Peer-reviewed paper; submitted May 2018; accepted January 2019

A case of cervical necrotising fasciitis following dental extraction

Fisher C, Singh T

Abstract

There are many possible complications of dental extractions in a medically compromised patient. While most dentists would look at a patient's medical history and identify issues with anticoagulants, bisphosphonates, or immunosuppression – it is unlikely that necrotising fasciitis would be considered as a potential complication. Necrotising fasciitis is a rare and severe soft tissue infection characterised by rapid and progressive spread to the fascia and subcutaneous tissues. The condition is rare in the head and neck region but the most common cause is odontogenic infection in those predisposed. Despite aggressive treatment, fatality from necrotising fasciitis remains high. Given its association with odontogenic infection and facial trauma, relevant predisposing factors and guidelines for practitioners are presented. We report a case of cervical necrotising fasciitis following dental extraction and its surgical management.

Introduction

Necrotising fasciitis is a destructive and rapidly progressive soft tissue infection with significant morbidity and mortality. Cervical necrotising fasciitis (CNF) is necrotising fasciitis of the head and neck region. Necrotising fasciitis in this locality is rare due to the robust blood supply, and only accounts for 1-10% of cases (Lin et al., 2001). When CNF does occur, it is most commonly caused by odontogenic infection, followed by oropharyngeal infection and facial trauma (Sarna et al. 2012; Oguz and Yilmaz, 2013; Gunaratne et al., 2018). Diagnosis is challenging in the cervical region where signs and symptoms can be attributable to other soft tissue infections (Sarna et al. 2012; Bayetto et al. 2017). Initial presentation may include a patchy erythema, mimicking cellulitis or erysipelas. Tenderness in the region may turn to intense pain disproportionate to or in the absence of clinical findings. As infection spreads through the fascial spaces, the patients may become febrile and develop typical signs of septicaemia (Oguz and Yilmaz, 2012). As underlying small vessels supplying superficial nerves are thrombosed, the affected area may become insensate. The development of anaesthesia can antedate the appearance of dusky skin necrosis, and signifies the presence of CNF rather than cellulitis. On palpation, there can be crepitation of the soft tissue due to subcutaneous bacterial gas production (Krenk et al., 2007). CNF is rapidly destructive due to the ease with which infection can spread through the deep fascial planes of the neck. If the disease progresses into the thorax it is termed descending necrotising mediastinitis. Without intervention, the patient gradually goes into septic shock and multi organ failure. Mortality rates for CNF range from 7 – 64% depending on the extent of neck involvement and progression into the thorax (Sarna et al. 2012).

The microbiology in the majority of CNF infections is polymicrobial with suggestion of a symbiotic relationship between aerobic and anaerobic pathogens (Bayetto et al., 2016). The predominant organism is streptococcus (Gunaratne et al., 2018). An association between Prevotella species and Streptococcus milleri and/or oralis has been described in odontogenic cervical necrotising fasciitis (Fihman et al., 2018; Guranaratne et al., 2018). Predisposing factors include diabetes mellitus, obesity, alcoholism, renal failure, immunosuppression, smoking, and gout (Murry et al. 2012; Kulasegaran et al. 2016; Bayetto et al., 2016; Gunaratne et al., 2018). The use of non-steroidal anti-inflammatory drugs and necrotising fasciitis remains controversial (Forbes and Rankin, 2001; Mitchell et al. 2011). Necrotising fasciitis has a significantly higher incidence in Maori and Pacific populations compared with other ethnic groups in New Zealand (Tiu et al. 2005; Das et al. 2011; Nisbet et al. 2011; Das et al. 2012). In New Zealand, a recent single-centre retrospective case review documents only 7 cases of CNF over an 11-year period in the South Auckland population (Kulasegaran et al. 2016).

Case Report

A 35-year-old Maori male underwent extraction of tooth 46 by his dentist. His medical history included hypertension, insulin-dependent type 2 diabetes mellitus, spondyloarthritis, and history of pancreatitis, gout, and cholecystectomy. His medications included Lantus insulin, Novorapid, Omeprazole 20mg daily, Cilazapril 5mg daily, Lipitor 20mg daily, Bezalip 400mg daily, Folic acid 5mg once weekly, Paracetamol 1g QID, Naproxen 1000mg daily, and Methotrexate 10mg once weekly. He had an allergy to penicillin. The extraction was uncomplicated and no antibiotics were prescribed post-operatively.

