



INSIGHT TO LUDWIG'S ANGINA: A REVIEW

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Article Info

Received: 01/07/2014

Revised: 15/08/2014

Accepted: 19/08/2014

Key words: Ludwig's Angina, Abscess, Odontogenic Infections, Submandibular Space, Submental Space etc.

ABSTRACT

Ludwig's angina is a potentially life-threatening, rapidly expanding, diffuse inflammation of the submandibular and sublingual spaces that occurs most often in young adults & children with odontogenic infections in whom it can cause serious airway compromise. Symptoms include severe neck pain and swelling, fever, malaise, dysphagia & stridor. Causative bacteria include many gram-negative and anaerobic organisms, streptococci and staphylococci. Initial treatment consists of high doses of penicillin G given intravenously, sometimes in combination with other drugs. Patients usually recover without complications. This review provides an overview of the anatomical and pathophysiological considerations in Ludwig's angina and describes practical management principles to assist dentist in the diagnosis and treatment of this disease.

INTRODUCTION

Originally described by Wilhelm Frederick von Ludwig in 1836, this condition is notorious for its aggressiveness, rapid progression to airway compromise and high mortality when not treated promptly. Ludwig's angina is a serious, potentially life-threatening cellulitis or connective tissue infection of the floor of the mouth that spreads to the structures of the anterior neck and beyond via connective tissue, muscle and fascial planes rather than by the lymphatic system. For this reason, adenopathy is not associated. It usually occurs in adults with concomitant dental infections [1]. Angina ludovici, cyananche, carbunculus gangraenosus, morbus strangulatorius and angina maligna are its synonymous terms. Ludwig's angina should not be confused with angina pectoris, which is also otherwise commonly known as "angina". The word "angina" comes from the greek word ankhon, meaning "strangling", so in this case, Ludwig's angina refers to the feeling of strangling, not the feeling of chest pain, though there may be chest pain in Ludwig's angina if the infection spreads into the retrosternal space. Ludwig's angina can be thought of as a cellulitis of the submandibular space [2-4].

Causes

About 80% of cases of Ludwig's angina are odontogenic in etiology, primarily resulting from infections of the second and third molars. These teeth have roots that lie at the level of the mylohyoid muscle and abscesses here can spread to the submandibular space. Once infection develops, it spreads contiguously to the sublingual space. Infection can also spread contiguously to involve the pharyngomaxillary and retropharyngeal spaces, thereby encircling the airway. The majority of cases of Ludwig's angina occur in healthy patients with no co morbid diseases. Nevertheless, there are several conditions that have been shown to predispose patients to Ludwig's angina [5]. These conditions include diabetes mellitus, alcoholism, acute glomerulonephritis, systemic lupus erythematosus, aplastic anemia, neutropenia and dermatomyositis. Although the widespread involvement seen in Ludwig's usually develops in immunocompromised persons, it can also develop in otherwise healthy individuals. Thus, it is very important to obtain dental consultation for mandibular-third molars at the first sign of



any pain, bleeding from the gums, sensitivity to heat/cold or swelling at the angle of the jaw. Other causes of Ludwig's angina (besides spread of dentoalveolar infection) include [6-8].

- Fractured mandible
- Trauma to neck & floor of mouth
- Foreign body in the floor of the mouth
- Tongue piercing & oral lacerations resulting in infection
- Secondary infections of an oral malignancy
- Otitis media
- Spread of infections around the oral region: submandibular sialadenitis, peritonsillar abscess
- Parapharyngeal infections
- Infected thyroglossal duct cyst
- Lymphatic vascular malformations
- Intravenous injections of drugs into the neck
- Traumatic bronchoscopy
- Endotracheal intubation

Anatomy

In order to appreciate the potential of this infection to spread from the floor of the mouth to the neck and mediastinal structures, a brief review of the involved anatomy is helpful (**Figure 1**). The submandibular space comprises part of the space above the hyoid bone. The total space is divided into the sublingual space superiorly and submandibular space inferiorly. The former, located between the geniohyoid and mylohyoid muscles and the latter, located between the mylohyoid muscle and the superficial fascia and skin, communicate around the free posterior border of the mylohyoid muscle. Once established in the submandibular space, the infection can then spread to adjacent structures. Typically affected structures, in order of most frequent contamination, are the anterior neck, the pharyngomaxillary space, the retropharynx and the superior mediastinum. The submandibular space comprises part of the space above the hyoid bone. Odontogenic infections break through the relatively thin cortex of the mandible below the mylohyoid ridge [9-10].

