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CASE STUDY

LUDWIG'S ANGINA – A BRAWNY CELLULITIS: A CASE REPORT

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ABSTRACT

Ludwig's angina is an aggressive cellulitis affecting the submandibular and sublingual spaces bilaterally. Formerly, doomed to be fatal, the disease at present can be effectively managed by prompt airway secure, adequate antimicrobial coverage and surgical drainage. We report a case of Ludwig's Angina, with a non odontogenic source, extending to the neck with elevation of the floor of the mouth resulting in dysphagia and restricted mouth opening. Regression of the disease was obtained by appropriate use of parenteral antibiotics and surgical drainage.

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INTRODUCTION

In 1836, German surgeon Wilhelm Friedrich von Ludwig described five cases of infections involving the floor of the mouth wherein progressive respiratory obstruction was a prominent feature. This condition, subsequently termed as Ludwig's Angina, is recognised as an aggressive cellulitis affecting the submandibular and sublingual spaces bilaterally. The infection doesn't remain contained and rapid spread occurs into the fascial planes of the neck. Involvement of the submandibular spaces produces a brawny induration of the suprahyoid region whereas a painful 'woody' oedema at the floor of the mouth can be seen owing to sublingual space engorgement. The posterior aspect of the edematous tongue, forces the equally edematous epiglottis backward, thus, narrowing the airway. The management of the disease lies in maintenance of the airway and regression is achieved following appropriate antibiotic therapy and surgical drainage (Holland, 1975). Death from asphyxia or septicaemia is the most dreaded complication of Ludwig's angina (Candamourty *et al*., 2012).

CASE REPORT

A 60-year-old male, reported to the Department of Oral and Maxillofacial Surgery with a chief complaint of difficulty in swallowing and restricted mouth opening since four days. There existed bilateral submandibular swelling (Fig. 1), the severity of which had reportedly increased over the past two days. The bilateral submandibular and sublingual swelling was brawny and hard with no evidence of fluctuance while the tongue was elevated against the palate (Fig. 2). The inter-incisal distance was limited to half a finger. The lower left first molar was missing while the lower left first and second premolars appeared to be periodontally compromised (Fig 3). The patient's past medical history seemed non- conducive. On general examination, he appeared frail and febrile. The diagnosis of Ludwig's Angina was made. Physical examination revealed a temperature of 37.6°C, a regular pulse of 92 beats per minute and blood pressure of 152/85 mm of Hg. Haematological investigation showed a total leucocyte count (TLC) of 15,400 per ml with derranged values of neutrophils and lymphocytes. An elevated serum urea level of 90 mg/dl was also obtained. Intravenous access was obtained and an infusion of normal saline was started. Intravenous administration of Penicillin with Clavulanic acid 1.2 g (two times), Metronidazole 100 mg (three times), Tramadol (two times), Paracetamol 150 mg (two times) and Dexamethasone 8mg (two times) was initiated. Ultrasonography (Neck) was suggestive of bilateral submandibular gland inflammation with cervical lymphadenopathy. CT (Brain + C-Spine) was suggestive of swelling at floor of the mouth, erosion and root abscess at left lower back region of jaw and right retromolar and parapharyngheal abscess with air foci (Fig. 3). General anaesthesia was induced using fibreoptic nasotracheal intubation. Submental incision was made, deepened by blunt dissection, right and left submandibular spaces explored and prompt pus collection was achieved. Alongside, a right submandibular incision was made, right para pharapharyngeal space was explored; no pus discharge was noted. A roller

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gauze was placed at the right submandibular region while a plain plastic drain was passed through adjacent stab incisons, secured using 3-0 monofilament nylon and left at the site (Fig. 4 and 5). Teeth with poor prognosis were extracted which included right upper third molar and left lower first and second pre-molars (Fig. 6). On completion of the surgical intervention, the endotracheal tube was left in *situ* and the patient was put on intermittent positive pressure ventilation until the swelling had resolved sufficiently so as not to endanger the airway. Intravenous medications and fluids were continued. Postoperative irrigation through the drain was done every 12 hourly while the roller gauze was removed after 48 hrs.

