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Life threatening septic shock following a dental hygiene appointment

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Abstract

Case description: A 34-year-old man was referred to a hospital dental department with a large facial swelling and major septic shock, following a routine dental hygiene appointment. This required intensive care treatment, and the patient was eventually discharged home after six days of multi-antimicrobial therapy.

Practical implications: While rare, life threatening infections can develop following routine hygiene procedures.

Introduction

Infections of dental origin have the potential to become rapidly progressive and life threatening. This case report describes the progression of an unusual facial infection in a young man with no underlying immunodeficiency, following a routine dental hygiene appointment, in which *Streptococcus anginosus* was found to be the causative organism.

Case report

A 34-year-old Chinese man visited his dentist for a routine scale and polish. His periodontal health was described as unremarkable apart from minor calculus deposits, most of which were removed at a hygiene appointment 7 months previously. The scaling was with an ultrasonic scaler and the patient did not recall any bleeding and did not consider that it was aggressive.

Over the next few days, he developed intermittent fevers and felt generally unwell. One week after his hygiene appointment, and with increasing malaise, he awoke with a large right-sided facial swelling and attended an appointment with his general practitioner. The practitioner noted tachycardia and a temperature of 37.1 degrees and referred him via telephone to the hospital dental department.

On presentation at hospital, he appeared faint, pale and weak and struggled to walk unaided to the bathroom. His medical history was unremarkable apart from recent smoking cessation and treatment for syphilis in 2010, which had been serologically cleared.

He had a large and firm right side buccal space swelling, about 4 cm thick over the anterior parotid region, and extending forward to the right lip commissure. There was no lingual or submandibular swelling and he had not experienced any swallowing or breathing difficulty. He had trismus with 2 cm opening but despite this, bitewing and periapical films were taken. He reported no tooth or gum pain but his right cheek was very tender to palpate. He had experienced no recent or historical tooth symptoms and his mouth appeared pristine with no caries or restorations and no abnormalities or trauma of the soft tissues on the upper and lower arches. His oral hygiene was good. There was no gingival inflammation, pocketing, or tooth mobility and no trauma history. His mandibular right third molar was horizontally impacted but with no indication of suppuration, tenderness or inflammation.

A tiny amount of a yellow/creamy substance was seen at the parotid duct and thought most likely to be food debris. No exudate other than clear saliva was expressed from the duct. Unfortunately, the hospital panoramic dental radiograph (PDR) machine was malfunctioning and remained non-operational for the next three days.

An ear, nose and throat (ENT) house officer was summoned to assess possible parotitis and to admit the patient with urgency, with a diagnosis of major septic shock. On arrival at the ward, he was noted to be severely hypotensive and peripherally shut down, with a heart rate of 85 beats per minute and respiratory rate of 18. Despite aggressive (6 L) fluid resuscitation with normal saline, Dexamethasone (8 mg), and intravenous Amoxycillin and Clavulanic Acid (1.2 g), his blood pressure became unrecordable and he was rapidly transferred to the intensive care unit (ICU) for further fluid resuscitation. Noradrenaline was also started to improve his blood pressure. On admission to ICU he was noted to have bilateral pleural effusions (fluid in the pleural space) and consolidation with atelectasis (Figures 1, 2).

Given the speed, severity and unclear focus of infection, microbiology specialists were consulted and intravenous antibiotic therapy commenced with Ceftriaxone (2g), Clindamycin (450 mg), Vancomycin (1.5 g) and Flucloxacillin (1.5 g). His white cell count on the first day was 26.2 (normal is 4.0-11.0) and C-reactive protein 226 (normal is 0-5). Most of his clinical chemistry, haematology and coagulation results were well outside the normal range (Table 1).

When the patient was examined by an ENT consultant later that day, the swelling had spread submandibularly, down the neck, and laterally towards the right shoulder. CT scanning showed a facial and neck cellulitis with mediastinitis. There was obvious subcutaneous oedema but no drainable collection of pus. Parotid infection was excluded by the ENT and Radiology teams after examining the CT and ultrasound results. Radiologists ruled out infection of submasseteric space origin.