Pathophysiology

Ludwig's angina is a rapidly progressing polymicrobial cellulitis of the sublingual and submandibular spaces and predominantly involves the oral flora. Polymicrobial infection occurs in over 50% of cases. The organisms most often isolated in patients with the disorder are *Streptococcus viridans* and *Staphylococcus aureus*. Anaerobes also are frequently involved, including bacteroides, peptostreptococci and peptococci. Other gram-positive bacteria that have been isolated include *Fusobacterium nucleatum*, *Aerobacter aeruginosa*, spirochetes, *Veillonella*, *Candida*, *Eubacteria* and *Clostridium* species. Gram-negative organisms that have been isolated include *Neisseria* species, *Escherichia coli*, *Pseudomonas* species, *Haemophilus influenzae* and

Klebsiella species. Patients with immunocompromising conditions, such as HIV, diabetes, transplant recipients, and alcoholics, are at risk for infection from a variety of atypical organisms [11].

Symptoms and Signs

True Ludwig's Angina is a cellulitic facial infection. The signs are bilateral involving lower facial swelling, pain & erythema around the lower jaw and upper neck (**Figure 2**). This is because the infection has spread to involve the submandibular, sublingual and submental spaces of the face. Swelling of the submandibular space, while externally is concerning the true danger lies in the fact that the swelling has also spread inwardly compromising, or in effect narrowing the airway. Dysphagia & odynophagia are symptoms that are typically seen and demand immediate attention.¹² The sublingual and submental spaces are anterior (beneath the middle and chin areas of the lower jaw) to the submandibular space. Swelling in these areas can often push the floor of the mouth, including the tongue upwards and backwards further compromising the airway. Localisation of infection to the sublingual space is accompanied by swelling of structures in the floor of the mouth as well as the tongue being pushed upwards and backwards. Spread of infection to the submaxillary spaces is usually accompanied by signs of cellulitis rather than those of an abscess. Submental and submandibular regions are swollen and tender. Additional symptoms include malaise, fever, tiredness, confusion, anxiousness, agitation, earache, drooling of saliva, fetid breath. There may also be varying degrees of trismus. Hoarseness, stridor, respiratory distress, decreased air movement, cyanosis and a "sniffing" position (i.e., the characteristic posture assumed by patients with impending upper airway compromise consisting of an upright posture with the neck thrust forward and the chin elevated) are all signs of impending airway catastrophe [13].

Patients may exhibit dysphonia. More specifically, they may have a muffled tone at higher registers a "hot potato" voice caused by edema of the vocal apparatus; this finding should be a warning to clinicians of potentially severe airway compromise. On oral examination, elevation of the tongue, woody, brawny induration of the floor of the mouth and anterior neck and nonfluctuant suprahyoid swelling typify the disease process. There is typically a bilateral submandibular edema, with marked tenderness on palpation and occasionally, subcutaneous emphysema. The swelling of the anterior soft tissues of the neck above the hyoid bone sometimes leads to the characteristic "bull's neck" appearance of affected patients [14].

Possible Complications

Prior to the development of antibiotics, mortality for Ludwig's angina exceeded 50%. As a result of antibiotic therapy, along with improved imaging modalities and surgical techniques, mortality currently averages



approximately 8%. Cellulitis, rather than abscess formation is the most common early presenting finding. As the infection progresses, edema of the suprahyoid tissues and supraglottic larynx elevate and posteriorly displace the tongue, resulting in life-threatening airway narrowing or obstruction. In advanced infection, cavernous sinus thrombosis and brain abscess have been described. Other reported complications of Ludwig's angina include carotid sheath infection and arterial rupture, suppurative thrombophlebitis of the internal jugular vein, mediastinitis, empyema, lung abscess, pericardial and/or pleural effusion, osteomyelitis of the mandible, subphrenic abscess, septic shock and aspiration pneumonia [15].

