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How serious are oral infections?

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Abstract
Life-threatening conditions following dental infections have been rare since antibiotics were introduced into the world of medicine. However, infections spreading through the soft tissues of the head and neck are encountered occasionally and mortality is still reported as a result of sepsis or airway embarrassment. A case of Ludwig's angina from odontogenic infection that progressed into mediastinitis and pericarditis is presented. The steps adopted in the management of this case highlight the significance of early recognition and diagnosis of the source of deep cervical infections, the importance of securing the airway, effecting surgical drainage and aggressive intravenous antibiotic therapy.

Key words: Ludwig's Angina; Mediastinitis; Pericorditis

Introduction

Early and aggressive treatment of odontogenic abscesses is essential. Despite the rarity of fatal complications, cases have been reported of death as a consequence of late recognition of such infections.[1] Severe odontogenic infections can be challenging to manage especially in the presence of underlying medical co-morbidity or when the causative organism is difficult to treat. As the incidence of such complications is low, the need for early aggressive treatment is often underestimated.[1,2,3] This article describes a case of mediastinitis associated with pleural empyema and pericarditis as a complication of odontogenic infection, looks at similar cases reported in the literature over the past 17 years and finally presents an overview of aetiology and management.

Case report

An 18-year old Caucasian male was referred to the oral and maxillofacial surgery department. He had had a one-week history of dental pain associated with a left submandibular swelling, three days earlier he was seen by his general medical practitioner who prescribed amoxycillin 500 mg eight hourly for five days. However, his condition did not improve and the swelling increased extending inferiorly and into the contralateral neck. He also complained of dysphagia, which had limited his diet to fluids only for two days. There was no specific past medical history but he admitted to an intravenous drug habit of about one year .

At presentation, he was toxic; febrile (37.2 Cº - axilla), tachycardic (pulse 125 beat/minute), mildly tachypnoeic (respiratory rate 20/minute) but normotensive. Maxillofacial examination, revealed a firm, tender bilateral submandibular swelling extending down into the root of the neck obliterating the sternal notch. His mouth opening was limited (maximum opening 1.5 cm), he had marked dysphagia but no upper airway compromise, oxygen saturation was 98 per cent on air measured by pulse oximetery. Intraorally, there was slight elevation of the floor of the mouth, his tongue was swollen and firm, he had poor oral hygiene with multiple carious teeth, the mandibular right and left second molars were grossly decayed and very tender to percussion. An orthopantogram revealed a periapical radiolucency related to the mandibular second molars (Figure 1).

A provisional diagnosis of Ludwig's angina was made. The patient was therefore admitted to hospital to monitor the airway, remove the causative teeth, effect surgical drainage and to administer intravenous antibiotics. The patient was taken to theatre for incision and drainage under general anaesthesia. An awake fibre-optic nasal intubation was necessary to secure the airway prior to induction of anaesthesia. Drainage of both submandibular, submental and sublingual spaces was effected via an extraoral approach. Pus subsequently drained from both submandibular spaces, following which corrugated drains were inserted and the involved teeth were extracted. The patient remained intubated and was admitted to the intensive care unit. Intravenous antibiotics were commenced immediately consisting of, co-amoxiclav 1.2 grams eight hourly and metronidazole 500 mg eight hourly, he was also given intravenous dexamethasone eight hourly. He was extubated the next day following demonstration of an adequate leak around the endotracheal tube, and a general improvement in his clinical condition.

Pre-operative haematological and biochemical investigations revealed that the patient was anaemic (haemoglobin 10.3 gram/L), white cell count was high (20.7 x 10^9/L), a low serum albumin of 28 gram/L (normal range: 35 - 50 gram/L) but his fasting blood glucose was

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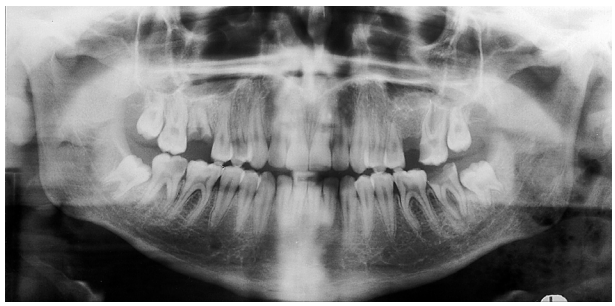

FIG. 1
Orthopantogram revealing periapical radiolucencies related to the mandibular second molars, the primary focus of infection.

within the normal range and blood cultures were negative. Unfortunately the initial pus specimen was not processed hence no culture and sensitivity results were available. Two days after admission the patient complained of right-sided chest pain and was noted to have reduced air entry at the right lung base, and a pericardial rub. Plain chest radiology demonstrated collapsed middle and lower lobes of the right lung associated with a moderate sized pleural effusion (Figure 2). An ECG revealed the typical saddle-shaped S-T segment elevation (Figure 3) indicative of pericarditis. Conventional ultrasound confirmed a large right-sided pleural effusion, echocardiography revealed a small pericardial effusion but no other cardiac abnormality.

