## **Case Report**

# A case of Ludwig's angina in a betel chewer

K V P Munasinghe<sup>1,2</sup>, R M R H Munasinghe<sup>1,2</sup>, P Pallipane<sup>3</sup>, F H D S Silva<sup>3</sup>

<sup>1</sup>Postgraduate Institute of Medicine, University of Colombo, <sup>2</sup>Colombo South Teaching Hospital, Kalubowila, <sup>3</sup>University of Sri Jayewardenepura, Sri Lanka.

Key words: Ludwig's angina, betel, antibiotics, restorative dentistry, airway compromise

Corresponding Author: K V P Munasinghe, E-mail:< paramivm@gmail.com > iD

Received: 13 Apr 2023, accepted: 11 Oct 2023, Published: 02 Nov 2023

Competing Interests: Authors have declared that no competing interests exist

© Authors. This is an open-access article distributed under a Creative Commons Attribution-Share Alike 4.0 International License (CC BY-SA 4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are attributed and materials are shared under the same license.



#### Introduction

Ludwig's angina is a severe form of diffuse cellulitis that affects the submandibular, sublingual and submental regions, bilaterally, manifesting as an emergency due to its fast onset and rapid development. [1] The submandibular space is the main location of infection in Ludwig's angina. Infection of the second and third molars are the primary cause in the majority of Ludwig's angina patients with odontogenic genesis. [2] In the pre-antibiotic era, Ludwig's angina had a mortality of greater than 50%. Presently, it averages approximately 8% due to effective chemotherapy as well as enhanced imaging modalities and surgical methods. [3] Most cases occur in adult males aged 20-60 years. Typically, a history and physical examination are sufficient to make the diagnosis. [2]

### **Case history**

A 50-year-old man with hypertension, ischaemic heart disease and dyslipidaemia presented with mild discomfort in breathing and swallowing of three days duration. Furthermore, there was extensive floor of the mouth and neck swelling of one week's duration. There were no respiratory or gastrointestinal symptoms at presentation. The patient had been treated with oral antibiotics in the primary care setting with no significant clinical improvement. He was an ex-smoker with 20 pack years, abstaining for 5 years. He was a habitual betel (*Piper betle*) leaf chewer using 3 quids per day with tobacco, lime chalk and arecanuts (*Areca catechu*).

The patient was afebrile with a blood pressure of 110/63 mmHg and a pulse rate of 58/min. The pulse oximetry was 98% on room air while the respiratory rate was 18/min. A tender,

http://doi.org/10.4038/jpgim.8427

soft tissue swelling with interspersed boggy and woody consistency extending from the submandibular area to the thyroid cartilage was observed (Figure 1).



The airway did not show any compromise and the vascular structures were not threatened. Multiple tender anterior and posterior cervical lymph nodes were present. The oral hygiene was poor, accompanied by multiple dental caries. The carotid pulses were palpable bilaterally. The rest of the clinical examination was normal.

The initial haemogram showed leukocytosis with a neutrophil predominance (total leukocytes –  $14.62 \times 10^9$ /L, neutrophils - 85.5%, lymphocytes – 7.5%). The haemoglobin was 11.4g/dl while the platelet count was  $297\times10^9$ /L. C-reactive protein was 55.5mg/dl while the ESR was 60 mm in the  $1^{st}$  hour. The renal and liver functions were normal.

Neck ultrasonography revealed extensive soft tissue oedema in the submental and submandibular regions. Several large submental lymph nodes with preserved fatty hila were present. The largest measured 15 mm x 9 mm. Bilateral parotid, thyroid, and submandibular glands appeared normal. A chest X-ray did not reveal any airway compression or neck vessel compromise. The lung fields appeared normal.

http://doi.org/10.4038/jpgim.8427

He was commenced on intravenous co-amoxiclav and metronidazole. The patient was intently observed for possible evolving airway compromise. Oral and maxillofacial consultation detected the presence of periodontal disease. He underwent an incision and drainage of the affected region. The patient was referred to the restorative dentistry unit where he underwent root canal treatment of the left lower second molar tooth, cleaning and irrigation. He was discharged with a review plan at the restorative clinic. Both clinical and biochemical improvement was achieved by the time of the discharge.

