Cervical Necrotizing Fasciitis: A case report

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ABSTRACT

Necrotizing fasciitis is a rare but rapidly progressive condition, with a high morbidity and mortality rate. This rapidly spreading soft tissue infection rarely occurs in the head and neck region, and when it does it is most caused by odontogenic origin. A variety of host factors such as immune status, hygienic practices and socio-economic status are role players in the disease process. This case report documents a 38-year old male who presented with cervical necrotizing fasciitis of odontogenic origin. The patient was managed and stabilized through removal of the necrotic tissue, extraction of all carious teeth, and optimization of the overall medical health status of the patient. HIV is widely prevalent in South Africa and therefore the patient consented to HIV testing, this alluded to an undiagnosed HIV positive status.

Uncontrolled HIV greatly suppresses the immune system and subsequently exacerbating the disease process thus its management is highlighted below. The patent is currently awaiting reconstructive plastic surgery which can only be addressed once his viral load is controlled. In the meantime, he has been educated on how to keep the wound clean and the importance of maintaining a healthy lifestyle, this includes HIV education and dietary information. Due to the poor prognosis often associated with necrotizing fasciitis, it requires early intervention, and a multidisciplinary approach is often needed.

INTRODUCTION

The earliest descriptions of necrotizing fasciitis date back to Hippocrates himself in 500 BC.¹ Necrotizing fasciitis is a rare polymicrobial soft tissue infection

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which rapidly progresses via haemotogenous spread, tracking along fascial planes. This infection occurs more frequently in the abdominal wall, perineum, and extremities; it is a rare find in the head and neck region. Its rapidly progressive nature is the greatest concern, resulting in a high mortality rate.²⁻³ It mainly affects those of immunocompromised and low socioeconomical status.²⁻⁶ According to the Joint United Nations Programme on HIV/AIDS (UNAIDS) it's estimated that 7 800 000 people in South Africa are living with Human Immunodeficiency Virus (HIV).⁷

Due to the high prevalence of HIV in this population, knowledge and swift intervention of this condition is of importance . 7 The microbiologic etiology distinguishes the four types of necrotizing fasciitis as explained in the table below by Miller et el.⁴

Туре	Microorganism	Notes
I	• • Polymicrobial	Mixed anaerobes and aerobes Better prognosis 70-90% of cases in head and neck region
II	• Monomicrobial	Most commonly streptococcus or staphylococcus Most commonly seen in the limbs
Ш	Clostridium •	Gas Gangrene Crepitis
IV	• Fungal	Most commonly candida Most commonly seen in immunosup- pressed host Aggressive with rapid extension

There is no clear consensus on the management of these cases however the literature does highlight a rough outline of management progression.⁸ This includes early diagnosis through clinical examination and investigations such as appropriate X-rays, contrast enhanced computer tomography (CECT), microscopy culture and sensitivity (MC&S) and blood cultures. Culture guided antibiotic therapy directed by the investigations, removal of causative factors and aggressive surgical debridement of the affected area are sequential steps in the management of necrotizing fascitis.⁹

CASE

A 38-year-old coloured male with no known medical history presented to the Maxillofacial and Oral Surgery clinic at Pelonomi Tertiary Hospital in the Free State,

560 > CASE REPORT

with a seven-day history of necrotic tissue localized to • the submental and submandibular regions.

Associated symptoms include dysphagia, odynophagia, and malaise. The patient's social history was negative for alcohol, smoking and illicit drug use. Vital signs were all in normal range. On extra-oral examination significant areas of necrotic tissue with suppuration were localized to the bilateral anterior triangles of the neck extending from the inferior border of the mandible to the hyoid bone anterior to the sternocleidomastoid muscle as shown above in Figure I and II. Intra-orally, there was trismus, multiple carious teeth and overall extremely poor oral hygiene.

Due to the extensive clinical presentation various blood tests were done to assess the overall health oof the patient. These results indicated an undiagnosed HIV positive patient with a viral load of 602 000 copies/ml and an absolute CD4 count of 25 L cells/ ul. Subsequent testing for TB, Hepatitis B and C were negative.



