African Journal of oral Health
Volume 2 Numbers 1 & 2 2005: 16-23

ORIGINAL ARTICLE

Ludwig's Angina: An Analysis of Sixteen Cases in a Suburban Nigerian Tertiary Facility.

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ABSTRACT

Objective: To document the prevalence and management of Ludwig's angina in a suburban population.

Methods: All consecutive cases of Ludwig's angina seen and managed at the Obafemi Awolowo University Teaching Hospital, Ile-Ife, Nigeria between 1988 and 2002.

Results: There were 16 cases with 10 males and 6 females whose ages ranged from 8 to 75 years; mean± SD; 43.8 ± 19.9 years. Symptom duration ranged from one day to three weeks; mean ± SD; 6.4 ± 4.9 days of the onset of illness. Odontogenic infection was the commonest etiologic factor in 12 cases (75%) and 4 of these were attributed to post dental extraction sepsis. Seven patients had underlying disease such as diabetes mellitus, lobar pneumonia, severe anaemia and mental retardation. The commonest was diabetes mellitus in 4 cases. Microbiological investigations showed a polymicrobial nature of the infection with *Staphylococcus aureus (6 cases)* out of 11 patients that had the examination. Treatment involved high doses of broad-spectrum parenteral antibiotics, immediate surgical drainage under local and general anaesthesia in 14 (75%) and single patients respectively. This was in addition to extraction of involved tooth/teeth where applicable. The complications recorded in 5 cases (31.3%) were septicaemia, mediastinitis, empyema thoracic, necrotising fasciitis, laryngeal spasm and renal failure. Mortality occurred in 4 cases (25%).

Conclusions: Ludwig's angina is a serious disease and late presentation remains a typical feature. Associated co-morbid condition is common. Prompt clinical evaluation and definitive care will considerably improve its prognosis and reduce the high mortality associated with the condition

Key words: Ludwig's Angina, Suburban Population, Tertiary Facility.

INTRODUCTION

Ludwig's angina is a potentially life threatening diffuse cellulitis involving the floor of the mouth and submandibular regions bilaterally and causing progressive airway obstruction[1]. The Stuttgart physician, Wilhelm Frederick von Ludwig, first described this condition in 1836 as a nearly always fatal facial space infection.

Typically, Ludwig's angina is characterized by fever, malaise,

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dyspnoea, dysphagia as well as a brawny hard tender swelling of the floor of the mouth and neck[2-4]. In most instances it develops as a complication of an odontogenic infection usually from the second and third molars[5, 6]. However, it may complicate cases of submandibular gland sialadenitis and sialolithiasis, tongue base lymphangioma, and tongue piercing[7-9] .

The microbiology of Ludwig's angina is polymicrobial and includes many gram-positive and negative aerobic/anaerobic organisms, but commonly isolated are *streptococcal spp, staphylococcus aureus, prevotella spp* and *porphyromonas spp*[3, 10].

Treatment invariably consists of securing the airway where necessary, aggressive broad-spectrum antimicrobial therapy, and surgical decompression of the facial planes with removal of source of infection[3, 4, 6, 11].

This paper is an analysis of sixteen cases of Ludwig's angina seen and managed in a suburban Nigerian Tertiary Facility.

PATIENTS AND METHODS

Sixteen consecutive cases of Ludwig's angina seen and managed by the Maxillofacial Unit of the Obafemi Awolowo University Teaching Hospital, Ile-Ife, Nigeria between January 1988 and December 2002 constitute the sample population for this study.

Data were collected on presentation using a questionnaire proforma designed to accommodate patients' demographics, cause and duration of infection, clinical features and diagnosis. In addition, the patients were made to undertake the following laboratory investigations such as full blood count, electrolytes and urea, microbiology, culture and sensitivity pattern of pus aspirate, skull and chest radiographs where indicated. Four cases that were known diabetics had their blood sugar levels regularly monitored in conjunction with physician. Other information obtained included treatment given, outcome and complications.

Data were then tabulated and analyzed using simple frequencies and descriptive statistics.

RESULTS

Over the study period, 16 patients presented with clinical features consistent with Ludwig's angina. There were 10 males (62.5%) and 6 females (37.5%) whose ages ranged from 8 to 75 years (mean$\pm$ SD; 43.8 $\pm$ 19.9 years, median 35 years). Symptoms and signs duration ranged from one day to 3 weeks; mean $\pm$ SD; 6.4 $\pm$ 4.9 days (Table1) with associated progressive difficulty in breathing in most cases.

Table 1: Patients' demographics, etiological factors and duration of disease at presentation.