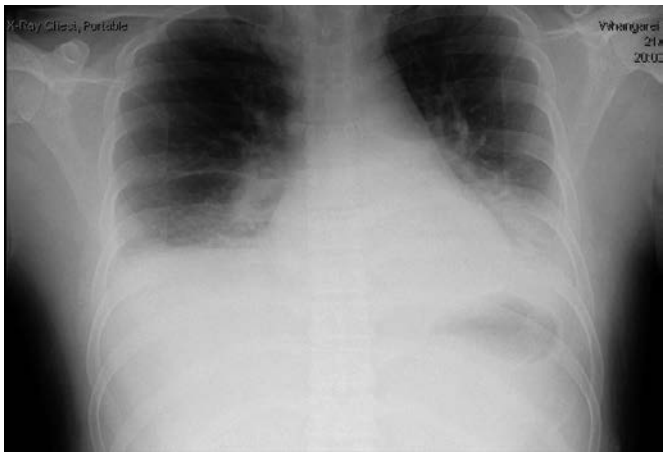

Figure 1. Chest radiograph showing pleural effusion.

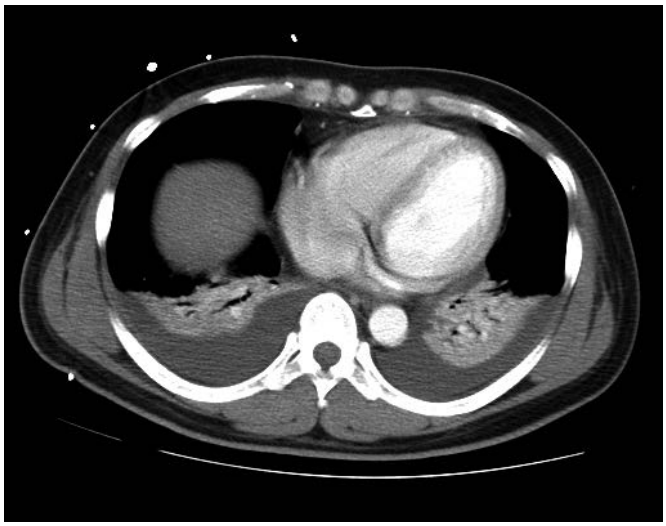

Figure 2. CT scan demonstrating pleural effusion and lung consolidation.

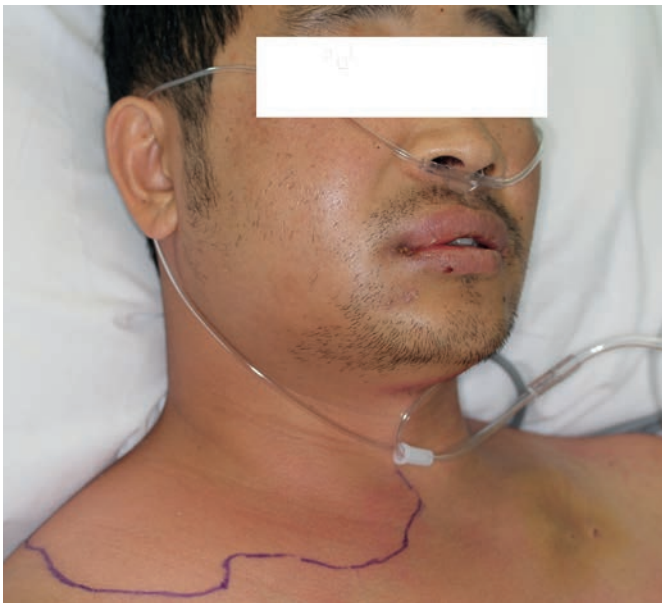

Figure 3. Improvement by day 3: pen line indicates extent of original swelling.

