

Predisposing Factors, Clinical Features, Culture Sensitivity Findings in Dentoalveolar Abscess versus Ludwigs Angina: An Original Research

Dr. Asma Ahmed¹, Dr. M. Rajmohan², Dr. Anjana Mohan Kumar³, Dr. Sunil Kumar Gulia⁴, Dr. Yash Pal Singh⁵, Dr. Monika Saini⁶, Dr. Rahul VC Tiwari⁷

¹PG 3rd year Dept of ENT & HEAD & NECK Surgery, Government medical hospital, Jammu. asmaahmed074@gmail.com;

²M.D.S., Associate Professor, Department of Dental Surgery, KAPV Government Medical College and Hospital, Trichy, Tamilnadu. omfsraj@gmail.com;

³Reader, Department of Oral Medicine and Radiology, Al Azhar Dental College, Thodupuzha, Kerala;

⁴Senior Lecturer, Oral and maxillofacial Surgery, SGT University, Gurugram, Badli, Jhajjar, Haryana. djgulia10@gmail.com;

⁵BDS, MDS, PhD, Assistant Professor, Department of Restorative and Prosthetic dental Sciences, College of Dentistry, Dar Al Uloom university, Riyadh, KSA. dryashpal.singh@gmail.com;

⁶BDS MDS PhD, Assistant Professor, Department of Restorative and Prosthetic Dental Sciences, College of Dentistry, Dar Al Uloomuniversity, Riyadh. drmonika.singh2009@gmail.com;

⁷OMFS, FOGS, PhD Scholar, Dept of OMFS, Narsinbhai Patel Dental College and Hospital, Sankalchand Patel University, Visnagar, Gujarat,

Corresponding Author:

Dr Asma Ahmed, PG3rd year Dept of ENT & HEAD & NECK Surgery, Government medical hospital, Jammu. asmaahmed074@gmail.com

ABSTRACT

Aim: Purpose of our research was to compare predisposing factors, clinical manifestations as well as culture sensitivity between cases of dentoalveolar abscess and ludwig's angina.

Methodology: A retrospective study was conducted of all cases of Ludwig's Angina (LA) and Dentoalveolar abscess that was seen in our institution for a period of 5 years (2015-2020). 31 cases of dentoalveolar cases and 13 cases of Ludwig's angina were considered in the present study. Information retrieved from the patients' case files included the demographics, aetiology, signs and symptoms at presentation and possible predisposing factors. Laboratory investigations that were done including culture and sensitivity of all aspirates obtained. Descriptive statistical analysis was conducted to initiate the comparison between both the diseases.

Results: Dentoalveolar abscess was common in mandibular molar region with the predominant isolates like Streptococcus species 14.2% of all isolates other than Streptococcus pneumoniae and Enterococcus species. On culture, in case of Ludwig's angina, it was found that streptococcus species, in 83.5% specimens especially streptococcus viridans along with actinomyces species were present in 11% cases of Ludwig's angina.

Conclusion: It is imperative to be equipped with the knowledge related to these two spreading odontogenic infections which spreads to fascial spaces, so that proper and early management can be carried out by dental surgeons.

Keywords Dentoalveolar abscess, Ludwig's angina, polymicrobial.