The patient presented to a local hospital 3 days later with pain and swelling of the right submandibular region. He was febrile (39°C) and had significant trismus. Blood samples indicated elevated C-reactive protein (CRP) (143 mg/L: normal reference range 0-5 mg/L) and white cell count (WCC) (15 x10^9/L: normal reference range

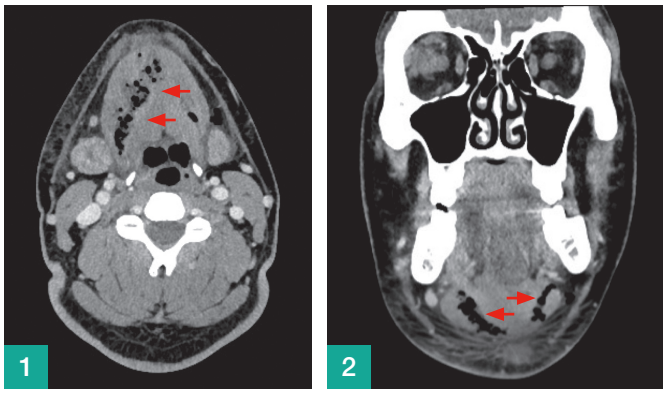

Figure 1 Axial CT scan demonstrating gaseous formation in the submandibular and submental region indicated by red arrows.

Figure 2 Coronal CT demonstrating gaseous formation in bilateral submandibular regions indicated by red arrows.

4-11 x10^9/L) He commenced intravenous fluids and clindamycin prior to transfer to a larger centre under Oral and Maxillofacial Surgery care. A computed tomography (CT) of the head and neck demonstrated fluid collection and gas in the right submandibular and submental spaces, and spreading to the contralateral side (Figures 1 and 2). Ciprofloxacin was commenced in recognition of his high risk of developing CNF, as per the hospital guidelines.

An incision and drainage was performed under general anaesthetic. Bilateral submandibular, submental, sublingual, right buccal space and right pterygomandibular spaces were explored. Purulent and haemoserous fluid was expressed from all spaces and multiple swabs were sent for microscopy, culture, and sensitivity. The tissues were irrigated with betadine, peroxide, and saline. The 46 socket was explored and debrided, finding a small communication from the lingual cortex to the neck incision. Several multi-tubular drains were inserted. The patient was extubated and monitored in the high dependency unit (HDU), only requiring a small amount of oxygen support post-operatively.

24 hours later he developed upper airway obstruction, stridor, and acute respiratory distress. He also had increased erythema extending from bilateral submandibular region to the superior aspect of the sternum. Nasoendoscopy showed new oedema of the epiglottis and oropharynx. An emergency intubation was performed to maintain the patient's airway. CT investigation demonstrated no discrete collection, but persisting areas of gas along the right mylohyoid muscle tracking posteriorly up to the skull base, and in the bilateral deep neck spaces. Significant inflammation of the subcutaneous tissues down to the sternum was noted.

The patient returned to theatre and underwent an apron neck incision. Significant areas of necrosis were present extending bilaterally to the deep cervical fascia and underlying muscle. Extension on the right side appeared to go down the thoracic inlet towards the sternum but did not extend into the chest cavity (Figures 3 and 4).

The necrotic tissue was debrided back to healthy tissue. Combinations of saline, peroxide, and betadine were used to irrigate the tissues. Once debrided, the surgical team rescrubbed to perform a tracheostomy. Betadine-soaked packs were inserted and secured. The patient was transferred to the intensive care unit (ICU). Radiopacity in the chest cavity was viewed on CT and Cardiothoracic Surgery input was sought regarding possible spread to the mediastinum. No drainable collection was discovered on repeat imaging. Cultures of tissue specimens demonstrated polymicrobial growth of gram negative bacilli, and gram positive cocci and bacilli. Organisms isolated were Streptococcus mitis/oralis, Granulicatella adiacens (previously Streptococcus adjacens), Staphylococcus epidermidis (coagulase negative staphylococcus), and Prevotella denticola from enrichment. Actinomyces cultures were negative. On advice from the Infectious Diseases team, the antibiotic regime was altered from Ciprofloxacin to Cefipime 2g IV BD, Metronidazole 500mg IV TDS, and a Vancyomycin 20mg/kg bolus followed by 1.5g IV BD. The patient was stepped down from ICU to HDU the next day.

