REVIEW ARTICLE

Ludwig's Angina - Review of Seven Cases

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lntroduction:

Ludwig's angina is a rare deep soft tissue infection that occurs on the floor of the mouth underneath the Tongue. This bacterial infection often occurs after a tooth abscess. Which is a collection of pus in the center of a tooth. It can also follow other mouth infection or injuries.

Ludwig's angina is also known as angina Ludwig's ,angina maligna or morbus strangularis because of the inflammation it causes in the floor of the mouth Causing a strangulating feeling like angina pectoris or chort pains .

Ludwig's angina is best described by Karl fried rich Wilhelm von Ludwig in 1836, as a rapidly and frequently fatal progressive gangrenous cellulitis and edema of the soft tissues of the neck and floor of the mouth[1-5]. The disease extends by continuity rather than lymphatic spread6. Airway compromise has been recognized as the leading cause of death. Mortality exceeded 5O% but since the introduction of antibiotics in 1940's, improved oral and dental hygiene and aggressive surgical approach, the mortality rate was reduced significantly[7-11]. This resulted in the rare occurrence of the disease leaving many physicians with increasingly limited experience of Ludwig's angina. ln most cases, the primary cause of Ludwig's angina is odontogenic infection [12,13]. Other etiologies include peritonsillar and para-pharyngeal abscesses, oral lacerations/piercing. lymphangiomas, mandibular fractures or submandibular sialoadenitis[14-18]. Predisposing factors include: dental carries, recent dental treatment, systemic illness such as diabetes mellitus, malnutrition, alcoholism, compromised immune system such as AIDS, and organ transplantation trauma[19-23]. ln children, it can occur de novo, without any apparent cause[24'25]. Early recognition of the disease is of paramount importance. Painful neck swelling, tooth pain, dysphagia, dyspnoea, fever, and malaise are the most common complaints. Neck swelling and a protruding or elevated tongue are seen in the vast majority of cases. Stridor, trismus, cyanosis and tongue displacement suggest an impending airway crisis26. Edema and induration of the anterior neck, often with cellulitis, may be present in advanced cases[27,28.]

Review of cases: Seven cases of Ludwig's Angina were diagnosed and treated who had edema of the floor of the mouth and the tongue along with submandibular swelling and inability to open the mouth. All the patients have dental infection.Incislon and drainage given to all of the patients through the sub mental region and submandibular region immediately after admission under local anesthesia. The edema and trismus subsided gradually.

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All the patients required daily dressing.After wound healing two patients require skin grafting.

Despite the decreasing incidence of the disease, Ludwig's angina remains an important cause of airway obstruction that can have disastrous consequences. Proper diagnosis, airway control, antibiotic therapy and surgical incision and drainage are essential ensure the safety of the patients.Seven cases admitted to otolaryngology Department of ENT - Head & Neck Surgery, Holy Family Red Crescent Medical College Hospital. Eskation, Dhaka with Ludwig's Angina was included in the study.

The study period was from January 2017 to January 2018. We studied seven patients who admitted into Department of ENT - Head & Neck Surgery, Holy Family Red Crescent Medical & College Hospital, Eskation, Dhaka in the last one years period. After admission into ENT department, we took them into emergency operation theatre immediately and give them horseshoe shape incision in sub mental region and drained the pus under local anaesthesia. Huge amount of pus came out which was very much foul smelling and pus sent for c/s .

After incision and drainage all the patients transferred into ward and we gave intravenous fluid, parenteral antibiotics like ceftriaxone and metronidazole, injectable analgesic and maintain oral hygiene by gargling with hydrogen peroxide mouth wash. They required daily dressing with aseptic precaution and patients were improved gradually. Five patients submardibular swelling, trismus disappeared within seven to ten days and We did skin grafting of two patients after one month of daily dressing . Patients were released with advice.