Diagnosis

Awareness and recognition of the possibility of Ludwig's angina is the first and most essential step in the diagnosis and management of this serious condition. There are 4 cardinal signs of Ludwig's angina: (1) bilateral involvement of more than a single deep tissue space; (2) gangrene with serosanguinous, putrid infiltration but little or no frank pus; (3) involvement of connective tissue, fasciae and muscles but not glandular structures; and (4) spread via fascial space continuity rather than by the lymphatic system. The presence of brawny induration of the floor of the mouth in a suggestive clinical presentation should prompt the clinician to move rapidly toward airway stabilization first, followed by further diagnostic confirmation [16].

Management

It involves appropriate antibiotic medications, monitoring and protection of the airway in severe cases and where appropriate, urgent maxillo-facial surgery and/or dental consultation to incise and drain the collections. Antibiotics should be initiated as soon as possible. Antibiotics should initially be broad-spectrum and cover gram-positive, gram-negative and anaerobic organisms. The antibiotic of choice is from the penicillin group. Recommended initial antibiotics are high-dose penicillin G, sometimes used in combination with an anti-staphylococcal drug or metronidazole (Flagyl I.V.). Benzylpenicillin 1.2g IV every 6 hours can be given. In penicillin-allergic patients, clindamycin hydrochloride (Cleocin HCl) 450 mg IV every 8 hours is a good choice. Alternative choices include cefoxitin sodium (Cefoxil) or combination drugs such as ticarcillin-clavulanate (Timentin), piperacillin-tazobactam (Zosyn) or amoxicillin-clavulanate (Augmentin). Intravenous dexamethasone sodium phosphate (Decadron) 8-12 mg IV initially then of 4-8mg every 6 hours given for 48 hours, has been beneficial in reducing edema, which helps maintain airway integrity and enhances antibiotic penetration [17].

Recent case reports have advocated the use of intravenous steroids. In these reports, corticosteroid administration potentially avoided the need for airway

management. To date, there are no randomized controlled trials that demonstrate the efficacy of corticosteroids in patients with Ludwig's angina [18].

Up to 65% of patients with Ludwig's angina develop suppurative complications that require surgical drainage. Physical examination alone is insufficient in determining which patients require a surgical procedure. In a recent study of deep neck space infections, the clinical exam underestimated the true extent of infection in 70% of patients. As a result, imaging is indicated in patients with Ludwig's angina once antibiotics have been administered and decisions in regard to airway management have been made. Although plain films can demonstrate submandibular soft-tissue swelling, they are inadequate in detecting patients who require surgical drainage. As a result, a CT scan with intravenous contrast is recommended to detect patients who have developed suppurative complications. Early detailed imaging is essential to evaluate the extension of tissue infection or necrosis and to guide decisions regarding surgical approaches when indicated. CT scan and MRI are of invaluable importance in the assessment of deep neck space infections and collections [19].

Miller et al [20] reported that combined clinical evaluation and CT findings lead to accuracy of 89%, sensitivity of 95%, and specificity of 80 % in identifying drainable collection. Plain chest radiographs are useful when looking for signs of mediastinum extension such as mediastinitis and pleural effusion. Although ultrasound is not as easily interpreted by clinicians and surgeons as other imaging modalities, its availability, cost effectiveness, reduced risk of radiation and accuracy in differentiating cellulites-related oedemas from abscess collections make it a reliable supplement modality to CT scan in resistant cases [21].

Ludwig's angina is a life-threatening condition and carries a fatality rate of about 5%. Airway management is the foundation of treatment for patients with Ludwig's angina. Recommended techniques include routine orotracheal intubation and fiber-optic nasotracheal intubation. A nasotracheal tube is sometimes warranted for ventilation if the tissues of the mouth make insertion of an oral airway difficult or impossible. In cases where the patency of the airway is compromised, skilled airway management is mandatory. Fiberoptic intubation is common. Blind nasotracheal intubation should not be attempted in patients with Ludwig's angina given the potential for bleeding and abscess rupture. In nonintubated patients with Ludwig's angina, airway equipment, including tracheostomy and cricothyroidotomy instruments must be at the bedside [22]. Although no specific guidelines are present for managing acute Ludwig's angina, decisions regarding airway protection are largely dependent on the "Practice Guidelines for Management of the Difficult Airway" that were adopted by the American Society of Anaesthesiologists in 1992 and updated in 2003. In these guidelines, a difficult airway is defined as "the



clinical situation in which a conventionally trained anaesthesiologist experiences difficulty with face mask ventilation of the upper airway, difficulty with tracheal intubation, or both.” When that is the case, patients are intubated via awake assisted fiberoptic bronchoscope. When this fails a surgical tracheostomy is performed under local anaesthesia. The guidelines specify that these recommendations may be adopted, modified, or rejected according to the clinical needs and constraints as these guidelines are not intended as standards or absolute requirements and their purpose is to assist the practitioner in decisions about health care [23].