### **Discussion**

The clinical presentation of Ludwig angina was first described by Karl Friedrich Wilhelm von Ludwig in 1836 as a severe, rapidly spreading cellulitis that affects the face, neck, and various facial areas. [1] The most frequent location for infections is the bilateral submandibular spaces, though deeper regions may also become infected. Posterior and superior displacement of the tongue and the floor of the mouth can compromise the airway. More serious respiratory difficulties could develop if the infection were to extend to the lateral pharyngeal and retropharyngeal regions. [4]

Ludwig's angina is characterized by mild upper neck pain and swelling, pain in the lower posterior teeth, trismus, swelling of the mouth with floor and tongue displacement to the superior or posterior, tachycardia, fever, and malaise. Persistent swelling of the neck's soft tissues also leads to increasing airway obstruction. Depending on the severity, dyspnoea, dysphagia, and drooling may happen. In the clinical setting, this obstruction characteristically presents with the neck extending into the so-called "sniffing position". If the airway obstruction is not addressed right away, this can be life-threatening as patients develop tachypnoea, stridor and cyanosis, which is followed by death. [5] . The differential diagnoses includes angioneurotic oedema, cellulitis of the floor of the mouth, lingual carcinoma, lymphadenitis, peritonsillar abscess, salivary gland abscess and sublingual hematoma. [6]

Predisposing factors include dental caries (70 – 90%), recent dental interventions and systemic illnesses (1/3<sup>rd</sup> of cases) such as diabetes mellitus, malnutrition, alcoholism and immunocompromised states (e.g. HIV/AIDS, immunosuppressive drugs). [1] Haemolytic *Streptococcus* sp. coupled with anaerobic bacteria like *Peptostreptococcus* sp. and pigmented *Bacteroides* sp. have been characterized as the causal agents. Deep neck infections have resulted in the isolation of *Staphylococcus aureus* (27.3%), *S. epidermis* (22.7%), and the viridans group of streptococci (40.9%). The antibiotics suggested for use prior to culture and antibiogram results are intravenous penicillin G, clindamycin, or metronidazole. [7] We employed parenteral co-amoxiclav and metronidazole

Patients may be treated with observation and scrupulous intravenous antibiotics in the early stages of the disease. Advanced infections necessitate surgical drainage to secure the airway. As our patient was at an early stage of the condition intent observation was carried out with

http://doi.org/10.4038/jpgim.8427

coverage of intravenous antibiotics. However, he required intra-oral surgical interventions later due to poor resolution. Among the consequences of this clinical presentation, life-threatening airway compromise is useful in prognostication which is evidenced by mortality reduction from 50% to 8% following early administration of antibiotics. [8]

### Conclusion

Since Ludwig's angina is a rare clinical presentation with little awareness even among healthcare professionals, it is important to be clinically vigilant to identify and treat immediately since the consequences of missing this kind of diagnosis are going to be detrimental.

#### References

- 1. Saifeldeen K, Evans R. Ludwig's angina. Vol. 21, Emergency Medicine Journal. 2004. p. 242-3. https://doi.org/10.1136/emj.2003.012336
- 2. Spitalnic SJ, Sucov A. Ludwig's angina: case report and review. J Emerg Med. 13(4):499-503. https://doi.org/10.1016/0736-4679(95)80007-7
- 3. Bansal A, Miskoff J, Lis RJ. Otolaryngologic critical care. Crit Care Clin. 2003 Jan;19(1):55-72. https://doi.org/10.1016/S0749-0704(02)00062-3
- 4. Marcus BJ, Kaplan J, Collins KA. A case of Ludwig angina: a case report and review of the literature. Am J Forensic Med Pathol. 2008 Sep;29(3):255-9. https://doi.org/10.1097/PAF.0b013e31817efb24
- 5. Duprey K, Rose J, Fromm C. Ludwig's angina. Int J Emerg Med. 2010 Jun 8;3(3):201-2. https://doi.org/10.1007/s12245-010-0172-1
- 6. Siti Nidzwani Mohamad Mahdi. Delay in Ludwig Angina Diagnosis is Fatal. Journal Article Medicine and Health. Volume 13. Page 170 174. 2018 https://doi.org/10.17576/MH.2018.1302.17
- 7. Har-El G, Aroesty JH, Shaha A, Lucente FE. Changing trends in deep neck abscess. A retrospective study of 110 patients. Oral Surg Oral Med Oral Pathol. 1994 May;77(5):446-50. https://doi.org/10.1016/0030-4220(94)90221-6
- 8. Nicholas Costain, BSc, Thomas J. Marrie, MD Dalhousie University, Halifax, Nova Scotia, Canada. Ludwig's Angina. The American Journal of Medicine, Vol 124, No 2, February 2011