INTRODUCTION

Most of the oral conditions are mainly inflammatory associated with pain due to infection originating from dental pulp. This requires operative intervention, rather than antibiotics.¹Inappropriate use of antibiotics is said to develop bacterial resistance that is becoming a major issue. The problem of resistance development in recent years might be due to dentist practice toward the prescription of broad-spectrum regimen instead into selective antibiotics. Even though the orofacial infections can be effectively managed through operative intervention and oral hygiene measures, antibiotic prescription practices for the treatment of several orofacial infections. Unnecessary and excessive use of antibiotics may lead to adverse effects such as gastrointestinal disorder, fatal anaphylactic shock, and other severe complications. Dentoalveolar abscess is an abscess around the root of a tooth in the alveolar cavity. It is usually the result of necrosis and infection of dental pulp following dental caries.²A formation and accumulation of pus in a tooth socket or the jawbone form around the base of a tooth. The pus results from a bacterial infection that is usually secondary to an infection or injury to the tooth or alveolar tissues. It is polymicrobial, with an average of 4–6 different causative organisms. It is also called periapical abscess. Abscess is a local collection of pus. It is composed of dead cells -leukocytes and bacteria. It is high in protein, often whitish-yellow, brownish, and occasionally greenish (pyocyanin).³Dental caries, poor dental hygiene, dental trauma are the common predisposing risk factors leading to dentoalveolar abscess. The etiology of dentoalveolar abscess is often polymicrobial with facultative anaerobes such as the viridans group streptococci and the *Streptococcus anginosus* group being the predominant pathogens. Strict anaerobes, especially anaerobic cocci, Prevotella and Fusobacterium species can also additionally contribute to the severity of the abscess. Fever, pains, headache, malaise, weight loss, facial asymmetry if any of the facial spaces is involved along with Palpable submandibular nodes, submental lymph nodes of the affected side. Mouth opening may also be limited. Intraorally, the gingiva surrounding the involved primary tooth will be hyperemic, tender, swollen, and a discharging sinus may also be present. The swelling is around the attached gingiva of the involved primary tooth as a result of the numerous accessory canals which open up in the furcation area. The pus will erode the alveolar bone around the furcation area and then form a swelling. This is often referred to as “gum boil” or inappropriately called periodontal abscess because it resembles the periodontal abscess seen in the permanent tooth. For the permanent tooth, the abscess is toward the apex of the root. The infection usually involves the whole pulp tissue and then causes swelling around the apex of the tooth. This is because permanent teeth have less number of accessory canals when compared to the primary teeth. Typical findings of dental abscess history are localized, constant, deep, throbbing pain, and pain worsens with mastication or exposure to extreme temperatures, tooth may be mobile gingival or facial swelling and tenderness (or both) may be present.⁴Ludwig’s angina otherwise known as Angina Ludovici is a potentially life-threatening diffuse cellulitis of the neck, the floor of the mouth, and submandibular regions bilaterally leading to airway obstruction.⁵ Patients have clinical features of preceding dental infection usually from the second and third molars. It may also complicate cases of submandibular gland sialadenitis and sialolithiasis,⁶ peritonsillar or parapharyngeal abscess,⁷ and cultural practices such as tongue piercing.^{8,9} Subsequent swelling can displace the tongue superiorly and posteriorly leading to potential airway obstruction and asphyxiation. Other clinical features include trismus, odynophagia, and dysphagia. The classical signs are brawny hard, tender induration of the submandibular space bilaterally with elevation of the tongue. The most commonly cultured organisms include Staphylococcus, Streptococcus, and Bacteroides species.^{10,11} However, the microbiology of Ludwig’s angina is polymicrobial with many Gram-positive and -negative aerobic/anaerobic organisms.^{12,13}

AIM OF THE STUDY

Purpose of our research was to compare predisposing factors, clinical manifestations as well as culture sensitivity between cases of dentoalveolar abscess and Ludwig's angina.

METHODOLOGY

A retrospective study was conducted of all cases of Ludwig's Angina (LA) and Dentoalveolar abscess that was seen in our institution for a period of 5 years (2015-2020). 31 cases of dentoalveolar cases and 13 cases of Ludwig's angina were considered in the present study. Information retrieved from the patients' case files included the demographics, aetiology, signs and symptoms at presentation and possible predisposing factors. Laboratory investigations that were done including culture and sensitivity of all aspirates obtained. In addition, predisposing factors, complications and treatment outcome were also noted.

Data analysis was done using SPSS version 22.0 statistical software package (SPSS Inc., Chicago, IL, USA) to present descriptive statistics and frequency charts.

