Original Research Article

A study of clinical profile and factors associated with Ludwig's angina at tertiary health care centre

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Abstract

Ludwig's anginais a rapidly progressive, serious, potentially life-threatening, bilateral cellulitis of the submandibular space. Many general symptoms such as pyrexia, weakness, and fatigue, develop as the result of the immune response associated with bacterial infection. Among the main etiologic factors of the angina is the tooth infection, for example, a recent tooth extraction, endodontic and periodontal condition and tooth trauma. A history of recent dental extraction or of poor oral hygiene and dental pain is significantly associated with odontogenic infection and Ludwig's angina. Cellulitis, rather than abscess formation, is the most common early presenting finding. Among the complications after Ludwig's angina will include cavernous sinusthrombosis and brain abscess, in addition to airway compromise, have been described.

Key Words: Ludwig's angina

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INTRODUCTION

Ludwig's angina is often an infection of odontogenic origin affecting the soft tissues of the submandibular, sublingual and submental area. It's a rapidly progressive, serious, potentially life-threatening, bilateral cellulitis of the submandibular space associated with elevation and posterior displacement of the tongue usually occurring in adults with concomitant dental infections.[1] The Ludwig's angina was firstly described in 1836 by Wilhelm Frederick von Ludwig as a cellulitis of fast evolution involving the region of the submandibular gland which is disseminated through anatomic contiguity without tendency towards abscess formation.[2] Among the main etiologic factors of the angina is the tooth infection, for example, a recent tooth extraction, endodontic and periodontal condition and tooth trauma.[3] However, Gulinelli *et al.* pointed out other factors such as in the cases of submandibular sialadenitis and parapharyngeal or peritonsillar abscesses.[4]In the case of Ludwig's angina, it refers to the feeling of strangling and choking secondary to lingual airway obstruction, which is the most serious potential complication of this condition. Ludwig's angina is known by many alternative names, including cynanche, carbuculusgangraenosus, angina maligna, morbusstrangularis, and garotillo.[5] Ludwig's angina preantibiotic era carried a very highmortality rate of around 50%,but it is still considerablyhigh today at around 8%–10%.The cause is often a polymicrobial bacterial infection that includes group A Streptococcus species. Other commonly cultured organisms include Staphylococcus, Fuso- bacterium, and Bacteroides species. Patients who are immunocompromised are commonly infected with an atypical organism, such as Pseudomonas, Escherichia coli, Candida, or Clostridium.[6]

MATERIAL AND METHODS

Its a prospective, observational type of study carried out at tertiary health care centre. Patients presenting with odontogenic infection were followed over a study period of one year i.e. fron January 2015 to December 2015. Those patients manifesting clinical features of Ludwig's

Angina were studied in details with regard to factors associated with the present situation. 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;
4. Spread via fascial space continuity rather than by the lymphatic system.[7]

The presence of brawny in duration 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. In 1939, Grodinsky developed criteria for the diagnosisof Ludwig's angina. There must be cellulitis, not an abscess, of the submandibular space that never involves only one space and usually is bilateral; produces gangrene with serosanguineous, putrid infiltration but very little frank pus; involves connective tissue, fascia, and muscles but not glandular structures; and is spread by continuity and not by lymphatics.[8]

Clinical Manifestations: The symptoms of Ludwig's angina vary depending on the patient and the degree of infection. Many general symptoms such as pyrexia, weakness, and fatigue, develop as the result of the immune response associated with bacterial infection. The inflammatory response leads to edema of the neck and tissues of the submandibular, submaxillary, and sublingual spaces. Significant edema may causetrismus and an inability to swallow saliva. Pain, especially with tongue movement, is common with Ludwig' sangina. Symptoms marking progressive disease with significant obstruction of the airway include respiratory distress with dyspnea, tachypnea, or stridor. Patients mayexhibit dysphonia. More specifically, they may have amuffled tone at higher registers (ie, a "hot potato" voice) caused by edema of the vocal apparatus; this finding should be a warning to clinicians of potentially severe airwaycompromise. Confusion or othermental changes may occur because of prolonged hypoxia. Otalgia, dysphagia, dysphonia, and dysarthria are also observed. As with any bacterial infection, sepsis may occur. Without immediate treatment, the submandibular infection may also rapidly spread to the mediastinal orpharyngomaxillary spaces or to the bone, resulting inosteomyelitis. Physical signs associated with more progressive disease and airway obstruction include audible stridor, dysphonia, severe dehydration, and enlargement of cervical lymphnodes.

Factors associated with Ludwig's angina: Patients with Ludwig's angina typically have a history of recent dental extraction or of poor oral hygiene and dental pain. It usually originates from an **odontogenicinfection**, especially from the second or third lower molars. Dental infections account for approximately 80% of casesof Ludwig's angina. Mixed infections are due to both aerobes and anaerobes, are of the cellulitis associated with Ludwig'sangina. Typically, these include alpha-hemolyticstreptococci, staphylococci and bacteroides groups. It is very important to obtain dentalconsultation for lower-third molars at the first sign of anypain, bleeding from the gums, sensitivity to heat/cold orswelling at the angle of the jaw. In a study that was conducted on 16different patients suffering from ludwigs angina, Odontogenic infection was the commonest aetiologic factor observed in 12 cases (75%), trauma was responsible for 2 (12.5%) while in the remaining 2 patients (12.5%) the cause could not be determined. Of those with odontogenicorigin, 4 (25%) were due to post dentalextraction sepsis.[9]

Other **less commonly reported causes** of Ludwig's angina includesialadenitis, peritonsillar abscess, open mandibular fracture, infected thyroglossal duct cyst, epiglottitis,intravenous injections of drugs into the neck, traumaticbronchoscopy, endotracheal intubation, oral lacerations,tongue piercing, upper respiratory infections, and trauma to the floor of the mouth. Cellulitis, rather than abscess formation, is the most common early presenting finding. Asthe infection progresses, edema of the suprahyoid tissuesand supraglottic larynx elevate and posteriorly displacethe tongue, resulting in life-threatening airwaynarrowing.[10]

Complications of Ludwig's angina[11]: In advanced infection, cavernous sinusthrombosis and brain abscess, in addition to airway compromise, have been described. Other reported complications of Ludwig's angina include carotidsheath infection and arterial rupture, suppurative thrombophlebitis of the internal jugular vein, mediastinitis, empyema, pericardial and/or pleural effusion, osteomyelitis of the mandible, subphrenic abscess, and aspiration pneumonia. The differential diagnosis of Ludwig's angina include sangioneuroticedema, lingual carcinoma, sublingualhematoma (following anticoagulation), salivarygl and abscess, lymphadenitis, cellulitis, and peritonsillarabscess

SUMMARY AND CONCLUSIONS

Ludwig's angina is a dramatic, life-threatening, softtissueinfection of the floor of the mouth and neck.

Ludwig's angina can arise from various sources such as odontogenic infection, or complicated cases of submandibular gland sialadenitis and sialolithiasis, tongue base lymphangioma, and tongue piercing. Early andliberal consultation with otolaryngology and anaesthesiology services will assure the greatest hope for speedy airwaycontrol, prompt institution of intravenous antibiotictherapy, and an uncomplicated recovery from this rare and dreaded condition.

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