Initially he was managed with a right chest drain releasing straw-coloured fluid on insertion that was sterile on subsequent culture. However, his condition deteriorated two days later, with a spiking pyrexia (peak 38.8C°) and worsening chest signs. Pus was now draining from the chest drain, and a right-sided pleural empyema was diagnosed. Empyemic involvement of the left hemithorax followed two days later and was managed with a second chest drain.

Computed tomography (CT) scan of the chest revealed a large pleural fluid collection on the right side with an enhancing rim consistent with empyema, a similar but a less marked appearance was noted on the left. There was also evidence of a mediastinal abscess and some compressive atelectasis of the right lung (Figure 4). Oesophagography was done to rule out the presence of a fistulous tract associated with the oesophagus, no oesophageal leakage nor constriction was discovered. Screening for hepatitis B and C was negative. Microbiological examination of the pleural drainage revealed a mixed infection with heavy growth of *Streptococcus milleri* which was sensitive to penicillin plus anaerobes mainly Gram negative bacilli; *Bacteroides* species and *Prevotella melaninogenica,* which were sensitive to metronidazole. No acid- fast bacilli were seen or cultured. The ESR was 103 mm/hour and C-reactive protein 272.7 mg/L (normal range: 0–0.05). Streptokinase therapy was used in both spaces to enhance drainage.

Unfortunately the right side signs persisted, and consultation was sought from the cardiothoracic surgeons who decided to perform open drainage. Therefore cardiothoracic decortication was carried out three weeks following initial admission, a further 500 ml of pus was drained. Following this intervention the patient's condition improved and two days later he was transferred to the ward. Repeat chest X-rays and ultrasound showed resolution of the pericardial effusion that did not require intervention. The patient was discharged one week later,

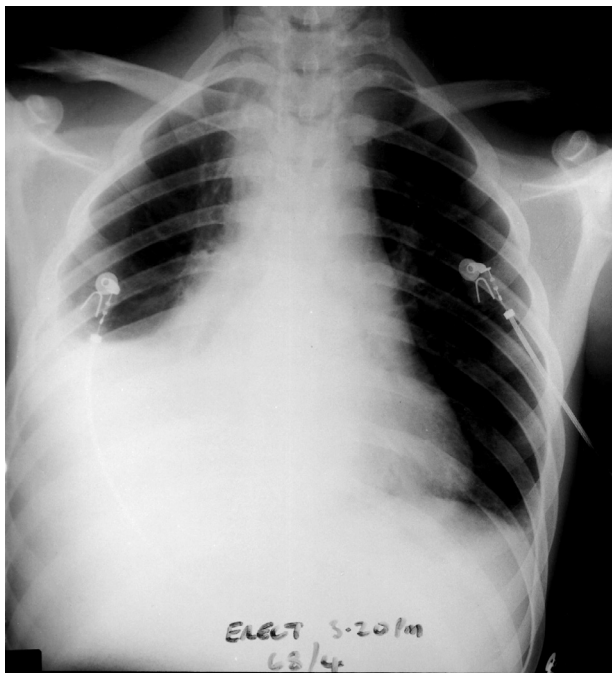

FIG. 2
A mobile AP chest showing pleural fluid collection on the right and gas in the subcutaneous tissues of the neck.

having spent one month in hospital and subsequent recovery was uneventful.

Reported cases in the literature

A review of reported cases of mediastinitis complicating orofacial infections, from 1985, was performed using Medline and Embase. The following keywords were used: mediastinitis, orofacial infections, odontogenic infection and Ludwig's angina. A total of 11 reports that included 25 patients were found, two of the 25 patients were females (eight per cent), mean age of the patients was 35 ± 13 years. Eleven out of the 24 patients (24 patients out of the 25 had a known treatment outcome) died (46 per cent) despite securing airway, achieving drainage and commencing antibiotics (Table 1).