Patient	Age (years)	Gender	Aetiology	Duration (days)
1	60	Female	Chronic periodontitis	4
2	27	male	unknown	5
3	30	female	chronic periodontitis	6
4	27	female	unknown	1
5	65	male	post extraction sepsis	7
6	25	female	post extraction sepsis	7
7	35	male	trauma	4
8	22	male	apical periodontitis	3
9	55	male	apical periodontitis	5
10	8	male	trauma	5
11	60	female	apical periodontitis	14
12	68	male	chronic periodontitis	21
13	75	male	post extraction sepsis	4
14	35	male	pericoronitis	7
15	58	male	post extraction sepsis	2
16	50	female	chronic periodontitis	7

Table 2: Underlying systemic illness, treatment regime, complications and outcome in 16 cases of Ludwig's angina.

Patient	Underlying illness	Treatment	Complications	Outcome
1	Nil	Surgical decompression/antibiotics	Nil	Alive
2	Nil	Surgical decompression/antibiotics	Nil	Alive
3	Nil	Surgical decompression/antibiotics	Nil	Alive
4	Severe anaemia in pregnancy	Surgical decompression/antibiotics	Nil	Alive
5	Lobar pneumonia	Surgical decompression/antibiotics	Nil	Alive
6	Mentally retarded	Surgical decompression/antibiotics	Nil	Alive
7	Diabetes mellitus	Surgical decompression/antibiotics	Nil	Alive
8	Nil	Surgical decompression/antibiotics	Nil	Alive
9	Diabetes mellitus & hypertension	Surgical decompression/antibiotics	Septicaemia, mediastinitis	Died
10	Nil	Surgical decompression/antibiotics	Nil	Alive
11	Nil	Surgical decompression/antibiotics	Nil	Alive
12	Nil	Nil	Laryngeal spasm, asphyxia, cardiac arrest	Died
13	Diabetes mellitus	Surgical decompression/antibiotics	Mediastinitis, empyema thoracic & renal failure	Died
14	Nil	Surgical decompression/antibiotics	Necrotising fasciitis	Alive
15	Diabetes mellitus	Surgical decompression/antibiotics Surgical decompression/antibiotics	Nil	Alive
16	Nil		Laryngeal spasm, asphyxia, cardiac arrest	Died

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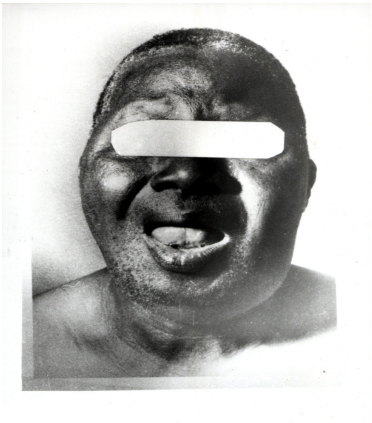
Odontogenic infection was the commonest aetiologic factor observed in 12 cases (75%), trauma was responsible for 2 (12.5%) while in the remaining 2 patients (12.5%) the cause could not be determined. Of those with odontogenic origin, 4 (25%) were due to post dental extraction sepsis. At presentation, 14 patients (87.5%) were pale, dehydrated

Fig. 1: Shows front view of patient 15 at presentation with "croaking toad" appearance.



and toxic looking with bilateral tender and brawny hard swelling involving the submandibular, submental, and sublingual spaces. **Figure 1** shows a typical case of Ludwig's angina. 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. (Table 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 10^7/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)*, α *haemolytic streptococcus (3 cases)*, *Klebsiella pneumonia (2 cases) and one each of Pseudomonas aeruginosa, Proteus mirabilis, Echerichia coli, Prevotelaa intermedia* and *Citrobacter freundi*. In three patients the culture yielded "no growth", while anaerobic culture was carried out in only one case which yielded *Citrobacter freundi* and *Prevotela denticola*. The facility for routine culture of anaerobes is not available in our centre.

Fifteen patients (93.8%) were subjected to surgical decompression using interrupted submandibular and submental skin incisions, followed by blunt dissection of the fascial planes and subsequent insertion of corrugated rubber drains. These remained in place from between twenty four to seventy two hours when there was virtually no further discharge from the incision sites. The drainage was to relief the airway and this proved quite satisfactory for the 13 patients who presented with airway compromise. Fourteen cases were carried out under local (infiltration) anaesthesia, while general anaesthesia with orotracheal intubation was used in one patient. With regards to the patients managed under local anaesthesia, airway access was maintained using a Guedel's tube while oxygen was delivered through a facemask. None of the patients in this series had tracheostomy. Nine patients had removal of the offending tooth/teeth and the incision sites were

closed following complete resolution of the infective process

Parenteral antibiotics, analgesics and multivitamin supplements were administered in all cases and the antimicrobial regimen was a combination of benzyl penicillin, gentamycin and metronidazole. Where appropriate, the antibiotics were modified as dictated by the results of sensitivity tests. The seven cases with associated systemic disease were managed in conjunction with the physicians and obstetrician.

