

Ludwig's Angina- a Forgotten Crisis in the Field of Dentistry?

[6].

Vasanth S^{1*}, Gnanam P² and Selvaraj S³

¹Department of Oral and Maxillofacial Surgery, AIMST University, Malaysia ²Department of Prosthodontics, AIMST University, Malaysia ³Department of Public Health Dentistry, AIMST University, Malaysia

***Corresponding author:** Satish Vasanth, Department of Oral and Maxillofacial Surgery, Faculty of Dentistry, AIMST University, Bedong 08100, Malaysia, Email: vasanthsmile@gmail. com

Abbreviations: LA: Ludwig's Angina.

Dear Editor

Ludwig's angina (LA) is an infection of the floor of the mouth that spreads rapidly. Originating as an oral infection, the patient exhibits symptoms like fever, and malaise involving the submandibular, sublingual, and submental spaces [1,2]. This can be seen at any age, even though middle age is the most common; the infection has a 2:1 male predilection. The most common comorbidities of LA are odontogenic infection (especially in the second and third mandibular molars), peritonsillar abscess, and mandibular fracture. Poor dental hygiene (responsible for 75-95% of cases), malnutrition, intravenous drug use, diabetes mellitus, acquired immune deficiency syndrome, immunosuppression, and systemic lupus erythematosus are risk factors for the development of this condition [2]. The most frequent pathogens implicated are alpha hemolytic streptococci, staphylococci, and Bacteroides, and people with a compromised immune system are more likely to acquire the condition. Furthermore, periapical dental abscesses of these molars infect the submaxillary region and the thin inner cortex of the jaw. Communication across the posterior edge of the mylohyoid muscle hastens sublingual space involvement. Cellulitis and swelling displace the tongue superiorly and posteriorly due to the unyielding presence of the mandible, hyoid, and cervical fascia, resulting in bronchospasm and asphyxiation if not treated [2]. Despite a sharp decline in Ludwig's angina mortality rate since the pre-antibiotic period, this illness remains a therapeutic crisis due to its unique life-threatening airway obstruction consequence [3].

In addition, respiratory distress and failure are characterized by difficulty in breathing, stridor, cyanosis,

and changes in the mental state. To add, examining the submandibular region may indicate tenderness, symmetry, stiffness, woodiness, and induration. The mouth may be kept open by a swollen lingual area, while the floor of the mouth may be erythematous, painful, and raised. As a patient's symptoms develop, he or she may lean forward in the tripod posture to maximize airway diameter [4]. Malaise, dysphagia, drooling, bilateral cervical swelling, neck soreness, dysphonia, elevation, posterior displacement and swelling of the tongue, pain in the floor of the mouth, painful throat, restricted neck mobility, and stridor are among the signs and symptoms of this condition which indicates imminent airway blockage. Streptococcus viridans, Staphylococcus aureus, B-hemolytic streptococcus species, Staphylococcus epidermis, Bacteroid genus, Fusobacterium nucleatum, Pepto streptococcus, and Enterobacter aerogenes are common bacteria that cause LA [2]. If a patient develops dyspnea, tachypnea, inspiratory stridor, and cyanosis, these symptoms are symptomatic of developing supraglottic edema, which, if left untreated, can cause airway obstruction and mortality [1] radiographs of the neck and chest may demonstrate the extent of soft tissue swelling and would suggest anaerobic infection if gas is present in the soft tissues [1]. Plain chest radiography can also be used to seek an intrathoracic extension of the infective process [5] with radiographic views of the jaw may indicate a dental focus of infection [6]. After the airway patency is assured, CT (Computed Tomography) scanning is a valuable modality to demonstrate the extent of soft tissue swelling, the presence of gas, fluid collection, and airway compromise

Moreover, the management of these usually young patients requires trained teams combining medical skills in surgery, antibiotic therapy, and resuscitation [7]. Many cases may be initially managed with antibiotics and close

