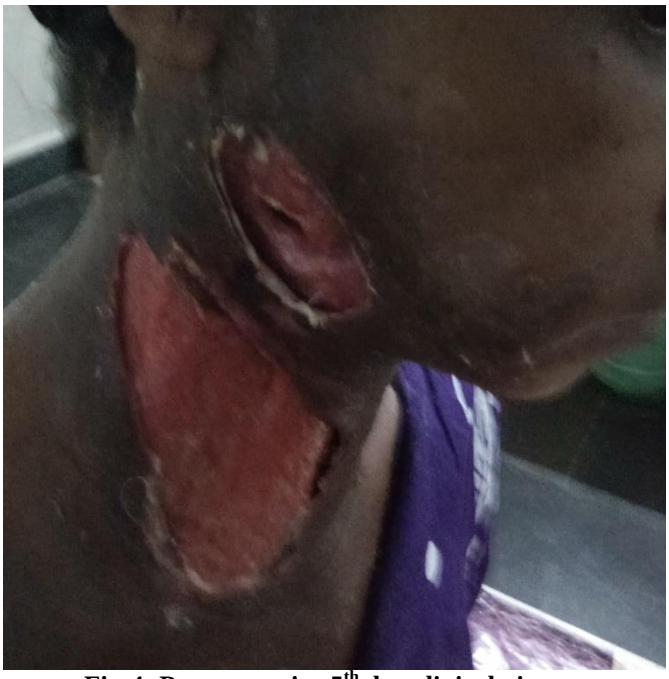

Fig-4: Post-operative 5th day clinical picture

Fig-5: Post-operative 14th day clinical picture

DISCUSSION

Necrotizing fasciitis is a progressive, fulminant bacterial infection of subcutaneous tissue that spreads rapidly through the fascial planes causing extensive tissue destruction. It can affect any part of the body and is the most serious presentation of necrotizing soft tissue infection. This entity is a rare consequence of odontogenic infection that can lead to involvement of neck, mediastinum and chest wall [5], although it is rare in cervicofacial region because of the higher vascularity in this region [6]. Prompt recognition and intervention is essential, as mortality is directly proportional time to intervention [7]. The most critical early distinctive feature of necrotizing fasciitis is a disproportionate pain in comparison with physical findings, on physical examination even a wound which seems small may lead to severe pain. Unlike cellulitis the necrotizing fasciitis begins at the junction between subcutaneous tissue and the deep fascia thus erythema and edema of skin is not remarkable in early stages. It is painful without clear demarcation between the normal and affected area. In cervicofacial necrotizing fasciitis, the patient presents with pain in head and neck, along with rapid exacerbation of symptoms including dysphagia, odontophagia, increasing pain, trismus, paraesthesia and dyspnea [8]. In latter stages 3 zones of demarcation are often seen where there is a central black necrotic area surrounded by purple zone, which is peripherally surrounded by erythematous zone. In very less time the patient starts developing manifestation of necrosis as a tendency to turn subcutaneous tissues into putrid, pulpy substances, fetid odour which is a hallmark of dead tissues [9]. Formation of insoluble gas leads to crepitation which represents presence of anaerobic

bacteria [10]. The patients present with blister and bullae formation in late phages of disease with extravasations of purulent discharge. When associated with diabetes mellitus it becomes life threating. Diabetes mellitus affects the microvascular circulation which limits the blood supply to surface and deep structures [11]. Necrotizing fasciitis may be aggrevated by a state of relative immunosuppression [12]. It can cause various complications such as airway obstruction, pneumonia, septic shock (i.e. tachycardia with hypotension, hypothermia, confusion, cardiac arrhythmias, metabolic acidosis, abnormal renal and liver function, coagulopathy and thrombocytopenia), jugular vein thrombosis and mediastinitis. The spread of infection in maxilla facial region to the mediastinum from space or prevertebral retropharyngeal shows a high mortality rate [13]. Necrotizing fasciitis is classified into four types based on the microbiology etiology [14]. Type I- It is polymicrobial and the most common type. Type II – is usually monomicrobial and due to gram positive organisms. Type III- is a gram negative monomicrobial infection, Type IV- is caused by fungal organism. Necrotizing fasciitis can also be classified into three stages based on clinical dermatological features. Stage I is defined with signs such as erythema, tenderness beyond the erythema, swelling and hot skin. Stage II is defined by the formation of skin bullae, blister and skin fluctuation. Stage III manifests with hemorrhagic bullae, cripitus, skin necrosis and gangrene. Pathogenesis: The fascial planes disintegrate with the ensuing necrosis with the release of tissue fluid [15]. Early in the development of the disease the veins that traverse the liquefying subdermal fat become inflamed and start to thrombose, which gives the skin first a red and then a mottled color. Later the arterial supply is also jeopardized and the skin becomes pale, which leads to necrosis and wet (coagulative) gangrene [16]. Perfusion is further reduced through arterial failure, the skin starts to blister. Thus the cause of classic spread of necrotizing fasciitis is avascularity of the fascial planes and this is probably the cause of their involvement and initially sparing the muscles and skin. The bacteria initiate an acute local inflammatory response within the dermis that is characterized by an intense polymorphonuclear infiltrate, focal necrosis, and micro- abscess formation. Sensory perception is lost as nerves [17] are destroyed and the wound weeps fluid from the underlying liquefaction. Biochemically serum creatinine kinase level [18] may be raised, which is indicative of myositis [19] or myonecrosis, and the effects of circulating toxins or ischemia. Hypocalcemia may be seen which is a sign of fat necrosis and calcium deposit in necrotic tissues. Bacterial infection, inflammation, and necrosis cause raised C-reactive protein (CRP). As in severe sepsis, abnormal renal function, hypoalbuminemia, hyponatremia, abnormal liver function, metabolic acidosis, and high serum lactate concentrations may occur [20].

CONCLUSION

Necrotizing fasciitis is a rare but potentially fatal condition. Delayed and inappropriate treatment contributes to high mortality and morbidity. A prior identification of the disease and instant medical, surgical and dental intervention may save life of patient. Also it is must to eradicate the source of infection if present as well as do the screening for predisposing underlying systemic conditions.

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