Ludwig's Angina as an Odontogenic Infection: Management and Characteristics of Fifteen Patients

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Abstract: Ludwig's angina is an acute infection that can be sourced from the teeth and involves submental, sublingual, and submandibular spaces which can rapidly develop into hemodynamic instability and airway obstruction. Objectives: To analyze the management and characteristics of fifteen Ludwig's angina patients in Hasan Sadikin Hospital, Bandung, Indonesia. A retrospective analysis of fifteen patients with Ludwig's angina who had been treated at Hasan Sadikin Hospital, Bandung, Indonesia in 2016. There were 15 cases with 12 males and three females. Odontogenic infections have caused all these cases. Twelve patients were sepsis, and six patients had an underlying disease such as diabetes mellitus and hypertension. Microbiological investigations showed a polymicrobial nature of the infection with Streptococcus viridans in 8 cases. Treatment involved maximum doses of broad-spectrum intravenous antibiotics, immediate surgical drainage under local anesthesia, including extraction of affected teeth as source control. Mortality occurred in two cases. Ludwig's angina is a severe odontogenic infection. Delay in treatment can be life-threatening. Immediate clinical evaluation and definitive treatment will significantly improve its prognosis and reduce the risk of mortality.

Keywords: Ludwig's Angina, odontogenic infection, complications

1. Introduction

Severe odontogenic infections can be the most challenging cases that an oral and maxillofacial surgeon will be called on to treat ^[1]Often the patient with a severe odontogenic infection has significant systemic or immune compromise, and the constant threat of airway obstruction due to infections in the oral and maxillofacial region raises the risk of such cases incalculably. But the incidence, severity, morbidity, and mortality have declined dramatically over the past 60 years. In 1940 Ashbel Williams published a series of 31 cases of Ludwig's angina in which 54% of the subjects died. Only 3 years later, he and Dr. Walter Guralnick published the first prospective case series in the field of head and neck infections, in which the mortality rate of Ludwig's angina was reduced to 10%. This dramatic reduction in mortality from 54% to 10% was not due to the first use of penicillin in the treatment of these infections. Rather, Dr. Guralnick applied the principles of the initial establishment of airway security, followed by early and aggressive surgical drainage of all anatomic spaces affected by cellulitis or abscess. Since then, with the use of antibiotics and advanced medical supportive care, the mortality of Ludwig's angina has been further reduced to 4%.^[2]

Ludwig's angina is an infectious process involving bilateral submandibular, submental, sublingual spaces that can rapidly progress to hemodynamic instability and airway obstruction. Ludwig's angina named after the German physician, Wilhelm Frederick von Ludwig, who described the condition fully in 1836. Since the most cases of Ludwig's angina are of dental origin, bacterial culture may mirror the oral flora. When these infections are not promptly managed, serious, even life-threatening complications can arise, including systemic sepsis, necrotizing fasciitis and descending mediastinitis^{.[3, 4]}

The objectives of this paper are to the analysis of the management and characteristics of Ludwig's angina patients in Hasan Sadikin Hospital, Bandung, Indonesia.

2. Methods

Fifteen patients presenting with Ludwig's angina seen and managed in the Emergency Room, Hasan Sadikin Hospital, Bandung Indonesia in 2016 were reviewed retrospectively.

Relevant data, including gender and age, were collected to accommodate patients' demographics, cause, and duration of infection, clinical features, and diagnosis. The patients have performed the following laboratory investigations such as full blood count, electrolytes and urea, blood gas analysis, microbiology, culture and sensitivity pattern of pus aspirate, soft neck tissue and chest radiograph where indicated. Other information obtained included the application of tracheostomy where needed to maintain the airway, treatment given, outcome, and complications. Data were then tabulated and analyzed using simple frequencies and descriptive statistics.

3. Results

Of the 15 patients evaluated with clinical features consistent with Ludwig's angina, 12 were males (80%) and three females (20%), whose ages ranged from 24 to 71 years (Table 1).