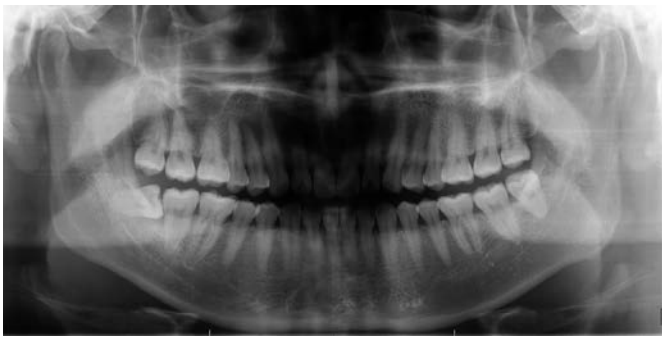

Figure 4. Panoramic dental radiograph.

There was no indication of infection associated with the horizontally impacted 48 and no other abnormalities such as occult pathology were found on CT exam. Orally, on presentation, the swelling had been confined to the right side buccal space. Ultrasonography confirmed no visible collection to be drained and his chest film showed a widened mediastinum.

His blood microbiology was unremarkable and HIV and hepatitis screens were both negative. Extensive investigations ruled out any underlying immunodeficiency and diabetes. His echocardiogram indicated no endocarditis. His temperature spiked several times during the first 24 hours. Over the next three days he remained in the ICU and showed a slow but steady improvement whilst continuing with antimicrobial therapy in reducing doses. By the third day he was able to eat and drink without much discomfort, the facial and neck swelling had markedly improved, and he was able to mobilise without dizziness (Figure 3). It was not until the third day that a positive blood culture of haemolytic *Streptococcus anginosus* was obtained. No anaerobic organisms were isolated. There were no abnormal findings from his PDR when this was eventually obtained (Figure 4).

He was transferred to a ward where he continued to make good progress and was discharged home six days after admission with a two week supply of oral Amoxicillin and Clavulanic Acid, and Clindamycin (white cell count 4.9 and C-reactive protein 56). Review two weeks later showed complete resolution of oral, extra-oral and mediastinal symptoms.

Discussion

This infection was puzzling in that there was no conclusive infection source, although the timing of onset of symptoms suggests that bacteraemia occurred during his hygiene appointment. The impacted mandibular third molar is unlikely to be the infective source as there had never been any symptoms associated with this tooth, the soft tissues were clinically normal and radiographs and CT scanning identified no foci of infection. A diagnosis of parotitis had been excluded. This complicated his treatment options, as no surgical management of the infection source was possible.

There is only one report of a similar life threatening infection occurring following a hygiene appointment, in the apparently pristine mouth of a fit healthy adult.

Table 1. Laboratory results showing deranged blood chemistry.

Full Blood Count	Result	High or Low	Ref range
White cell count	20.3 10^9/L	H	4.0-11.0
HAEMOGLOBIN	**127 g/L**	**L**	**130-175**
Red Cell Count	**4.29 10^12/L**	**L**	**4.30-6.00**
Haematocrit	**0.35**	**L**	**0.40-0.52**
Mean Cell Volume	82 fl		80-99
Mean Cell Haemoglobin	30 pg		27-34
M.C.H.C.	**361 g/L**	**H**	**320-360**
Red cell Dist. Width	12.30%		11.5-15.0
PLATELET COUNT	**132 10^9/L**	**L**	**150-400**
Neutrophils	**16.7 10^9/L**	**H**	**1.9-7.5**
Lymphocyte	**0.6 10^9/L**	**L**	**1.0-4.0**
Monocyte	1.0 10^9/L		0.2-1.0
Eosinophil	0.0 10^9/L		0.0-0.5
Basophil	0.0 10^9/L		0.0-0.2
IMMATURE GRANULOCYTES	**2.0 10^9/L**	**H**	**0.0-0.1**
PROTHROMBIN RATIO	**1.5**	**H**	**0.8-1.2**
A.P.T.T.	**47 sec**	**H**	**25-35**
Fibrinogen Assay	**5.0 g/L**	**H**	**1.8-4.0**

In that case, a patient developed multiple brain abscesses following professional tooth cleaning, with *Streptococcus intermedius* and *Staphylococcus warneri* implicated (Pallesen et al., 2014).