Discussion

This case initially presented as a classical example of Ludwig's angina first described by Wilhelm Fredrick von Ludwig in 1836.[4] Ludwig's angina is a clinical diagnosis given to a massive, firm, brawny rapidly spreading cellulitis affecting simultaneously the submandibular, the submental and the sublingual spaces bilaterally. Ludwig's angina most commonly occurs as a complication of dental or periodontal infection. Infection of the second and third mandibular molars account for 70 per cent to 85 per cent of the cases.[5,6] Other less common causes include submandibular sialadenitis, tonsillitis, infected neoplasms, compound fractures of the mandible, penetrating wounds in the floor of the mouth and one case without a known aetiological factor has also been reported.[2, 7 -11]

Complications of Ludwig's angina are rare but serious because of the progressive and direct extension of the infection to the neck and mediastinum, this is associated with a high mortality rate ranging from 40 per cent to 65 per cent.[5,12 -14] Deep neck infections mostly affect young males (reported range 78 per cent-94.5 per cent).[14,15]

Since the introduction of antibiotics there has been a significant decrease in the incidence of Ludwig's angina

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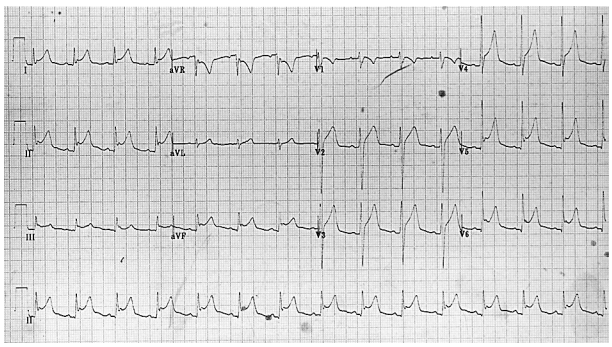

FIG.3
ECG showing concave-upwards (saddle-shaped) ST segments in all the leads except a VR.

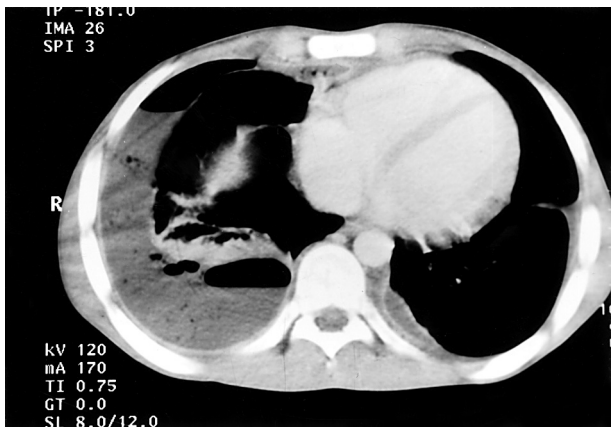

FIG. 4
CT chest showing large pleural fluid collection on the right and fluid collection on the left, both have rim enhancement consistent with empyema. There is also localized fluid collection in the anterior mediastinum representing a mediastinal abscess.

and its associated complications. However, there has been an increase in the role of dental infection in the aetiology of deep neck infections compared with the pre-antibiotic era, when peritonsillar infection was the most common aetiological factor.[16]

Predisposing systemic conditions have been mentioned in the literature mainly: diabetes mellitus and alcoholism. Our patient who admitted to intravenous drug use, was anaemic and had a marginally low albumin, factors which could point to malnourishment that might have aggravated the course of his disease. A test for human immunodeficiency virus (HIV) was not carried out, interestingly Miller *et al.* in a pilot study in 1998[17] concluded that HIV-positive patients were not at a higher risk of developing serious odontogenic infections.

Microbiological findings were consistent with the literature, complex and, in general, representative of the indigenous microflora of the oral cavity.[18] The viridans streptococci, of which the *Streptococcus milleri* is a group, are common inhabitants of the oral cavity. The anaerobic Gram negative bacilli, of which *Prevotella melaninogenica* is a member, previously referred to as *Bacteroides melaninogenicus,* comprise a substantial proportion of plaque microflora. Their isolation in this case supports the oral origin of the infection. *Staphylococcus aureus* is less frequently incriminated.[3,13,16] However, in some instances no organisms were isolated and this could be attributed to a failure to isolate and culture anaerobic bacteria or a prior empirical antibiotic treatment that made the pus sterile.[13]

TABLE I
SUMMARY OF PATIENTS WITH MEDIASINITIS FROM ORAL INFECTION

Reference	Year	Patient No.	Age (years)	Gender	Outcome
Bounds *et al.*[2]	1985	1	48	Male	Died
Esgaib *et al.*[28]	1986	2	52	Male	Died
Levine *et al.*[29]	1986	4	25	Male	Died
		5	51	Male	Discharged
		6	19	Male	Discharged
		7	24	Male	Discharged
		8	44	Male	Died
Rubin and Cozzi[30]	1987	9	27	Male	Died
Zachariades *et al.*[3]	1988	10	?	Male	Discharged
Garate-Creglo and	1991	11	18	Female	Discharged
Gray-Escoda[31]		12	32	Male	Discharged
		13	64	Male	Discharged
Colmenero *et al.*[32]	1993	14	32	Male	Discharged
		15	54	Male	Died
		16	24	Male	Discharged
		17	48	Male	?
		18	17	Male	Died
		19	39	Male	Discharged
		20	28	Male	Died
Bonapart *et al.*[1]	1995	21	41	Male	Died
Zeitoun and	1995	22	37	Male	Discharged
Dhanarajani[13]		23	38	Male	Died
Dugan *et al.*[5]	1998	24	25	Male	Discharged
Tsunoda *et al.* [20]	2000	25	27	Female	Discharged

