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Case Report

Ludwig's angina an analysis of cases seen at tertiary care hospital

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ABSTRACT

We diagnosed and treated four cases of Ludwig's angina 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. Incision aid drainage given to all of the patients through the sub mental region immediately after admission under local anaesthesie. The edema and trismus subsided gradually. All the patients required daily dressing. Despite the dressing 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 to ensure the safety of the patients.

Keywords: Ludwig's angina, Scar mark, Rupture of abscess

INTRODUCTION

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 spread.[6] Airway compromise has been recognized as the leading cause of death. Mortality exceeded 50% 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. In 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 sialoadentitis.[14-18] Predisposing factors include: dental carries, recent dental treatment, systemic illness such as diabetes mellitus, malnutrition, alcoholism, compromised immune system such as AIDS, ana organ transplantation and trauma.[19-23] In children, it can occur de novo, without 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 crisis.[26] Edema and induration of the anterior neck, often with cellulitis, may be present in advanced cases.[27,28]

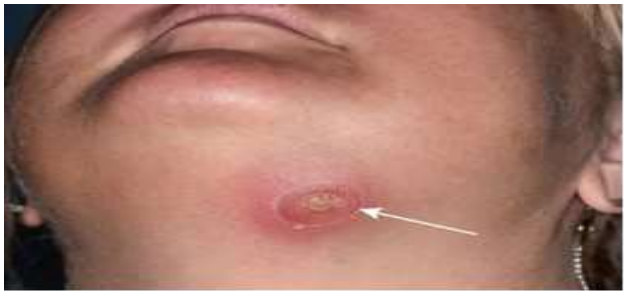

Figure 1: Scar mark over the chin after rupture of abscess due to Ludwig's angina in female patient.

This is a prospective study. Four cases admitted to otolaryngology department of medical college for women and hospital, Uttara, Dhaka with Ludwig's angina was included in the study. Tile study period was from January to June 2020.

CASE REPORT

We studied four patients who admitted into ENT department of Shaheed Tajuddin Ahmed medical college, Gazipur in the last six-month period. The age range of our cases are in between 3 to 50 years. The lowest age limit was 3 years old boy and highest age limit was 50 years. Among 4 patients 3 were male patients and one was female, and male female ratio was 3:1. 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. All the patients have history of dental infection. Their temperature on arrival was 100 and the rest of their vital signs were stable. They did not 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.

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. 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. Their submandibular swelling, trismus disappeared within seven days and patients were released with advice.

DISCUSSION

The condition we know as Ludwig’s angina was mentioned in writing dating back Hippocrates and Galen.[1,3,7] In 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 fatal.[1,6] 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. 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 mortality 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] It was not until the antibiotic era and the wider spread practice of good oral hygiene that the mortality rate dropped to less than ten per cent". In 1982, Patterson et al reported no deaths or complication in serials of 20 patients.[26] In our series there was no death. Only one patient had abscess over chin which burst before reported to us and having scar over the chin.

A thorough understanding of the anatomy of the spaces of the deep neck and the facial 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 mylohyoid 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 tile submandibular space.

Our current of Ludwig's angina is that it is a potentially lethal, rapidly spreading cellulitis of the sublingual and submandibular space. The clinical features of 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 for the diagnosis of Ludwig’s angina.[14,15] He said the disease could be recognized by five identifying characteristics: The infection is a cellulitis of the submandibular space, not an abscess, it never involves only one space, and it is usually bilateral, the cellulitis causes gangrene with serosanguineous infiltration and very little or more frank pus, the cellulitis attacks the connective tissue, fascia, and muscles, but not the glandular structures, and the cellulitis is spread by continuity, not by the lymphatic.

An odontogenic disorder is the most common etiology, accounting for approximately 70% of cases.[28,29] 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'. 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 angina.[14,15] Bilateral sialadenitis and sialolithiasis in and of itself is a rare entity.[30] Because the incidence of Ludwig's angina has steadily declined, fewer physicians are experienced in diagnosing it and in identifying tile 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. In others (e.g., those with a more tenuous airway and those scheduled for surgery), the airway must be secured. Routine orotracheal intubation 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 long been considered gold standard, might be necessary in severely compromised patient.[32,33] Aggressive antibiotic therapy and decompression of submandibular spaces can be instituted once the airway has been deemed secure.[34,35] Steriod therapies do not have any role except in laryngeal oedema.[34] Adrenalin administered via a nebulizer in adult patients in the treatment of Ludwig's angina was not satisfactory.[35] In our study WE neither prescribe steroid nor nebulize any patient.

When incision and drainage is performed, the incision can be made extra-orally. If the submandibular gland has been identified as the source of infection, it should be 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 leads to an increase in injury to the hypoglossal or facial nerve 24. After giving incision and drainage and daily dressing all our patients improved dramatically. Patients were discharged with advice after seven days. The outcome 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 the 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.

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