Patient recovery was satisfactory, with a maintaining pulse rate of 68 beats/min, blood pressure of 130/84 mmHg and oxygen saturation of 97%. The total leucocyte count was 9,600 mm/cu ml 24 hours postoperatively. On the second post-operative day, feeding was initiated through Ryle's tube. The culture report revealed presence of pus cells. On the 6th day postoperatively, the swelling had regressed considerably, the drain was removed, a mouth opening of one finger was achieved and the patient was discharge with periodic recall instructions (Fig. 7). The Ryle's tube was removed on 10th postoperative day.

Fig. 1. Facial photographs depicting bilateral swelling involving the submandibular and submental regions

Fig. 2. Restricted mouth opening with raised floor of the mouth

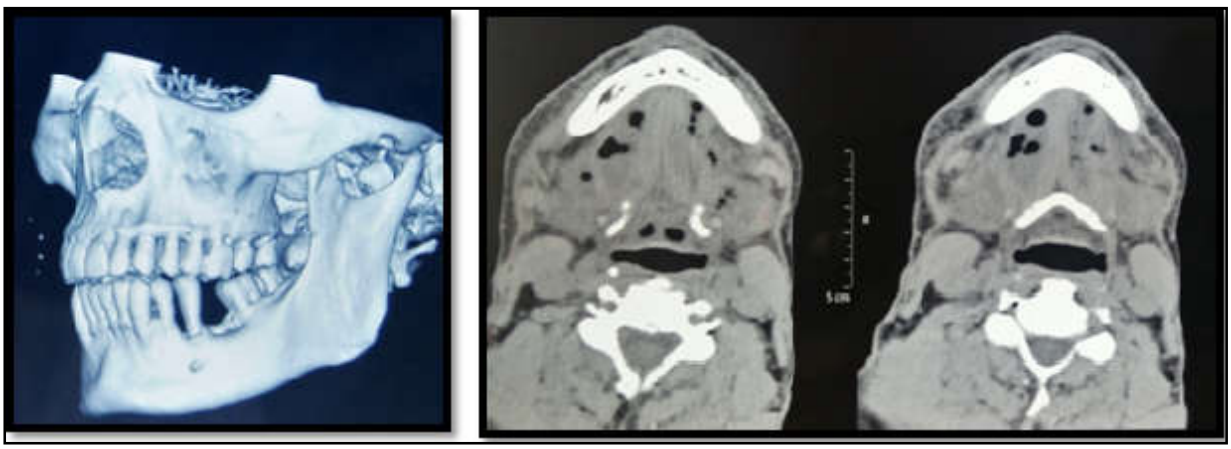

Fig. 3. CT (Brain + C-Spine) Images; A. The lower left first molar is absent showing bony erosion while the first and second premolars appear periodontally compromised. B. Right retromolar and parapharyngheal abscess with air foci seen

Fig. 4. Submental and submandibular incisons made and plastic drain placed

Fig. 5. Roller gauze placed at the submandibular region and irrigation done through the drain

Fig. 6. Maxillary Right Third molar and Mandibular first and Second Premolars Extracted

Fig. 7. Post-Operative photograph showing regression of swelling and increased inter-incisal distance

DISCUSSION

Ludwig's angina, in the pre-antibiotic era, carried a high mortality rate. The causative organisms isolated includes Streptococci viridans, Staphylococcus aureus, and Staphylococcus epidermidis. Although dental infection is responsible for the majority of cases, (Iwu, 1990) it has also been attributed as a complication of sublingual lacerations, sialadenitis (Bounds, 1985), compound mandibular fractures (Dormer and Babett, 1972), and infected malignancy (George *et al*., 1965). A case without any identified local factor has also been reported (Roger *et al*., 1980). Prompt airway securing with surgical drainage and adequate antibiotic coverage remains the appropriate line of treatment. The significance of intensive antibiotic therapy have always been over emphasized in literature (Meyers *et al*., 1972). In the event of perforation of the lingual plate of the mandible, the submandibular tissue spaces will be affected directly (Dubrul, 1980). Tracheostomy remains the choice of airway management while cricothyroidotomy and intubation aided by fibreoptic light can also be used. Delay, until advanced signs of obstruction occur, will almost certainly prove fatal.

Conclusion

Management of Ludwig's angina should be based on patient's clinical condition. It is essential to identify Ludwig's angina in the earlier stages of the disease, when it is easier to manage. In advanced cases, airway management and surgical drainage with organism specific antibiotic therapy are important in avoiding complications.

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