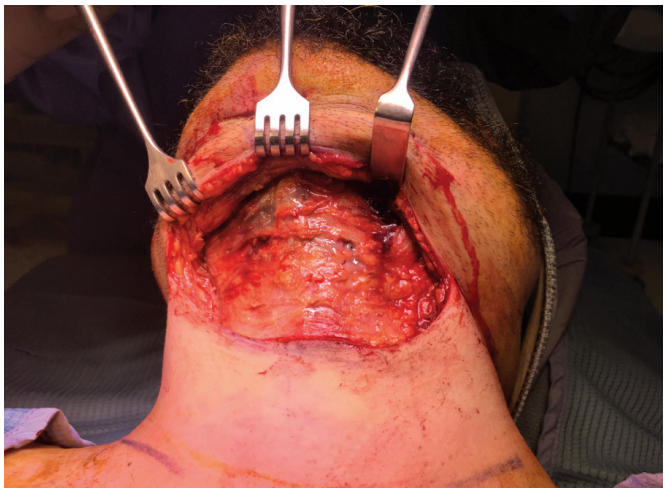

Figure 3 Intra-operative clinical photograph of necrotic anterior cervical tissues prior to surgical debridement.

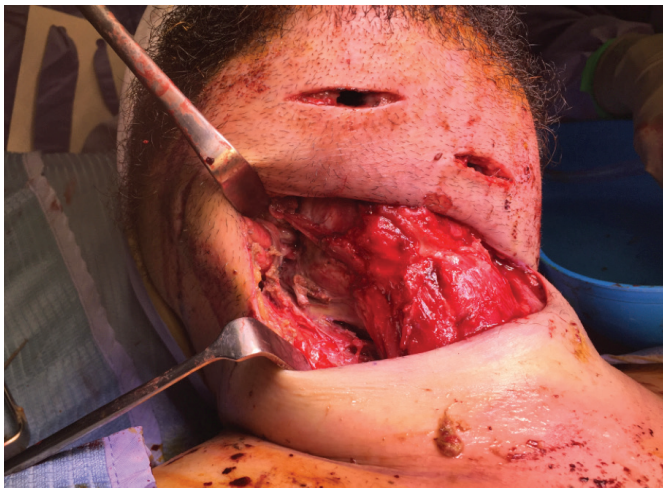

Figure 4 Intra-operative clinical photograph of necrotic submandibular tissue extending posteriorly, and anterior tissues following surgical debridement.

A repeat washout was planned and performed 2 days after the initial debridement. Between initial debridement and the second washout, the CRP fell from 232 mg/L to 141 mg/L. The vast majority of tissue appeared viable. Areas of non-vital tissue, predominantly affecting the left posterior belly of digastric, were debrided and sent for histopathological examination and microculture. Exploration along the right pterygomandibular region revealed a small locule of pus. Exploration along the sternomastoid gutter and carotid sheath did not show any evidence of infection. Irrigation was performed in a similar fashion as previously stated. Multi-tubular drains were inserted on each side and secured. A nasogastric tube was inserted for enteral feeding. The patient was transferred back to HDU.

A tissue swab taken from the submandibular region revealed Methicillin-resistant Staphylococcus aureus. The patient continued with Cefepime, Metronidazole, and Vancomycin as advised by the Infectious Diseases team. A repeat CT scan took place 4 days after the second debridement. Small pockets of fluid and gas were demonstrated within the neck, but were consistent with surgical sites in the neck and no intervention was indicated. The antibiotic regime was changed 1 week after the second washout to oral clindamycin 450mg TDS as the sole agent for the next 2 weeks. During this time the patient experienced a general downward trend of inflammatory markers. He continued to improve until discharge, and his diabetic management was optimised during his stay. Overall he had three operations and spent 21 days as an inpatient, with 9 of those under intensive care.