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| Table 1: Patients demographics | | | | |
|--------------------------------|-------------|--------|--|--|
| Patient | Age (years) | Gender | | |
| 1 | 55 | F | | |
| 2 | 35 | М | | |
| 3 | 49 | М | | |
| 4 | 45 | М | | |
| 5 | 41 | М | | |
| 6 | 37 | М | | |
| 7 | 40 | М | | |
| 8 | 29 | М | | |
| 9 | 37 | F | | |
| 10 | 39 | М | | |
| 11 | 29 | М | | |
| 12 | 71 | М | | |
| 13 | 24 | М | | |
| 14 | 60 | М | | |
| 15 | 40 | F | | |

Symptoms and signs of most patients clinically presented swelling at the lower jaw, redness, throbbing pain, limited opening of the mouth, fever, dyspnea, dysphagia, hoarseness, malaise (Fig. 1), and most of the cases were intraorally with elevated tongue (Fig. 2).



Figure 1: Clinical view of patients with Ludwig's angina extraorally



Figure 2: Clinical view of patient with Ludwig's angina intraorally

The etiology of all these cases was the odontogenic infection, which one patient (6.7%) was impacted tooth, and 14 cases (93.3%) the cause was chronic periodontitis from gangrene of tooth, mostly from the molar region while duration ranged from four days to 20 days (Table 2).

All of the patients were performed laboratory diagnostic investigations such as full blood count, electrolytes and urea, blood gas analysis and antimicrobial culture and sensitivity test. The useful laboratory diagnostic tests drew to a constant leukocytosis with values more than 11.500/mm3 to 24.000/mm3. Before antibiotic treatment was administered, all of the patients were performed pus aspiration for antimicrobial culture and sensitivity tests (Fig 3), and the result was revealed Streptococcus viridans in 8 cases (53, 3%), Klebsiella pneumonia, Staphylococcus aureus and Pseudomonas aeruginosa for each were found in 2 cases (13.3%), Staphylococcus sciuri in 1 case (6.7%) (Table 2).



Figure 3: Pus aspiration for antimicrobial culture and sensitivity test

Table 2: Etiological factors, duration of disease at presentation and the result of microbiology culture and

| sensitivity test | | | | | |
|------------------|---------------|--------------------|----------------|--|--|
| Patient | Etiology | Duration (days) | Microbiology | | |
| 1 | Chronic | 10 | Streptococcus | | |
| | periodontitis | 10 | viridans | | |
| 2 | Chronic | 5 | Klebsiella | | |
| | periodontitis | | pneumonia | | |
| 3 | Chronic | 7 | Streptococcus | | |
| 5 | periodontitis | | viridans | | |
| 4 | Impacted | 8 | Staphylococcus | | |
| 4 | tooth | | aureus | | |
| 5 | Chronic | 10 | Staphylococcus | | |
| 5 | periodontitis | 10 | aureus | | |
| 6 | Chronic | 7 | Streptococcus | | |
| 0 | periodontitis | | viridans | | |
| 7 | Chronic | 7 | Pseudomonas | | |
| , | periodontitis | | aeruginosa | | |
| 8 | Chronic | 4 | Streptococcus | | |
| 0 | periodontitis | | viridans | | |
| 9 | Chronic | 7 | Staphylococcus | | |
| | periodontitis | | sciuri | | |
| 10 | Chronic | 6 | Streptococcus | | |
| | periodontitis | 0 | viridans | | |
| 11 | Chronic | 20 | Klebsiella | | |
| 11 | periodontitis | | pneumonia | | |
| 12 | Chronic | 5 | Pseudomonas | | |
| 12 | periodontitis | 5 | aeruginosa | | |
| 13 | Chronic | 7 | Streptococcus | | |
| | periodontitis | | viridans | | |
| 14 | Chronic | 10 | Streptococcus | | |
| | periodontitis | 10 | viridans | | |
| 15 | Chronic | 6 | Streptococcus | | |
| 15 | periodontitis | | viridans | | |

To identify the specific spaces involved and early or potential complications, plain film radiographs of the chest and anteroposterior and lateral neck films were performed.

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All patients were performed chest radiograph (100%), of those patients, 12 (75%) neck soft tissue radiograph was also filmed (Table 3). From the results of the neck,

radiograph showed 1 case was found the retropharyngeal abscess (Fig. 4).

