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REVIEW OF LITERATURE

NECROTISING FASCIITIS; IMPORTANCE IN MAXILLOFACIAL SURGERY A REVIEW WITH CASE REPORT

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ABSTRACT

Necrotising fasciitis is a rare soft tissue infection of soft tissues characterized by rapidly progressive, necrotizing process that has high rate of fatality. It can occur in an occult fashion wherein the necrosis progresses within the fascia and subcutaneous tissues leaving the upper muscular and dermal layer intact. Unless a high degree of suspicion is expressed by the surgeon this condition can go unnoticed and lead to death. In maxillofacial region, infections of odontogenic origin and peri-tonsillar regions are the main causative factors, responsible for this condition .The possibility of acquiring this disease is accentuated in patients with on low immunity status like diabetes. In this review we analyse a detailed review of this morbid disease and present two case reports with their effective management.

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INTRODUCTION

Necrotizing fasciitis is a rare life-threatening, multi-microbial soft tissue infection characterized by progressive, usually rapid, necrotizing process of the sub-cutaneous tissues and fascial planes, with resulting skin gangrene and systemic toxicity (Reed and Anand, 1992; Scott *et al*., 1994; Balcerak *et al*., 1988; Valko *et al*., 1990; Freischlag *et al*., 1985). By terminology, Necrotizing refers to "causing the death of tissues" and fasciitis signifies "inflammation of the fascia of a muscle or organ". Necrotizing fasciitis is rarely seen in head and neck region and when it so occurs, it tends to have a high morbidity owing to its proximity to vital structures in this region (Shaikh *et al*., 2012). It occurs as a spread of infection from teeth or pharynx. Occlusion of vascular structures, airway compromise, spread of infection to plural and mediastinal spaces are known to occur with the progression of the disease (Carter and Banwell, 2004; Vaid *et al*., 2002; Ali and Zayed, 1997). Patients with low immunity, especially those patients with Diabetes Mellitus, have been found to play a major role in initiation and progression of this disease (Shindo *et al*., 1997). Hence, early recognition and surgical intervention are the most important steps in determining the prognosis of this condition (Freischlag *et al*., 1985; Carter and Banwell, 2004).

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Synonyms

It has been also called by other names including hospital gangrene (Brooks, 1966), Meleney's gangrene (Meleney, 1924), hemolytic streptococcal gangrene, gangrenous erysipelas, flesh eating disease and synergetic necrotizing cellulitis (Shaikh *et al*., 2012; Carter and Banwell, 2004)

Historical background

Suppuration in the tissue beneath the skin *(sopha),* with danger of pus spreading to surrounding healthy tissues has been mentioned in SusruthaSamhita (Shaikh *et al*., 2012). This disease was narrated as early as 5th century BC by Hippocrates. An army surgeon, Joseph Jones, studied 'hospital gangrene' extensively and noticed 50% death ratein 2642 cases (Shaikh *et al*., 2012). The first reported case of this infection affecting the perineum was given by Baurienne in 1764 (Carter and Banwell, 2004). In 1883, a study conducted by Fournier who described and reported this infection in 5 young adults (Fournier's gangrene) (Shaikh *et al*., 2012; Carter and Banwell, 2004; Vaid *et al*., 2002). In 1924 Meleney reported on 20 cases of 'necrotising erysipelas' in China and was the first to

establish the haemolytic streptococcus as the aetiological agent (Shaikh *et al*., 2012) The term necrotizing fasciitis was given by Wilson in 1952 when he described the principle chareteristics of the disease which included inflammation and necrosis of fat and deep fascia which did not effect the muscles (Carter and Banwell, 2004; Vaid *et al*., 2002).