Figure I and II: Clinical photograph showing the initial presentation before treatment

Orthopantomogram (Figure III) showed multiple carious teeth and root rests and early-stage osteomyelitis A contrast enhanced computer tomography (CECT) was taken of the head and neck to track the spread of the infection.



Figure III: Orthopantomogram (Figure III) showed multiple carious teeth and root rests and early-stage osteomyelitis

It showed:

- Submental soft tissue defect involving floor and anterior cervical triangle up to level of laryngeal cartilage;
- Platysmas, geniohyoid and digastric muscle necrosis;

- Mild periosteal reaction over inferior aspect of mandible with chronic mandibular osteomyelitis
- (dental origin);
- Hyoid bone and laryngeal cartilage were intact;
- Tongue base demonstrated early necrosis with no deep extension;
- Submental salivary glands are absent and
- Necrotic tissue debris over epiglottis, with preservation of epiglottic cartilage and hypothyroid cartilage

The report concluded that there was necrotizing fasciitis of the oral floor extending to hyoid bone with chronic dental origin mandibular osteomyelitis.

TREATMENT

The infected area was debrided under local anesthesia (Figure IV), a pus swab and tissue sample were obtained.

The MC&S of the pus swab and tissue sample showed numerous gram-positive bacilli and scanty grampositive cocci and gram-negative bacilli. The bacterial culture produced a heavy growth of Citrobacter freundii (CITFR); this could be due to the prolonged exposure to broad spectrum antibiotics as the patient reported visiting the dentist and getting antibiotics for toothache. A moderate growth of Streptococcus anginosus (STRAN) and Streptococcus alpha-haemolytic (STRAH) was also demonstrated. The antibiotic sensitivity analysis showed resistance to ampicillin, amoxicillin, and amoxicillin – clavulanic acid. Therefore, once the cultures came back, his antibiotic regime was changed from Augmentin, Flagyl and Gentamycin to Ciprofloxacin.



Figure IV: shows affected area immediately after local debridement

A full dentectomy was done to remove the odontogenic cause of infection.

Internal medicine commenced treatment of the patient's HIV infection with Acriptega. This is an antiretroviral drug that contains Dolutegravir, Lamivudine and Tenofovir. The latest treatment guidelines advise that all patients newly diagnosed with HIV, be treated with a Dolutegravir based antiretroviral regime.¹⁰

CASE REPORT < 561



Figure V: Affected area 3 days after debridement



Figure VI: Affected area 6 days after debridement

The affected area was cleaned twice a day with chlorhexidine and covered with gelonet, drawtex and wound dressings. The patient was monitored for 4 weeks.

Speech therapists and dieticians were consulted due to the patient having a communication between the intraoral and extra-oral environment. The speech therapists conducted a swallow trail and noted that the patient had a poor pharyngeal - laryngeal transit and thus





Figure VII: Affected area 3 weeks after debridement Figure VIII: Affected area 4 weeks after debridement

needed to be monitored. Accordingly, the dieticians opted for a liquid diet at first and then progressed to a soft diet to ensure nutritional sufficiency.

Four weeks later (Figure VIII), a biopsy was done and confirmed a microbial count of less than 1: 100 000. The patient was then referred to plastic surgery for closure of the affected area. Due to the patient being newly diagnosed with HIV, plastic surgery decided to wait until the patients CD4 count is at an acceptable level to prevent rejection of the tissue flap.