Discussion

This case illustrates a case of CNF following dental extraction in New Zealand. Previous public health concerns were raised regarding necrotising fasciitis when local research demonstrated an increasing incidence between 1990 and 2006 in New Zealand (Das et al. 2011). However, a recent retrospective case review of a single-centre in New Zealand over 11 years demonstrated a significant decrease in population incidence since 2004 (Kulasegaran et al. 2016). Of the 138 patients included, only 7 had necrotising fasciitis occurring in the head and neck. No differentiation by location of necrotising fasciitis is given when reporting overall hospital mortality as 21.7%. The incidence of CNF remains significantly higher in Maori and Pacific populations compared with other ethnic groups. This may be due to the higher risk of these populations developing cellulitis, diabetes mellitus, or other related co-morbidities (Tiu et al. 2005; Nisbet et al. 2011).

Prompt diagnosis and treatment are pivotal to reducing CNF morbidity and mortality. A continuous problem is the paucity of clinical signs during the early stages of the disease. Imaging plays a crucial role, and should be utilised in diagnosis, surgical planning, and assessing response to treatment or progression of disease (Gunaratne et al. 2018). CT scans are more useful in CNF diagnosis than basic radiography, ultrasonography, and MRI. CT scans can help identify subcutaneous gas (estimated to be present in ~56% of CNF patients), to monitor progression, and guide surgical intervention (Gunaratne et al. 2018). There is evidence supporting a role for magnetic resonance imaging in early differentiation of non-necrotising cellulitis from necrotising fasciitis (Edlich et al., 2010).

Early surgical debridement is the most important factor in determining outcome (Bucca et al. 2013). Surgical debridement is aggressive and combined with medico-intensive care including antibiotic therapy, physiological support, and a low threshold for retreatment (Bayetto et al. 2017). Tissue should be debrided back to healthy bleeding edges, as vascular thrombosis in the region may compromise effective delivery of antibiotics to the site of infection (Mohammedi et al., 1999). Initial broad-spectrum empiric antibiotic therapy is essential and should be targeted following subsequent microbial culture and sensitivity results. There is no standardized regime for empirical antimicrobial therapy, but combination therapy covering gram-positive, gram-negative, and anaerobic bacteria is commonly prescribed. Nitroimadazoles, cephalosporins, penicillins, and aminoglycosides are widely utilised (Gunaratne et al., 2018). Airway management is an important consideration, and an early tracheostomy is warranted to avoid future difficult intubations and to facilitate repeat debridement (Mohammedi et al., 1999; Gunaratne et al., 2018).

Hyperbaric oxygen therapy (HBO) has been recommended in the literature as an adjunct in the management of CNF. HBO increases the normal oxygen saturation in the infected wounds by a thousand fold, leading to improved polymorphonuclear cell function, angiogenesis and collagen formation, and is directly bactericidal to anaerobic bacteria (Krenk et al., 2007; Gunaratne et al., 2018). HBO has been demonstrated to reduce mortality, duration of hospitalization, and the number of surgical debridements required (Lin et al., 2001; Oguz and Yilmaz, 2012). However, its use is significantly limited by its availability and no consensus protocol for treatment implementation exists (Bayetto et al., 2017; Gunaratne et al., 2018). HBO was considered in this case, however it was not deemed necessary given the patients progressive clinical improvement following the second washout. Intravenous immunoglobulin is another adjunctive treatment that may be of benefit as it has demonstrated reduced mortality in patients with severe group A streptococcal infections, however its use in CNF is scarcely reported.

While the incidence of CNF is low, the most common cause is odontogenic infection and is worth highlighting to dental clinicians. In contemporary practice, clinicians should be most wary of treating patients who present with a combination of comorbidities associated with CNF. Serious consideration should be given to whether these patients require appropriate antibiotic cover for invasive procedures. If a clinician suspects a patient is developing or at risk of developing CNF, appropriate and prompt referral to a tertiary centre is recommended.

References

Bayetto K, Cheng A, Sambrook P (2017). Necrotising fasciitis as a complication of odontogenic infection: a review of management and case series. *Australian Dental Journal* 62: 317-322.