Epidemiology and risk factors

The Incidence of Necrotizing fasciitis is 0.40 cases per 100,000 people in US population (Shaikh *et al*., 2012). It has been found that patients aged 50 years and older are at higher risk with incidence levels reaching as high as 12 per 100,000 over the age of 80 (Moss *et al*., 1990). No significant difference in incidence is observed between men or women, but children and younger individuals are also at risk of developing necrotizing fasciitis, varicella infection being a common predisposing factor (Moss *et al*., 1990; Lancerotto *et al*., 2012). Although it can occur anywhere in the body, the condition is more commonly seen in the upper (48%) & lower extremities (28%), abdominal wall & perineum (21%) (Lancerotto *et al*., 2012). Head and neck region has occurrence rate of 5% with the commonest cause of cervical necrotising fasciitis being dental infections (Deganello *et al*., 2009). The other causes include peritonsillar and pharyngeal abscesses, osteoradionecrosis, neck surgery &steroid neck injections (Lancerotto *et al*., 2012; Deganello *et al*., 2009). Necrotizing fasciitis has always been associated with at least one disease that increases their susceptibility to infection (Vaid *et al*., 2002; Moss *et al*., 1990). Apart from Diabetes, the other diseases thatare implicated with increased incidence of causing necrotizing fasciitis include chronic renal failure, peripheral vascular disease, alcohol abuse, malignancy, immunosuppressive therapy, HIV infection, and chronic cardiac and pulmonary diseases (Carter and Banwell, 2004).

Microbiology

Necrotizing Fasciitis in most cases is a polymicrobial infection.A, β-hemolytic streptococci and staphylococci were previously thought to be the main agents in this condition. However research shows that anaerobes too includingthose such as Provetelladentiola, Bacteroidesfragilis, also playa very important role. Other organisms such as Streptococcus, Staphylococcus, Enterococcus species, Lactobacillus, and Corinebacterium seem to be the most common aerobic organisms (Lancerotto *et al*., 2012; Wang and Lim, 2014). Gram negative aerobic pathogens such as *E. coli, A. hydrophila, V. vulnificus are encountered more frequently as well. More recently, an increase incidence of* methicillin-resistant *Staphylococcus aureus* (MRSA) and methicillin-sensitive *Staphylococcus aureus* (MSSA) species have been frequently isolated from necrotizing fasciitis infections (Wang and Lim, 2014).

Clinical features

Necrotizing fasciitis is can be diagnosed by a plethora of findings such as Local erythema and swelling accompanied by pain and raise in local temperature, with an ill-defined border is seen initially. Infective process can progress within a few hours and local pain could be replaced by numbness or analgesia as a result of cutaneous nerve destruction. Demarcation between the involved and uninvolved tissue also develops in most cases. As the disease progresses, it assumes a pale purple hue, which later tends to become hemorrhagic and gangrenous in nature. Late signs of necrotising fasciitis include large hemorrhagic bullae, skin necrosis, fluctuancy, crepitus and sensory and motor deficits. Surgical emphysema may be elicited during palpation which may indicate presence of gas-forming organisms. Based on the clinical signs NF can be staged as Stage I (early) includes clinical signs of tenderness, erythema, and swelling. Stage II (intermediate) involves blistering or bullae & Stage III involves crepitus, skin anesthesia, and skin necrosis (Vaid *et al*., 2002; Lancerotto *et al*., 2012; Wang and Lim, 2014; Weiss *et al*., 2011).

Investigation and diagnosis

Laboratory findings in necrotizing fasciitis are non-specific but with increased white blood cell count (WBC), Creatine phosphokinase, Albumin, Sodium, prothrombin time, or activated partial thromboplastin time have been suggested as useful parameters to identify this condition but are not exclusive. Wall *et al*. suggested White Blood Cells> 15.4 X 10^9 and Sodium levels of<135mmol/L, the former more important than the latter, as laboratory parameters to distinguish necrotizing fasciitis from non-necrotizing soft tissue infections (Weiss *et al*., 2011; Harrison and Kapoor, 2016). In 2004, Wong *et al* published the results of their Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) (Table-1) which is a useful diagnostic adjunct, but less useful in achieving an early diagnosis (Harrison and Kapoor, 2016; Wong *et al*., 2004).