Letter to Editor

Volume 7 Issue 3 Received Date: September 05, 2022 Published Date: September 14, 2022 DOI: 10.23880/oajds-16000344 observation in an intensive care setting, but patients with significant airway swelling, dyspnea, stridor, cyanosis, or worsening airway symptoms require airway intervention [4] clinicians should prepare for a surgical airway, cricothyrotomy may be particularly challenging in these patients due to the distortion of the anterior neck in cases of extensive infection. Broad-spectrum antibiotics covering anaerobic, aerobic, and oral flora are recommended. Clindamycin alone is not recommended, as resistance rates approach over 30% for streptococcal species and MRSA (Methicillin Resistant Staphlyococcus Aureus). Surgical intervention typically includes debriding necrotic tissue and draining all pathologic fluid collections the mortality rate has decreased, ranging from 10% if expediently treated to 50% in those not receiving appropriate management, several complications can occur, with descending mediastinitis comprising one of the most severe complications. Others include necrotizing fasciitis of the neck and chest, pericarditis, carotid artery rupture, jugular vein thrombosis, pleural empyema, pneumonia, and acute distress syndrome [4]. Additional treatment may include intravenous dexamethasone (e.g., 10 mg every 8 hours) for 48 hours to reduce edema helping to preserve airway integrity. Edema in addition to the external compression secondary to inflammation and induration, may cause a narrowing in the laryngeal cavity and result in airway obstruction [2]. A mouth opening of less than 4 cm, which is quite common in LA patients, has been associated with difficult intubation, as well as the presence of loose teeth, dentures, or prominent anterior teeth. The most used intubation techniques for patients with LA include elective tracheostomy, awake blind nasal intubation, and flexible fiber nasal or oral intubation [2] first step is assessment and maintenance of a patent airway. The second step is aggressive antibiotic therapy and the third is surgical evaluation and, if required, operative decompression [1]. Case reports and retrospective studies that have documented that early intervention is the mainstay of treatment recommend that adequate early surgical intervention and intravenous antibiotics should be the treatment of choice in individuals with early Ludwig's angina in resource-challenged centres as the risk of airway compromise will outweigh the benefit of watchful waiting in the event of antibiotic failure [3].

Finally, many studies suggest that the inappropriate use of antibiotics, steroids, and nonsteroidal anti-inflammatory drugs may affect the clinical signs and symptoms of infection and slow the disease course, potentially delaying a correct diagnosis. Reports of steroid use in Ludwig's angina are few, and the literature detailing the effects of steroidal management in these cases remains weak [7]. The hypothesis that NSAIDs facilitate progression to abscess formation is also supported by experimental data [8]. Over the years, the widespread use of antibiotics has decreased the mortality

Open Access Journal of Dental Sciences

rate of Ludwig's angina from 50% to less than 10% [7]. The prognosis is related to surgical delay and quality factors of an individual, such as income, employment, education, and residence which have an influence on the oral health of an individual, which shows that these factors affect inequalities in oral health status [9,10]. Based on myriad studies, it has been found that there is limited awareness of this condition among various populations around the globe, especially in developing and poorly developed nations, where this may still be a life-threatening illness. To conclude, we believe that this is the right time to incorporate awareness for diagnosis and care of these patients and call for the collaboration of global health care professionals by insisting that prevention is better than a cure.

• **Conflict of Interest:** Authors declare that there is no conflict of interest.

References

- 1. Wasson J, Hopkins C, Bowdler D (2006) Did Ludwig's angina kill Ludwig? J Laryngol Otol 120(5): 363-365.
- Dowdy RAE, Emam HA, Cornelius BW (2019) Ludwig's angina: Anesthetic management. Anesth Prog 66(2): 103-110.
- Edetanlen BE, Saheeb BD (2018) Comparison of Outcomes in Conservative versus Surgical Treatments for Ludwig's Angina. Med Princ Prac 27(4): 362-366.
- 4. Bridwell R, Gottlieb M, Koyfman A, Long B (2021) Diagnosis and management of Ludwig's angina: An evidence-based review. Am J Emerg Med 41: 1-5.
- Barakate MS, Hemli JM, Jensen MJ, Graham AR (2001) Ludwig's angina: Report of a case and review of management issues. Ann Otol Rhinol Laryngol 110(5): 453-456.
- 6. Lemonick DM (2002) Ludwig's Angina: Diagnosis and Treatment. Hosp Physician.
- Othman TS, Sudhakar A, McKinnon BJ (2020) Ludwig's angina and steroid use: A narrative review. Am J Otolaryngol 41(3): 102411.
- Vallée M, Gaborit B, Meyer J, Malard O, Boutoille D, et al. (2020) Ludwig's angina: A diagnostic and surgical priority. Int J Infect Dis 93: 160-162.
- Selvaraj S, Naing NN, Wan Arfah N, de Abreu MHNG (2021) Assessment on Oral Health Knowledge, Attitude, and Behavior and its Association with Sociodemographic and Habitual Factors of South Indian Population. Pesqui

Open Access Journal of Dental Sciences

Bras Odontopediatria Clin Integr 21: e0135.

10. Selvaraj S, Naing NN, Wan Arfah N, de Abreu MHNG (2021) Demographic and habitual factors of periodontal

disease among south Indian adults. Int J Environ Res Public Health 18(15): 7910.