Septicaemia, mediastinitis, empyema thoracic, necrotising fasciitis, laryngeal spasm and renal failure were the complications recorded in 5 cases. These accounted for the death of 4 patients. Of the dead, 2 had uncontrolled diabetes mellitus in addition to Septicaemia, mediastinitis and renal failure while the third case expired at presentation from respiratory obstruction due to laryngeal spasm during clinical examination in the emergency room. The fourth patient also died from laryngeal spasm during extubation under general anaesthesia.

DISCUSSION

Ludwig's angina is a rare condition and this has been ascribed to the introduction of antibiotics several years ago[5] and improved standards in dental practice[4]. This may also be responsible for the number seen during the period of the study. There is no consensus of opinion from previous investigations on the demographic pattern of Ludwig's angina probably because of its uncommon occurrence and the fact that majority of studies were case reports[7, 10, 12, 13]. However our findings are consistent with earlier reports showing male adults preponderance with sporadic incidents in children[3, 4, 7, 14]. Most reports have been silent on the duration of symptoms before presentation at the hospital. It is expected that patients will present early because Ludwig's angina is a rapidly progressive disease. However, we observed that over eighty percent of the patients in this series presented after the third day of the onset of their illness. This delay in presentation could be ascribed to distance from the rural referring centers, self-medication and abuse of antibiotics, ignorance and patronage of unorthodox medical practitioners.

Ludwig's angina can arise from various sources such as odontogenic infection[5, 6], or complicated cases of submandibular gland sialadenitis and sialolithiasis, tongue base lymphangioma, and tongue piercing[7-9, 13], but several studies support our finding that there is usually a dental focus of infection[5, 6, 12, 16]. Details of the pathway of spread have been well documented[4, 17]. In previous reports[14, 18, 19] the role of underlying disease, particularly diabetes mellitus in the aetiopathogenesis of severe orofacial infections was highlighted. Despite the fact that underlying systemic disease was elicited in some of our cases (7/16), the literature is inconclusive as to whether systemic illness predisposes to Ludwig's angina. While sickle cell anaemia, chronic alcoholism and pulmonary infection were reported by Odusanya[2] and Hought et al[17], none of the patients reported by other workers[4, 10, 12] had associated systemic illness. However, what appears certain is that such patients are immuno-compromised and should be thoroughly evaluated and monitored, as they are readily susceptible to life-threatening complications. The role of microbiology, culture and sensitivity tests in the management of orofacial infections

cannot be overemphasized. Overall, the isolates in the present study are consistent with findings in previous reports[4, 13, 18, 19]. However, inability to culture for anaerobes makes our investigation incomplete and may be responsible for the negative routine culture.

The treatment of Ludwig's angina consists of airway maintenance, surgical drainage and broad-spectrum parenteral antibiotics[4, 7, 10, 13]. When there is airway compromise, airway management is probably the most important aspect of immediate care and should not be delayed[6, 13, 20]. Miller et al[21] stressed the importance of airway management and recommended early contrast-enhanced computerized tomographic (CECT) imaging, as clinical examination alone has a sensitivity of 55%. Neff et al[20] recommended awake-fibreoptic intubation to secure the airway if computerized tomographic (CT) scan showed significant airway deviation or narrowing. In the face of limited anaesthetic manpower and equipment, we relied on insertion of the Guedel's airway and facemask for access/maintenance of the airways and supply of oxygen respectively. Prompt incision and drainage undertaken in all the cases available for treatment facilitated decompression of the facial spaces and also provided immediate and progressive relief to the airway. In contrast with what obtained in other reports[22-24], none of our cases had tracheostomy.

The empirical antibiotic regimen in this series was combination chemotherapy involving benzyl penicillin, gentamycin and metronidazole and this was predicated on past clinical experience[4, 18, 19] and knowledge of the usual prevailing organisms. However, the drugs were appropriately modified depending on the outcome of sensitivity tests.

Life threatening complications such as respiratory obstruction, mediastinitis, pleural empyema, pericarditis, pericardial tamponade are often associated with Ludwig's angina[6, 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, 16, 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 economic constraints with inability to procure more effective prescribed antibiotics.

Our findings suggest that inspite of the severity of this condition, late presentation remains a typical feature amongst the suburban populace but prompt and thorough clinical evaluation and definitive care will considerably improve the prognosis. While the need to secure the airway remains paramount, local anaesthesia could be used safely and it's quite effective.

Acknowledgement: The authors wish to express their appreciation to Mr. Joe Oyebanji of the Medical Illustrations Unit for his assistance with the photographic illustrations.

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