Imaging

Currently along with lab-investigations Ultrasound (US), Computed tomography (CT), and Magnetic Resonance Imaging (MRI) are recommended diagnostic methods used (Kehrl, 2014) diffuse thickening of fascial planes, abnormal fluid collections along the fascial plane and irregularity of the fascia can be detected using ultrasonography. In cervical necrotizing fasciitis, Computerized Tomographic (CT) scanning is useful in demarcating the spread of infection, along the fascial planes (Tsai *et al*., 1996; Maisel and Karlen, 1994). Magnetic Resonance Imaging (MRI) with sensitivity 90% - 100% and specificity of about 50% - 85% is considered by many as the diagnostic test of choice radiologically, in differentiating necrotizing and non-necrotizing soft tissue infections Hyper-intense signal on T2-weighted images at the deep fascia and within muscles with Soft tissue thickening is characteristic feature of the MRI finding that would help to establish the diagnosis. MRI provides a better tissue contrast, is more sensitive to soft-tissue fluid accumulation, with increasing visual access of pathologic process (Edlich *et al*., 2010; Green *et al*., 1996).

Histopathology

In order to establish a definitive diagnosis, the ideal location to retrieve a biopsy specimen is from the interface between live and dead tissue. Increased leukocytic infiltration edema of superficial fascia, reticular dermis & subcutaneous fat of tissue with necrosis of the superficial fascia is characteristic histologic feature of necrotizing fasciitis while angiothrombosis of the vessels supplying the skin is another noticeable microscopic feature too. The spread of necrosis is observed sequentially in the following manner -initial horizontal spread through the superficial fascia, then later proceeds to involve the

Classification

Types of NF incidence	Aetiology	Organism	Clinical progress	Outcome/mortality
Type I (70-80% of cases)	Immunocompromised patients/complicated abdominal surgery/perianal abscess Polymicrobial synergistic, often bowel flora derived	Mixed aerobes and anaerobes (*E. coli, Psuedomonasspp, Bacteroides*	More indolent better prognosis, easier to recognize clinically	variable: depends on underlying comorbidities
Type II (20-30% of cases)	skin or throat derived direct inoculation from trauma/intramuscular injection pharyngitis/vaginitis/proctitis often monomicrobial	Usually group A – β hemolytic streptococcu Occasionally *S .aureus*	Aggressive protean presentation easily missed. Rapidly progresses to myonecrosis	Depends if associated with myositis or toxic shock syndrome(STSS) STSS negative- <32% STSS positive >67%
Type III (commoner in Asia)	Gram negative, often marine related organisms	*Vibrio sppmainly Aeromonashydrophilia Enterobacteriaceae*	seafood ingestion or water contamination of wounds	30-40% despite prompt diagnosis and aggressive therapy
Type IV (fungal)	Usually trauma/burns Associated immunocompetent patients	*Candidia spp*. Immuno-compromised patients. Zygomycetes in immunocompetent patients	Aggressive with rapid extension especially if immune-compromised	>47%(higher if immunocompromised)

muscles and the overlying skin. The degree of infection is generally larger than what is exhibited on the skin surface (Stamenkovic and Lew, 1984; Barker *et al*., 1987).

Pathophysiology

Necrotizing fasciitis is characterized by a minor injury fast progressing to necrotizing infection of the superficial fascia. It spreads through the fascial planes without involvement of skin. The bacteria accumulates at the site of initial trivial injury & into the superficial fascia, proliferating and producing an enzyme called hyaluronidase (Andreasen *et al*., 2001; Majeski and Majeski, 1997). This enzyme catalyzes, degradation and necrosis of the fascial planes; promoting growth of bacteria and enhancing spread of infection. The onset of vertical spread of infection marks the involvement of the deeper fascia, muscles, subcutaneous tissues and skin signifies the further progress of disease (Majeski and Majeski, 1997). Infiltration of leukocytes into the deeper fascial planes and thrombosis of arteries and veins results in blocking of nutrient vessels of the dermis leading to ischemia and necrosis of the skin above (Cheung *et al*., 2009).