The age range of our cases are in between 20 to 60 years. The lowest age limit was 20 years old male and highest age limit was 60 years. Among 7 patients 5 were male patients and two was female, and male female ratio was 5:2. The presenting complaints were swelling in the submandibular region, and floor of the mouth, inability to open the mouth, dysphagia and foul smelling from the mouth. Two patients has respiratory distress and burst would with skin lost over the neck region. All the patients have history of dental infection. Their temperature on arrival was 100o F and the rest of their vital signs were stable. One patient have stridor or cyanosis. Their physical examination was significant for tongue swelling with decreased mobility. The floors of their mouth were indurate, swollen and tender on palpation.Two patients have open wound and one patient need tracheostomy .We did skin grafting of two patients .

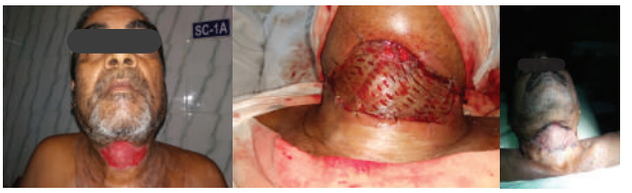
Discussion:

The condition we know as Ludwig's angina was mentioned in writing dating back to Hippocrates and Galen[1,3,7]. ln 1836 German surgeon Wilhelm von Ludwig provided the first detailed description of the disease[1,5,7]. Ludwig characterized the condition as the occurrence of a certain type of inflammation of the throat, which despite the most skillful treatment, is almost always fatal1, [6]. The classic description of Ludwig's angina is an inflammation of the cellular tissues that begin around the submandibular gland and subsequently involves the floor of mouth and neck. Patients who recover do well gradually. Those whose course progressively worsens usually die in 10 to 12 days. The mortality rate reported by Ludwig was approximately sixty percent[4.5]. The mechanics of death was originally attributed to sepsis, but by the 1900s it had become evident that death occurred because of airway obstruction, as pressure on the airway resulted in asphyxia[8, 10]. Another factor that has been implicated in death is the impairment of the medullary respiratory center by apnoea or hypersensitivity of the carotid sinus pressure receptors. The high morlality rate of this disease persisted even after the advent of surgical decompression as a treatment, because either the procedure was under taken too late or the drainage of the infection was inadequate[11]. lt was not until the antibiotic era and the more wide Spread practice of good oral hygiene that the mortality rate dropped to less than ten per cent[12].

In 1982, patterson et al reported no deaths or complication in serials of 20 patients [3,26]. In our series there was no death. Only two patients had abscess over chin which burst before reported to us and having scar overthe chin (Figure-1). A thorough understanding of the anatomy of the spaces of the deep neck and the fascial planes is a prerequisite for treating the disease process properly. Grodnisky and Holyoke in 1939, , described that, the submandibular space in a potential space above the hyoid bone[14,15,27]. The submandibular space is made up of both the sublingual spaces, which lies superior to the mylohyod muscle and the submandibular gland, these spaces can be considered as one single unit because the free border of the mylohyod muscle posteriorly allows them to communicate. The superficial layer of the deep cervical fascia acts as a barrier to the spread of infection. Along with the mandible and the hyoid bone the fascia limits the amount of edema that can occur. Any significant swelling that arises in the submandibular space will cause a superior and posterior displacement of the floor of the mouth and the tongue.

The superficial layer of the deep cervical fascia also envelops the submandibular gland. This layer first contains any infection or swelling that occurs in this gland. However, any prolonged swelling and inflammation can weaken the fascia and allow the infection to rapidly spread into the submandibular space. Our current understanding of Ludwig's angina is that it is a potentially lethal, rapidly spreading cellulitis of the sublingual and submandilbular space. The clinical features of this inflammation include swelling under the tongue, a wood like swelling of the neck and difficulty to speech, deglutition and occasionally respiration. Grodinsky developed strict criteria forth diagnosis of Ludwig's angina[14,15]. He said the disease could be recognized by five identifying characteristics:

1) the infection is a cellulitis of the submandibu l a r space, not an abscess,
2) it never involves only one space, and it is usually bilateral,
3) the cellulitis causes gangrene with serosanguineous infiltration and very litile or more frank pus,
4) the cellulitis attacks the connective tissue, fascia, and muscles, but not the glandular structures, and
5) the cellulitis is spread by continuity, not by the lymphatic.



