Ludwig’s Angina: Causes Symptoms and Treatment

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Abstract:
Ludwigs angina is a disease which is characterised by the infection in the floor of the oral cavity. Ludwig's angina is also otherwise commonly known as "angina". Previously this disease was deemed as fatal but later on it was concluded that with proper treatment this infection can be removed and the patient can recover. It mostly occurs in adults and children are not affected by this disease. As the infection spreads further it would affect the wind pipe and lead to swellings of the neck. The skin around the neck would also be infected severely and lead to redness. The individual would mostly be febrile during this time. Since the airway is blocked the individual would suffer from difficulty in breathing. If the infection spreads to the internal ear then the individual may have audio impairment. The main cause for this disease is dental infections caused due to improper dental hygiene.

Keywords: Ludwigsangina, trasechtomy, fiberoptic intubation

INTRODUCTION:
Ludwig's angina, otherwise known as Angina Ludovici, is a serious, potentially life-threatening cellulitis, or connective tissue infection, of the floor of the mouth, usually occurring in adults with concomitant dental infections and if left untreated, may obstruct the airways, necessitating tracheotomy. It is named after the German physician, Wilhelm Friedrich von Ludwig who first described this condition in 1836. Other names include "angina Maligna" and "Morbus Strangularis". 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 ankhn, 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. 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. (4) The microbiology of Ludwig’s angina is polymicrobial and includes many grampositive and negative aerobic/anaerobic organisms, but commonly isolated are streptococcal spp, staphylococcal aureus, prevotellaspp and porphyromonas spp (3)(10)

CAUSES:
Dental infections account for approximately 80% of cases of Ludwig's angina. Mixed infections, due to both aerobes and anaerobes, are of the cellulitis associated with Ludwig's angina. Typically, these include alpha-hemolytic streptococci, staphylococci and bacteroides groups. (5) The route of infection in most cases is from infected lower molars or from pericoronitis, which is an infection of the gums surrounding the partially erupted lower (usually third) molars. 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 lower-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.

There has been a single case reported where Ludwig's angina was thought to be caused by a recent tongue piercing (6)(8)(7). In a study that was conducted on 16 different patients suffering from ludwigs angina, Odontogenic infection was the commonest aetiological 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 dental extraction sepsis.

There was associated respiratory difficulty due to gradual progression of the inflammatory lesion to the neck in 11 cases (68.8%). Seven patients (43.8%) showed clinical evidence of underlying systemic illness. These were diabetes mellitus 25% (4 cases) and 1 case (6.3%) each of bilateral lobar pneumonia, severe anemia in pregnancy and mental retardation. (2) With exception of the diabetic patients, others had packed cell values ranging from 11 to 23%, the white blood cell count also ranged from 8 to 15,000 X 107/L while the electrolytes and urea levels were within the normal limits. None was positive for the human immunodeficiency virus. The results of microbiology, culture and sensitivity tests from pus swabs in 11 patients (68.8%) revealed Staphylococcus aureus (6 cases), haemolytic streptococcus (3 cases), Klebsiella pneumonia (2 cases) and one each of Pseudomonas aeruginosa, Proteus mirabilis, Echerichia coli, Prevotellaintermedia (8) and Citrobacterfreundi. In three patients the culture yielded “no growth”, while anaerobic culture was carried out in only one case which yielded Citrobacterfreundl and Prevotelladenticola. The facility for routine culture of anaerobes was not available in the centre.

SYMPTOMS AND SIGNS:
The infected area swells quickly. This may block the airway or prevent you from swallowing saliva. The first and most important symptom that would be shown by a patient suffering from ludwigs’s angina is that he would face breathing difficulties. This is mostly due the blockage of the airway after the infection has spread to the extent of infecting the wind pipe.

Neck infections and swellings are also a common symptom of this disease because once infected the patient would feel uncomfortable in swallowing and deglutination. The patient would also complain of severe neck pain as a part of the infection. Redness of the skin and increase of surficial...
temperature around this area is accounted for the same area. It has also been reported in one of the cases that the patient had discharge of intra oral pus.(10) Patients become febrile due to the spread of infection but this is generally not noted as a primary symptom. (10) This is complicated by pain, trismus, airway edema, and tongue displacement creating a compromised airway. It is also possible that the patient may feel mental distortion and lack of proper cognitive functioning under such cases. This has been accounted by some specialists due to lack of oxygen supply to the brain. But this particular symptom is still put under research to confirm its frequency of occurrence. If the infection spreads to the auditory canal the patient may suffer from severe ear pain and headache. In 10% of cases the patient suffered from impaired hearing. This is mostly because the infection would have spread to the internal ear.