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- **Since the use of antibiotics, life-threatening conditions following dental infections are rare**
- **A case of Ludwig's angina of odontogenic origin is presented that progressed into mediastinitis and pericarditis**

Whilst all such patients should have routine haematology, biochemistry and other investigations dictated by medical co-morbidity, if mediastinitis is suspected CT is a more reliable diagnostic tool when compared to chest radiography, since the findings of the latter are often non-specific.[19] It is strongly advised to perform CT scans of the chest in patients with extensive cervical infections to exclude suppuration in the chest. [2] In case of mediastinal involvement, the scan may aid the thoracic surgeon in choosing the optimal approach for drainage as can be seen in this case.[20]

The successful management of this serious scenario stemmed from our commitment to abide by the all three lines of treatment:

Securing airway

The airway must be closely monitored, establishing a definitive airway is imperative when the airway is compromised.[21] Options for managing the airway include awake fibre-optic intubation, awake blind nasal intubation, gaseous or intravenous induction followed by laryngoscopy and intubation and awake tracheostomy under local anaesthesia. [22] Tracheostomy as a gold standard in the management of Ludwig's angina has been challenged recently, the risk of spreading infection to the mediastinum, difficulty of surgical access and the inability of the patient to lie supine have made awake fibre-optic intubation the preferred method by anaesthetists for managing Ludwig's angina, hence adopting it in securing airway in this case.[22,23] However, surgical airway management is strongly advocated when the retropharyngeal space is involved since not only is intubation technically difficult but rupture of the lateral pharyngeal abscess may result in contamination of the tracheobronchial tree. [2,5]

Medical therapy

Improved results in managing Ludwig's angina have been attributed to the availability and rational use of antibiotics and not to surgery.[2] Empirical treatment, against the causative agent in this case odontogenic pathogens, should be commenced immediately and in high doses via an intravenous route. Penicillin G, 2.4 grams daily in four divided doses, combined with either metronidazole, 500 mg every eight hours, or clindamycin, 600 mg every eight hours, in the normal host is highly effective while in known immunocompromised patients other antibiotics such as piperacillin or imipenem may be required.[24]

The debate on the use of steroids in the management of head and neck airway threatening infections has been ongoing. The controversy stems from the anti-inflammatory and immune-suppressant actions of steroids. However, recent literature has recommended intravenous dexamethasone, 10 mg as an initial dose followed by 4 mg every six hours for 48 hours.[25,26] The anti-inflammatory effect of dexamethasone resolves oedema and cellulitis, providing chemical decompression, protecting the airway and allowing better antibiotic penetration into the area. It has eliminated the need for surgical airway in many instances allowing intubation, if required, to be carried out under more controlled conditions. In addition, the potential risk of spreading infection to adjacent areas, such as the mediastinum, is far greater from establishing a surgical airway than from short-term treatment with steroids.[26,27] Lastly, a regimen including aggressive antibiotic treatment and short-term steroids and has been shown to reduce the length of hospitalization.[27]

Surgical intervention

Performing incision and drainage has been recommended in the management of Ludwig's angina, even though most cases initially do not produce pus, it is believed that this provides some decompression and can relieve airway obstruction.[2] The focus of infection should be addressed as well. In cases of descending mediastinitis three approaches for mediastinal drainage have been recommended:[20] transcervical, standard thoracotomy, and the clamshell incision. Bonapart *et al.*[1] reported that posterolateral thoracotomy provides a good approach to all compartments of the mediastinum, the pleural cavity and the pericardium, allowing adequate drainage, it improves survival rate from 40 per cent up to 83 per cent.

Conclusion

The patient in this case, presented with findings consistent with Ludwig's angina. Management included airway support, intubation, intravenous antibiotics, steroids and surgical decompression of the cervical swelling. However, his condition deteriorated quickly and a possible systemic immunocompromise may have been a factor in view of his intravenous drug abuse and the likely malnourished state. The microbiogical findings were consistent with what has been reported in literature, i.e. oral flora. Surgical decompression of the chest was required to bring his illness to an end.

It should be emphasized that orofacial infections can progress, and as in this case lead to 'Ludwig's angina' which, if not treated promptly, may cause airway embarrassment and in rare cases mediastinitis, especially in the presence of underlying immunocompromise. Thus such infections should be treated promptly with appropriate surgical drainage, definitive management of the focus of infection and the use of suitable antibiotics.

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