The potential for bacteraemia following manipulation of the periodontal tissues is well studied and documented (Takai et al., 2005; Crasta et al., 2009; Tomas et al., 2012; Matthews, 2012; Zhang et al., 2013).

Streptococcus anginosus is a species of bacteria that together with *Streptococcus constellatus* and *Streptococcus intermedius* makes up the *Streptococcus anginosus* group (SAG), formerly known as the milleri group. *Streptococcus anginosus* may be haemolytic or non-haemolytic, with the haemolytic form being found only orally (Ruof, 1988). Members of the SAG are considered to be part of a normal commensal flora, and are commonly isolated from the oral cavity, oropharynx, gastrointestinal tract and vagina.

In general, members of SAG have a propensity to form abscesses and cause invasive pyogenic infection, when able to travel to remote sites or into tissue planes they do not normally inhabit, especially in patients with underlying poor health or immunodeficiency (Singh, 1988). In such cases, they behave aggressively and have been isolated in brain and liver abscesses, head and neck and intrathoracic infections (Mylonas et al., 2007; Neumayr, 2010). Haemolytic *Streptococcus anginosus* has been infrequently localised in liver, spleen and brain abscesses in patients with poor oral health (McKenzie, 2010; Yilmaz et al., 2012). Infective endocarditis has been reported in a significant number of patients with SAG bacteraemia. The *Streptococcus anginosus* group is also known to release extracellular products that have immunosuppressive effect, further permitting them to proliferate within the confines of an abscess. They are also believed to have a polysaccharide capsule that helps them evade the body's natural defence systems and escape phagocytosis prior to infection (Sinner, 2010).

Head and neck infection by SAG is generally associated with a dental origin. Mediastinal spread is a rare complication of a dental abscess with a mortality rate of up to 40% even with aggressive antimicrobial therapy and intensive management (Jarboui et al., 2009). Anginosus group bacteria are often involved in oral and maxillofacial infections alone or in association with other aerobic and/or anaerobic bacteria, with *Streptococcus anginosus* by far the most frequently isolated species within the group (Bancescu et al., 1999). Odontogenic infection cannot be ruled out in patients with SAG bacteraemia of unknown source, especially in patients with poor oral hygiene and active dental disease.

Although immunity levels in healthy people can vary due to stress, hormones and other precipitating factors, such infections are rare in immunocompetent adults

The recommended treatment is rapid initiation of intravenous antibiotic therapy, surgical drainage and removal of the infective source. A combination of several antibiotics with a wide spectrum of activity is advised initially as the causative organism or organisms are often not identified for some time. These infections are usually satisfactorily treated with penicillin G and cephalosporins (Giuliano, 2012).

An antiseptic regimen prior to the patient's scaling may have been of some benefit in preventing this outcome, however such sequelae are extremely rare, especially in a fit and healthy patient. Routine antiseptic prophylaxis may be advisable in immuno-compromised individuals. There is considerable debate around the effectiveness of chlorhexidine pre-operative mouth washing in preventing bacteraemia.

Conclusion

Streptococcus anginosus infections are uncommon in healthy individuals, and very rare in adults with well-maintained mouths and no underlying immunodeficiency. It is assumed that a minor breach of the oral integrity and subsequent bacteraemia allowed this overwhelming infection to take hold in this patient, following a dental hygiene visit. Rapid multidisciplinary management was needed to manage this patient's life threatening infection and, in such cases, treatment should be initiated quickly and aggressively to facilitate a successful outcome.

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