A study on 41 patients, 24% being children and 76% adults was conducted. In children, 70% were controlled with conservative medical management while 81% of adults required incision and drainage. Tracheostomy was necessary in 10% of the children and in 52% of the adults. Mortality rate was 10% in both groups [24]. A 9-year review by Greenberg et al. of 29 cases of deep neck space infections reported 21 patients (72 %) treated conservatively following initial clinical assessment. One of these patients subsequently deteriorated requiring emergency intubation. Of those treated non-conservatively at initial presentation, 7 (24%) patients were able to be intubated using fiberoptic nasoendoscopy and 1(3%) patient required tracheostomy under local anaesthesia [25].

Larawin et al. retrospectively studied a total of 103 patients with deep neck space infections from 1993 to 2005. Ludwig’s angina was the most commonly encountered infection seen in 38 (37%) patients of treatment. 13 (34%) patients managed successfully with

medical therapy and only 4 (10%) patients required a tracheostomy tube [26].

Kurien et al. reported a 13-year review of patients with Ludwig’s angina between 1982 and 1995. Patients were either admitted to the ENT or paediatric surgical units [27].

Initial airway assessment is based on respiratory rate, oxygen saturation, and findings on fiberoptic laryngoscopy. Patients are then categorised as having either a severe airway compromise or a stable airway. In the severely compromised group (patients unable to maintain saturation on room air above 95%, respiratory rate > 25, or a significant airway compromise on fiberoptic laryngoscopy) a definitive airway is required. Awake fiberoptic-assisted intubation should be attempted first; if this fails then a surgical tracheostomy is performed under local anaesthesia.

In the other group, where patients are able to maintain normal oxygen saturation and respiratory rate on room air and where no significant airway compromise is evident on fiberoptic examination, airway is managed conservatively. This involves close airway observation (oxygen saturation, respiratory rate, and serial fiberoptic laryngoscopy) in a high dependency unit (HDU) or ENT ward. After the initial clinical assessment and airway decision all patients should undergo CT scanning of their neck and thorax for further detailed airway and deep neck spaces evaluation. Any abscess or collection cavity should be drained, and both groups should be kept in an HDU or ENT ward for hourly airway assessment for 24 – 48 hours [28].

Figure 1. Sublingual Space, Superior to Mylohyoid Muscle. The Submandibular Space Is Inferior to the Mylohyoid Muscle

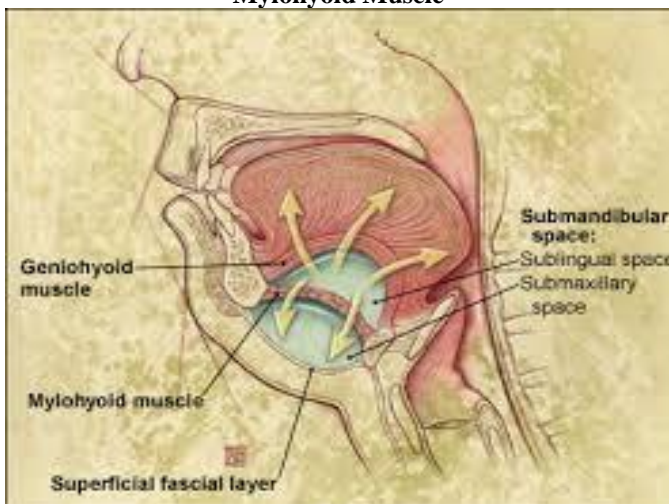


Figure 2. Clinical Picture of Ludwig’s Angina



CONCLUSION

The life-threatening nature of this condition generally necessitates surgical management with involvement of critical care physicians such as those found in an intensive care unit. Early diagnosis and immediate treatment is the

key for successful management of Ludwig’s angina. In advanced cases, securing the airway, surgical drainage and antibiotics following culture and sensitivity test are important.



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