Management

Administration of broad-spectrum antibiotics, followed bysurgical debridement after early diagnosis is the main line of treatment for necrotizing fasciitis. The first step of management may involve tracheotomy in order to secure the airway, combination of electrolyte fluids, vasoactive drugs & insulin before any surgical intervention is carried out (Durrani and Mansfield, 2003). In the early stages (Stage I) it may be difficult to diagnose necrotizing fasciitis but when it involves deeper fascial planes, surgical intervention must be carried out to establish diagnosis that is to be followed by early debridement that provides a better prognosis (Durrani and Mansfield, 2003; Ord and Coletti, 2009).

Surgical Debridement

Timing and adequacy of surgical debridement is a crucial factor influencing the outcome and must be done at the earliest. Surgical debridement is a mandatory life-saving step and should be performed as soon as possible. There is a nine-fold increase in mortality rate when debridement is delayed for more than a day after hospital admission (Wong *et al*., 2004) Debridement must be carried out until brisk bleeding occurs from adjacent tissues and underlying muscles, if they are involved, further removal of necrotic tissues decreases the bacterial load, thus reducing the inflammation that leads to recovery. Anaerobic bacteria may also be lysed as surgical exploration exposes tissues to oxygen. In areas where infections have extended to pocket areas in subcutaneous & / or sub-muscular regions, blunt probing of the wound multi-directionally is generally advocated. Wound debridement should be repeated subsequently in the next 24 - 48 hours, or longer, depending on the viability of the tissues and severity (Durrani and Mansfield, 2003; Ord and Coletti, 2009; Sarani *et al*., 2009).

Antibiotic Therapy

Broad-spectrum antibiotics must be initiated when necrotizing fasciitis is suspected. Multidrug combinations have also been prescribed including high-dose Clindamycin/ Penicillin/ Aminoglycoside / Metronidazole for coverage of Gram-negative bacteria including penicillin-resistant staphylococci. Antibiotics to combat Methicillin resistant streptococcus aureus such as Linezolid or Daptomycin are often considered when deemed necessary (Miller *et al*., 2005). Hyperbaric oxygen (HBO) therapy, Intravenous immunoglobulin therapy are gaining importance in the treatment of necrotizing fasciitis as adjuvant modes of managing this condition.

Case Report 1

A 45 years old female patient reported with swelling on the right side of face with existing extra-oral sub-mandibular and sub-mental incision, previously treated elsewhere with an incision and drainage surgical procedure. The swelling had increased since then with purulent discharge seen from site of incision. The patient's right side of the face, neck and upper part of the chest were swollen with tense, warm skin and crepitus could be felt. Per oral examination was not possible owing to severe trismus and pain. There was a bilateral, tender, diffuse, edematous swelling on lower third of the face and neck region with local raise in temperature. Patient had fever of 100 °F. The patient was admitted in the ICU and intravenous line was secured and carefully monitored. Her random blood sugar was 518mg/dl. Intravenous antibiotics such as Ceftriaxone, Cloxacillin and Metronidazole were advised along with Intravenous hydration. Medical consultation was sought regarding her uncontrolled diabetes and soluble insulin was started with frequentmonitoring of

blood and urine sugar and serum electrolytes. An informed consent was obtained and the patient was taken up for debridement under general anaesthesia. With a patent airway being maintained with an elective Tracheostomy. There were plenty of dirty grayvery foul smelling pus (Figure 1) pockets in almost all the cavities of the neck extending above up to the zygomatic space. Wound debridement and drainage were performed (Figure 2). Post-operatively; the wound was cleaned twice daily with hydrogen peroxide and povidone-iodine (Figure 3). The wound healed by secondary intention after 2 weeks.