Table 3: Neck soft tissue radiograph and application of tracheostomy

| Radiograph | Patients |
|----------------------------|----------|
| Chest | 15 |
| Neck soft tissue | Nil |
| Chest and Neck soft tissue | 12 |
| No investigation | Nil |

Table 4: Underlying systemic illness, treatment given, complications and outcome patients

| Patient | Underlying illness | Treatment | Complications / Outcome |
|---------|----------------------------------|--|---|
| 1 | Hypertension | Incision drainage, antibiotics & source control | Sepsis, renal failure/ Good |
| 2 | Diabetes mellitus | Incision drainage, antibiotics & source control | Sepsis/ Good |
| 3 | Nil | Incision drainage, antibiotics & source control | Sepsis/ Good |
| 4 | Nil | Incision drainage & antibiotics | Nil/ Good |
| 5 | Diabetes mellitus | Tracheostomy, incision drainage, antibiotics & source control | Sepsis/ Good |
| 6 | Nil | Incision drainage, antibiotics & source control | Nil/ Good |
| 7 | Diabetes mellitus | Incision drainage, antibiotics & source control | Sepsis, necrotizing fasciitis/ Good |
| 8 | Nil | Tracheostomy, incision drainage, antibiotics & source control | Sepsis/ Good |
| 9 | Nil | Incision drainage, antibiotics & source control | Sepsis/ Good |
| 10 | Nil | Incision drainage, antibiotics & source control | Sepsis/ Good |
| 11 | Diabetes mellitus | Tracheostomy, incision drainage, antibiotics & source control | Sepsis, renal failure/ Died |
| 12 | Diabetes mellitus & hypertension | Incision drainage, antibiotics & source control | Sepsis, MODS, Respiratory failure/ Died |
| 13 | Nil | Incision drainage, antibiotics & source control | Nil/ Good |
| 14 | Nil | Incision drainage, antibiotics & source control | Sepsis/ Good |
| 15 | Nil | Incision drainage & antibiotics | Sepsis/ Good |



Figure 4: Anteroposterior and lateral neck radiograph showing aircolumn shifting to left side

The clinical evidence of an underlying systemic illness showed that one patient had hypertension and coronary heart disease. Four patients (26, 7%) had diabetes mellitus (DM), and of these DM patients, one patient also had hypertension (Table 4).

All patients underwent surgical incision and drainage on bilateral submandibular and submental region with the application of through and through Penrose drain (Fig. 5). About ten patients (66, 7%) were treated with surgical incision drainage, administration of antibiotics, and source control. Two patients (13, 3%) were treated with incision and drainage and administration of antibiotics only. Three patients (20%) were performed tracheostomy as airway management, surgical decompression, antibiotics, and source control as treatment (Table 4).



Figure 5: The treatment of Ludwig's angina consists of tracheostomy for an airway maintenance (where needed),

Volume 8 Issue 7, July 2019 <u>www.ijsr.net</u> Licensed Under Creative Commons Attribution CC BY surgical drainage with through and through penrose drain placement, broad-spectrum parenteral antibiotics and removal of source

Sepsis was the complication recorded in 12 patients (80%). Of those patients with sepsis, two patients were also presented with renal failure, and one patient presented with necrotizing fasciitis (Table 4). The outcome of 13 cases (86.7%) was good after treatment. Patient's recovery was satisfactory after hospitalization. And two patients died due to the complication.

4. Discussion

Ludwig's angina is characterized by a firm swelling with an elevation of the tongue, a relatively spreading cellulitis with no tendency to form abscesses and involvement of bilateral submandibular and sublingual spaces. The floor of the mouth contains the sublingual space and submental space, and there is ready communication across the midline through the opposite side. Due to this anatomy, the infection may readily spread from the site initially involved the most or all of the spaces on the floor of the mouth^[5]

History, physical examination, laboratory tests, and diagnostic imaging yield vital information in diagnosing and managing patients with Ludwig's angina. In physical examination, there is increasing neck rigidity, trismus, odynophagia (painful swallowing) and drooling. The floor of the mouth will become tense and indurated with extensive mucosal swelling. Intense pain is usually present, but fluctuance is unusual. The tongue is pushed superiorly, and its movements become stiff. ^[6]The patient can develop a toxic condition, with high fever, tachycardia, and malaise often observed. As the swelling progresses, there is increasing encroachment upon the airway, resulting from elevation of the tongue and extension to the lateral pharyngeal space. It should be noted that progression from the onset of symptoms to respiratory obstruction often occurs within 12-24 hours. Early diagnosis and immediate treatment planning could be a life-saving procedure^[5, 7, 8]