Figure IX: Affected area 2 months after debridement

DISCUSSION

According to Kaul et al, the global incidence of necrotizing fasciitis is 0.4 case per 100 000 population.¹¹ The largest study from a single health center in Nigeria reported that there is a definite rise in prevalence of necrotizing fasciitis as they have seen a gradual increase to around 4 cases a year.⁶

A systematic review conducted by Dhanasekana et al on necrotizing soft tissue infections from across the globe, found that the most common organisms involved are Staphylococcus aureus followed by Streptococcus pygenes and E. coli in North America, Asia, Middle East, and Africa. Interestingly, 16% of necrotizing soft tissue infections were accounted for by Methicillinresistant Staphylococcus aureus.12

Trauma is the predominant aetiology of necrotizing fasciitis. Less common causes include odontogenic infections, tonsillitis, and nasal malignancies.13

Common clinical features include cellulitis and erythema, which results in the skin of the affected area becoming painful, necrotic and the patient developing a fever, sepsis or multi organ failure.8 As the infection spreads via the fascial planes the skin will depict various stages of decomposition such as mottling, blistering or a rash. Microscopically, the tissue will show coagulative necrosis due to inadequate blood supply.4 Goh et al concluded that characteristic features of patients with necrotizing fasciitis included disproportionate pain, failure of improvement to broad spectrum antibiotics and gas present in 24.8% of patients.14

562 > CASE REPORT

Compromised immune systems due to diabetes, HIV and poor living conditions can exacerbate the spread of necrotizing fasciitis and make the patient more susceptible to developing it.⁸ Thus, it is always important to examine the patient for common comorbidities. Patients with necrotizing fasciitis are likely to require a multidisciplinary treatment approach spanning across surgical, medical, diagnostic and allied health care disciplines.

CONCLUSION

In conclusion, early detection and rapid response with the removal of the local aetiology, culture guided antibiotic therapy and aggressive debridement are essential to patient outcome. Systematic diseases which contribute to disease susceptibility and progression, should be managed. Overall, a multidisciplinary team is required to successfully manage the patient.

Ethical Approval was granted by the University of Pretoria

The authors declare no conflict of interest and the above case report aligns itself with the declaration of Helsinki.

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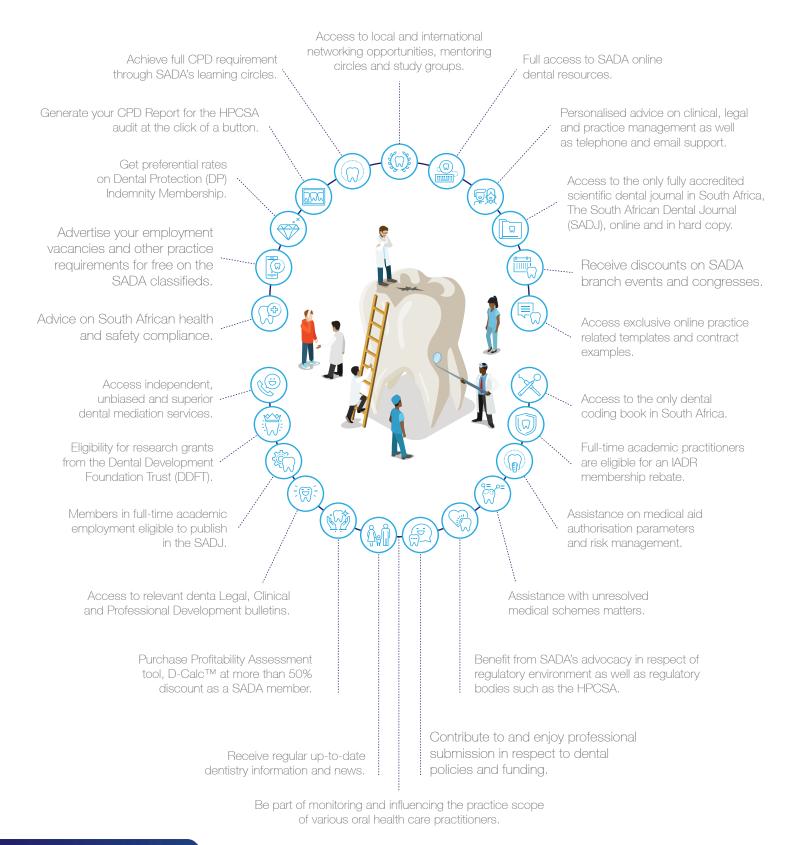
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