Bucca K, Spencer R, Orford N, Cattigan C, Athan E, McDonald A (2013). Early diagnosis and treatment of necrotizing fasciitis can improve survival: an observational intensive care unit cohort study. *ANZ Journal of Surgery* 83(5):365-370.

Das D, Baker M, Venugopal K (2011). Increasing incidence of necrotising fasciitis in New Zealand: a nationwide study over 1990-2006. *Journal of Infection* 63: 429-433.

Das DK, Baker MG, Venugopal K (2012). Risk factors, microbiological findings and outcomes of necrotising fasciitis in New Zealand: a retrospective chart review. *BMC Infectious Diseases* 12: 348-356.

Edlich RF, Cross CL, Dahlstrom JJ, Long WB (2010). Modern concepts of the diagnosis and management of necrotizing fasciitis. *Journal of Emergency Medicine* 39:261-265.

Fihman V, Raskine L, Petitpas F, Mateo J, Kania R, Gravisse J et al (2008). Cervical necrotizing fasciitis: 8-years' experience of microbiology. *European Journal of Clinical Microbiology & Infectious Diseases* 27:691-695.

Forbes N, Rankin AP (2001). Necrotizing fasciitis and non-steroidal anti-inflammatory drugs: a case series and review of the literature. *New Zealand Medical Journal* 114(1124):3-6.

Gunaratne D, Tseros E, Hasan Z, Kudpaje A, Suruliraj A, Smith M et al (2018). Cervical necrotizing fasciitis: Systematic review and analysis of 1235 reported cases from the literature. *Head and Neck* 00: 1-9.

Krenk L, Nielsen HU, Christensen ME (2007). Necrotizing fasciitis in the head and neck region: an analysis of standard treatment effectiveness. *European Archives of Oto-Rhino-Laryngology* 264: 917-922.

Kulasegaran S, Cribb B, Vandal AC, McBride S, Holland D, MacCormick AD (2016). Necrotising fasciitis: 11-year retrospective case review in South Auckland. *ANZ Journal of Surgery* 86: 826-830.

Lin C, Yeh FL, Lin JT, Ma H, Hwang CH, Shen BH et al., (2001). Necrotizing fasciitis of the head and neck: an analysis of 47 cases. *Plastic and Reconstructive Surgery* 107: 1684-1693.

Mitchell A, Williams A, Dzendwoskyi P (2011). Necrotising fasciitis: an 8.5-year retrospective case review in a New Zealand Intensive care unit. *Critical Care and Resuscitation* 13: 232-237.

Mohammedi I, Ceruse P, Duperret S, Vedrinne J, Bouletreau P (1999). Cervical necrotizing fasciitis: 10 years' experience at a single institution. *Intensive Care Medicine* 25: 829-834.

Murray M, Dean J, Finn R (2012). Cervicofacial necrotizing fasciitis and steroids: Case report and literature review. *Journal of Oral and Maxillofacial Surgery* 70(2): 340-344.

Nisbet M, Ansell G, Lang S, Taylor S, Dezendrowskyj P, Holland D (2011). Necrotising fasciitis: review of 82 cases in South Auckland. *Internal Medicine Journal.* 41: 543-548.

Oguz H, Wilmaz MS (2012). Diagnosis and management of necrotizing fasciitis of the head and neck. *Current Infectious Disease Reports* 25:829-834.

Sarna T, Sengupta T, Miloro M, Kolokythas A (2012). Cervical Necrotising Fasciitis with Descending Mediastinitis: Literature Review and Case Report. *Journal of Oral and Maxillofacial Surgery* 70: 1342-1350.

Tiu A, Martin R, Vanniasingham P, MacCormick AD, Hill AG (2005). Necrotising fasciitis: analysis of 48 cases in South Auckland, New Zealand. *ANZ Journal of Surgery* 75: 32-34.

Author details

Calum Fisher BDS Hons (Otago) Waikato Hospital Oral and Maxillofacial Department Pembroke St, Hamilton 3200, NZ (corresponding author: ccmmfisher@gmail.com)

Thasvir Singh BDS (Otago) MBChB (Auckland) MPhil (Melbourne) FRACDS OMS
Waikato Hospital Oral and Maxillofacial Department