An odontogenic disorder is the most common etiology, accounting for approximately 70% of cases28, 29. Tschiassny described how the roots of the second and third lower molars penetrate the thin inner cortex of the mandible and extend inferiorly to the insertion of the mylohyoid muscle[1]. A periapical abscess can result in an infection of the submandibular space. Mandibular trauma, penetrating injuries of the floor of mouth, oral neoplasm, and lymphangiomas have all been reported as potential causes of Ludwig's angina. Despite Grodinsky's strict criteria, sporadic cases of submandibular infections have also been recorded in the development of Ludwig's anginala, [14,15]. Bilateral sialadenitis and sialolithiasis in and of itself is a rare entity3o. Because the incidence of Ludwig's angina has steadily declined, fewer physicians are experienced in diagnosing it and in identifying the etiologic agent. Stridor, and cyanosis are the late manifestations of impending airway obstruction[20,29-31]. Airway management should remain the primary therapeutic concern. Management should be tailored to each patient and to the experience of the treating physician. Some patients can be managed adequately with intravenous antibiotic therapy and observation in a monitored care setting. ln others (e.g. those with a more tenuous airway and those scheduled for surgery), the airway must be secured. Routine orotracheal iniubation is usually not feasible in view of the edema and swelling that this disease causes[31]. Fiber optic nasotracheal intubation is an acceptable method, but it requires as experienced

anesthesiologist. Tracheostomy, which has Iong been considered the gold standard, might be necessary in a severely compromised patient[32,33]. Aggressive antibiotic therapy and decompression of the submandibular spaces can be instituted once the airway has been deemed secure[34,35].

Aggressive antibiotic therapy and decompression of the submandibular spaces can be instituted once the airway has been deemed secure[34'35]. Steroid therapies do not have any role except in laryngeal oedema34. Adrenalin administered via a nebulizer in adult patients in the treatment of Ludwig's angina was not satisfactory[35]. ln our study we neither prescribe steroid nor nebulize any patient.

When incision and drainage is performed, the incision can be made extra-orally. lf the submandibular gland has been identified as the source of infection, it should he removed. Moreover, Colp has suggested that the removal of the gland will also allow for adequate drainage of the fascial spaces[11]. One must be cognizant that removal of the gland during an infection can lead to an increase in injury to the hypoglossal or facial nerve[24].

After giving incision and drainage and daily dressing all our five patients improved dramatically and two patients need skin grafting and one patients need trachoestomy which was removed after one month. Patients were discharged with advise after seven days. The out come of the treatment of our series was excellent.

Conclusion:

It is concluded that early diagnosis, adequate drainage and proper antibiotic therapy is always required for treatment of Ludwig's angina. Before 1900 Ludwig's angina was a fatal disease. But now-a-days it is not fatal due to early diagnosis, adequate surgery and use of proper antibiotics.

References:

1. Tschiassny K. Ludwig's angina: An anatomic study of the role of the lower molar teeth in its pathogenesis. Arch Otolaryngol 1943; 38: 485-96.

2. Hought RT. Fitzgerald BE, Zallen RD. Ludwig's angina: report of two cases and review of the literature from 1945to January 1979, J Oral Surg 1 980; 38: 849-55.

3. Patterson HC, Kelly JH, Strome M. Ludwig's angina: An update. Laryngoscope 1982; 92: 370-8.

4. Finch RG, Snider GE, Sprinkle RM. Ludwig's angina. JAMA 1980; 243: 1171-3.

5. Weisengreen HH. Ludwig's angina: Historical review and reflections. Ear Nose Throat J 1986; 65:457-61.

6. Tasca RA, Myatt HM, Beckenham EJ. Lymphangioma of the tongue presenting as Ludwig's angina. lnt J Pediatr Otorhinolaryngol 1 999; 51 : 201 -5.