The ability to identify the specific spaces involved and potential early complications has designed contrastenhanced computed tomography (CECT) scanning as a critical component of the diagnostic workup. While plain films have generally limited utility relative to other imaging modalities, they may be useful in the detection of certain types of deep neck space infection. ^[3]All these cases were not CT; only the anteroposterior and lateral radiographs of the neck were done. The anteroposterior and lateral radiographs of the neck can show soft tissue thickening that allows pushing the trachea and narrowing of the airways.

The majority of infections of Ludwig's angina are of odontogenic origin, and in all odontogenic infections, the examination usually reveals the presence of deep caries, periodontal inflammation, or impacted or fractured teeth as the cause. Other causative factors include submandibular gland sialadenitis, compound mandibular fracture, oral soft tissue lacerations, puncture wounds of the oral floor, and secondary infections of oral malignancies.^[3, 4].In these cases, most of the causes are infections of chronic periodontitis. The teeth as etiology are mostly posterior mandibular teeth.

Almost all odontogenic infections are caused by multiple bacteria. Because the mouth flora is a combination of aerobic and anaerobic bacteria, it is not surprising to find that the most odontogenic infections are caused by anaerobic and aerobic bacteria the predominant aerobic bacteria are the streptococcus mileri group, which consists of three-member of the streptococcus viridian's group of bacteria.^[7] The most common organisms are Staphylococcus. Streptococcus, Peptostreptococcus, Fusobacterium, Bacteroides and Actinomyces.^[3]Early infections appearing initially as cellulitis may be characterized as predominantly aerobic streptococcal infections, and late, chronic abscesses may he characterized as anaerobic infections.

Treatment invariably consists of securing the airway where necessary, aggressive broad-spectrum antimicrobial therapy, and surgical decompression of the facial planes with the removal of a source of infection. In odontogenic infection, the causative tooth or teeth are usually removed, although other therapies may occasionally be appropriate. Although some authorities advocate high doses of antibiotics without surgery until fluctuance develops, in most surgeon's experience, fully developed Ludwig's angina requires prompt and deep surgical incision because fluctuance is uncommon and late.^[3]If the patient has signs of laryngeal edema like dyspnea, to save the life of the patient, emergency tracheostomy should be performed promptly.^[6]

Most of the cases of Ludwig's angina occur in healthy patients with no comorbid diseases. Nevertheless, several conditions have been shown to predispose patients to Ludwig's angina. These conditions include diabetes mellitus, alcoholism, acute glomerulonephritis, systemic lupus erythematosus, aplastic anemia, neutropenia, and dermatomyositis. ^[6, 9] Although the widespread involvement seen in Ludwig's usually develops in immunocompromised persons, it can also develop in otherwise healthy individuals.^[10]In some of these cases, diabetes mellitus possibly encourages the severity of the disease and causes death.

Other life-threatening complications of head and neck infection include invasive streptococcal infections, streptococcal or staphylococcal toxic shock, necrotizing fasciitis, descending necrotizing mediastinitis (perhaps the same process as necrotizing fasciitis, occurring in a deeper anatomic plane), internal jugular vein thrombosis, thrombosis. cavernous sinus carotid arterv pseudoaneurysm or rupture, and systemic inflammatory response syndrome.^[3] Renal failure and Respiratory failure are thought to be the cause of death in these cases. The mortality rate for Ludwig's angina has decreased since the advent of prompt surgical intervention, maintenance techniques and antibiotic therapy.^[1, 10] airway

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5. Conclusion

Ludwig's angina is a severe odontogenic infection. Often the patient with Ludwig's angina has significant systemic or immune compromise, and the constant threat of airway obstruction due to infection in the maxillofacial region raises the risk of such cases incalculably. In most cases, emergency care is needed. Delay in treatment can be lifethreatening. The treatment of Ludwig's angina consists of airway maintenance (where needed), surgical drainage, broad-spectrum parenteral antibiotics, and removal of source. Immediate clinical evaluation and definitive treatment will significantly improve its prognosis and reduce the risk of mortality.

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