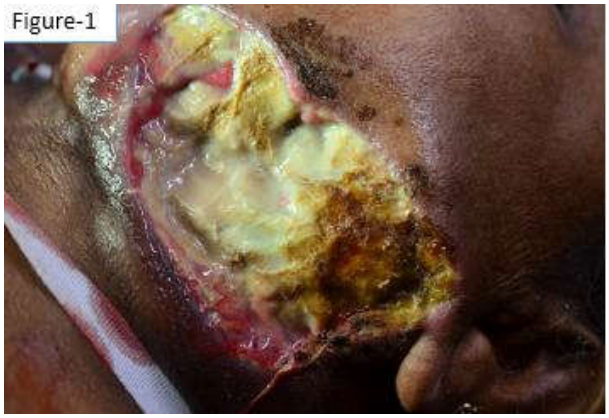

Figure 1. Pre-operative

Figure 2 a) Intra operative debridement in progress

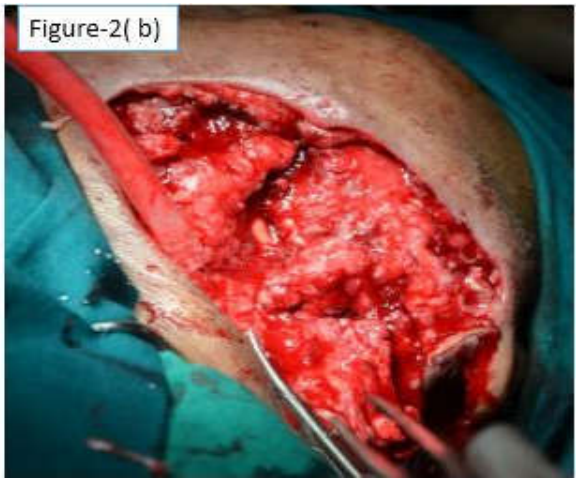

Figure 2. b) Debridement completed

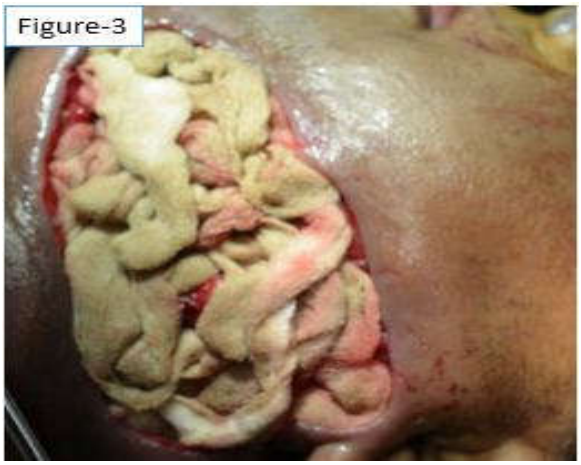

Figure 3. Postoperative wound dressing

Case Report 2

A 70 year old male patient reported with bilateral swelling in the submandibular and submental region with history of 1week duration. There was draining sinus in the left submandibular region with pus discharge. Skin over the swelling appeared edematous and with bluish black discoloration in the midline. The other medical parameters were investigated and found to be normal. Patient was febrile with elevated WBC count, had difficulty in swallowing. Patient was admitted and the pus discharge was sent for culture and sensitivity testing. On the 3rd day of hospital admission, ulceration of the skin with copious pus discharge were noticed in the midline. The skin around the ulceration appeared necrotic. Patient was taken up for debridement of necrotic tissue and 7cm and 5cm of skin and underlying fascia was debrided. Culture reported streptococcus sensitive to amoxicillin with clauvinic acid. On the 6h post-operative day, the wound was grafted with split thickness skin graft which had taken up. The patient recovered uneventfully within 3 weeks.

Conclusion

An early diagnosis and vigorous surgical intervention are the key factors in the management of necrotizing fasciitis. Initially the patient may not present with any distinguishing features of necrotizing fasciitis. Therefore it is essential to have a highly suspicious view especially when diabetic or immuno-compromised patients present with symptoms of pain, inflammation, and infection with no apparent predisposition. Necessary resuscitation, Radical surgical debridement and secondary exploration/debridement when needed, coupled with appropriate antibiotic therapyplay a very important role in management of Necrotizing Fasciitis.

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