7. Muckleston HW. Angina Ludovici and kindred affections: Historical and clinical study. Ann Otol Rhinol Laryngol 1 928; 37 : 7 11 -35.

8. Hall SF. Ludwig's like angina (pseudo-angina Ludovici). J Otolaryngol 1984; 13:321-4.

9. Burke J. Angina ludovicii: A translation, together with biography of Wilhelm F.V. Ludwig. Bull Hist Med 1939; 7:1115-26.

10. Lindner HH. The anatomy of the fasciae of the face and neck with particular reference to the spread and treatment of intraoral infections (Ludwig's) that have progressed into adjacent fascial spaces. Ann Surg 1986; 204: 705-14.

11. Colp R. The treatment of deep neck infections of the submaxillary triangle. Surg Clin North Am 1933; ,13: 315-8.

12. Toffel M. Harvey SC. Ludwig's angina:Analysis of 46 cases. Surgery 1942; 11: 841 -50.

13. Thomas TT. Ludwig's angina, an anatomic, clinical, and statistical study. Am Surg 1908;47: 161-83.

14. Grodinsky M. Ludwig's anagina: An anatomical and clinical study with review of the literature. Surgery 1939; 5: 678-96.

15. Grodinsky M. Holyoke E. The fasciae and fascial spaces of the head, neck, and adjacent regions. Am J Anat 1 938; 63: 367-407.

16. Lerner DN, Troost T. Submandibular sialadentitis presenting as Ludwig's angina. Ear Nose Throat J 1991;70:807-9.

18. Lutcavage GJ, Schaberg SJ. Bilateral submandibular sialolithiasis and concurrent sialadenitis:Acase report. J Oral Maxillofac Surg 1991 ; 49: 1220-2.
19. Marple BF. Ludwig's angina: A review of current airway management. Arch Otolaryngol Head Neck Surg 1999; 125: 596-9.
20. Quinn FB. Ludwig's angina. Arch Otolaryngol Head Neck Surg 1999; 125: 599.
21. Owens BM, Schuman NJ. Ludwig's angina. Gen Dent 1994;42:84-7 .
22. Owens BM , Schuman NJ . Ludwigs angina : historical perspective . j Tenn Dent Assoc 1993;73:19-21.
23. Le Jenue HB , Ademee RG. A review of odontogenic infections .j La Stare Med Soc 1994;146:239-41.
24. Finch RG , Snider GE , Sprinkle PM .Ludwigs angina . JAMA 1980;243:1171-3.
25. Kurien M, Mathew J , job A, et al . Ludwigs angina Clin Otolaryngol 1997;22:263-5.
26. Har- ELG, Aroesty JM , Shaha A, et al . Changina trends in deep neck abscesses. Oral Surg Med Pathol1994;77:446-50.
27. Moreland LW , Corey J, Mckenzie R. Ludwigs angina : report of case and review of the literature . Arch intern Med 1988;148:461-1.
28. Shockley WW , Ludwigs angina-a review of current airway management .Arch Otolaryngol Head neck Surg 1999;125:603-3.
29. Bradly F , Maripe . Ludwigs angina . review of current airway management .Arch Otolaryngol Head neck Surg 1999;125:596-9.
30. Neff S PW ,Mery AF, Anderson B . Airway management in Ludwigs angina .Anaesth Intensive Care 1999;27;659-61.
31. parhiscar A , Har- EI G .Deep neck abscess : a retrospective review of 210 cases . Ann Otol rhinol Laryngol 2001;110:1051-4.
32. Bush RF , Shaha D. Ludwigs angina – improved treatment . Otolaryngol Head Neck Surg 1997;117:172-5.
33. Freud B. Timon C.Ludwigs angina : a place for Steroid therapy in its management . Oral Health 1992;82:23-5.
34. Macdonnell SPJ , Timmins AC , Watson JD . Adrenalin administered via a nebulizer in adult patients with opper airway obstruction .Anaesthesia 1995;50:35-6.