**OTHER SYMPTOMS:**

True ludwigs angina is acellulitic facial infection. The signs are bilateral, lower facial swelling around the lower and upper neck. This is because the infection the infection has spread to involve the sub mandibular sublingal and submental spaces of the face. Swelling of the submandibular space, while is concerning, the true danger lies in the fact that the swelling has also spread inwardly-compromising, or in effect narrowing airway. Dysphagia (difficult in swallowing), odonphagia (pain during swallowing) are symptoms that are typically seen and demand immediate attention. The sublingual nadsamental 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. Localization of infection to the sublingual space is accompanied by the 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 such signs of cellulitis rather than those of an abscess. Submental and submandibular regions are swollen and tender. Additional symptoms include malaise, fever, dysphagia, odonphagia and in severe cases stridor or difficulty breathing. There may also be varying degrees of trismus. Swelling of submandibular and/or sublingual space is imminent. The patient's speech would sound very unusual and resembles the sound of a person who has a "hot potato" in the mouth. The patient will also be generally weak and suffer from fatiguess and excessive tiredness.

**DIAGNOSIS AND TREATMENT:**

Ludwig's angina was formerly invariably fatal but now, with adequate surgical and antibiotic treatment, has a much reduced rate of mortality. Treatment involves appropriate and antibiotic medications, monitoring and protection of the airway in severe cases, and appropriate urgent maxillofacial surgery and/or dental consultation to incise and drain the collections. The antibiotic of choice is from the pencillingroup. Incision and drainage of the abscess may either be intra oral or external. An intra oral incision and drainage procedure is indicated if the infection is localized to the sublingual space. External incision and drainage is performed if infection involves the peri-mandibular spaces. A naso-tracheal tube is sometimes warranted for ventilation if the tissues of the mouth make incision of an oral airway difficult or impossible. In cases where the patency of the airway is compromised, skilled airway management is mandatory. Fibro-optic intubation is common. Dental treatment may be needed for tooth infections that cause Ludwig's angina. If the swelling is interfering with the breathing then the main treatment is to remove the blockage of the airway and restore proper breathing in the individual. (3) If the swelling blocks the airway, medical help would be needed right away. You may need to have surgery called tracheostomy that creates an opening through the neck into the windpipe. Antibiotics are given to fight the infection. They are usually given through a vein until symptoms go away. Antibiotics taken by mouth may be continued until tests show that the bacteria have gone away. A diagnosis of Ludwigs's angina was made and the patient was scheduled for emergency drainage of the abscess. Surgery may be needed to drain fluids that are causing the swelling. (3) Awake fiberoptic intubation was planned, with tracheostomy as a backup. The procedure and need for awake nasal intubation was explained to the patient and written informed consent was obtained for awake intubation and tracheostomy. The patient was premedicated with intramuscular glycopyrrolate 0.4 mg. No acid aspiration prophylaxis was administered. Nasal decongestion was accomplished using oxymetazoline 0.05% nasal drops, one drop in each nostril, and lignocaine 4% topical, two drops in each nostril, was used to anesthetize the nasal mucosa. The base of the tongue and pharyngeal walls were anesthetized with lignocaine 2% viscous gargle 5 ml which was spat out, and 10% lignocaine two puffs, which was sprayed onto the posterior pharyngeal wall. (10) The following morning the patient was comfortable, with a pulse rate of 68 beats per minute, blood pressure of 110/70 mmHg and oxyhemoglobin saturation of 97%. The neck swelling had subsided. A thorough oral suction was performed and the trachea was extubated. The fibroscope was kept handy; however, no elaborate preparations for tracheostomy or similar procedures were made in view of the edema having subsided and as there was no significant anticipation of airway difficulty. Postextubation recovery was uneventful. The patient was discharged 4 days later. (10)

**CONCLUSION:**

Thus Ludwigs’s angina is a life threatening condition, and carries a fatality rate of about 5%. Ludwigs’s angina can arise from various sources such as odontogenic infection, 6, or complicated cases of submandibularglandsialadenitis and sialolithiasis, tongue base lymphangioma, and tongue piercing7-9, 13, but several studies support our finding that there is usually a dental focus of infection. Life threatening complications such as respiratory obstruction, mediastinitis, pleural empyema, pericarditis, pericardial tamponade are often associated with Ludwigs’s angina6, 12, 13. This is in conformity with our study and in conjunction with other factors accounted for the fatalities we recorded. Research
has also shown that Ludwig’s angina has a mortality rate of 8-10%(6) (10)(23) and this occurs most often due to hypoxia or asphyxia rather than overwhelming sepsis(12 which this study has substantiated. The relatively higher rate in the present study can be attributed to late presentation, presence of uncontrolled underlying disease especially diabetes mellitus, and socioeconomic constraints with inability to procure more effective prescribed antibiotics.

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