RESULTS

Mostly dentoalveolar abscess was noted in mandibular 1st and 2nd molar region followed by maxillary molar region. The teeth involved were grossly carious and showing a clear periapical abscess beneath the roots of involved teeth along with erosion of surrounding bone as well in 23 cases out of 31 cases. (Table 1) All dentoalveolar abscesses investigated yielded microorganisms. Only one abscess yielded pure growth of *Staphylococcus aureus*. Total of 70 isolates were recovered. Twenty-eight (40%) were facultative organisms, and 42 (60%) were anaerobes. Among the facultative organisms, the predominant isolates were *Streptococcus* species 14.2% of all isolates other than *Streptococcus pneumoniae* and *Enterococcus* species. Among the anaerobes the predominant isolates were gram negative rods. The predominant gram-negative anaerobic rods recovered were *Prevotella* species. (Table 2) A total of thirteen cases diagnosed with LA were retrieved within the study period. There were 7 males and 6 females. (M; F ratio 1.16:1) with a mean age of 47.7 ± 16.8 years (range of 24- 80 years). All patients presented with trismus, pain, toothache, poor oral hygiene, halitosis, swelling involving the submandibular and sublingual spaces with a raised floor of mouth and displaced tongue. Fever was present in eleven patients with temperatures greater than 38 degrees Celsius, whilst two patients did not present with fever. Nine patients presented with respiratory distress out of which four had emergency tracheostomy done. 11 patients underwent surgical treatment which consisted of an extraction of the offending teeth and incision and drainage of abscesses where necessary as well as decompression for cellulitis with insertion of a passive rubber drain. Nine (69.2%) patients underwent their surgical treatment under local anesthesia. Three (23.1%) patients underwent surgical treatment under general anesthesia. These cases were complicated by airway obstruction requiring emergency tracheostomy. On culture, it was found that streptococcus species, in 83.5% specimens especially *Streptococcus viridans* along with *actinomyces* species in 11% cases of Ludwig's angina.

Table 1- Presenting signs and symptoms of Ludwig's Angina and Dentoalveolar abscess and severity of occurrence

Signs and symptoms	Dento- alveolar abscess	Ludwig's angina
Trismus	+	+++
Fever	+	+++
Pain	+++	++
Swelling	+/-	+++
Halitosis	++	++

Toothache	+++	+
Respiratory Distress	-	++
Intraoral Pus Discharge in relation to Swelling	+++	+
Poor oral Hygiene	++	+++
Raised tongue	-	++

Table 2- Organisms isolated from cases of dentoalveolar abscesses and Ludwig's Angina

Organisms isolated	Dento- alveolar abscess	Ludwig's angina
alpha-Hemolytic streptococci	14.2%	83.5%
Streptococcus constellatus	6.6%	-
Staphylococcus epidermidis	23%	2.3%
Staphylococcus aureus	34%	6%
Neisseria species	1.1%	1%
Corynebacterium species	-	1%
Eikenella corrodens	0.25%	0.3%
Capnocytophaga species	11%	0.1%
Peptostreptococcus	2%	3%
Porphyromonas gingivalis	2%	-
Fusobacterium nucleatum	0.1%	0.02%

DISCUSSION

The pathway of spread from the odontogenic origin to the submandibular, sublingual, and submental spaces has been well documented in the literature.¹⁴ Dentoalveolar abscesses (i.e., periapical, apical, or endodontal abscesses) generally originate from infected, necrotic dental pulp via the root canal. The presence of a dentoalveolar abscess is often associated with relatively rapid destruction of the alveolar bone that supports the tooth. The amount and route of spread of the infection depend upon the location of the affected tooth as well as the virulence of the causative organisms. Dentoalveolar abscesses are significant for their potential to spread to the sinuses and other spaces of the head and neck. Therapy for dentoalveolar abscesses generally consists only of root canal therapy or extraction of the affected tooth, but incision and drainage and/or antimicrobial therapy is also occasionally necessary. Historically, viridans streptococci have been thought to be the predominant cause of dentoalveolar abscesses.¹⁵ However, more-recent studies have shown that dentoalveolar abscesses are usually polymicrobial infections with a high proportion of strictly anaerobic isolates.¹⁶ Ludwig's angina has been reported as a rare clinical condition and mortality in the preantibiotic era was 50%. However, with the advent of current therapies, mortality has reduced to <5%.¹⁷ One will expect that patients would report to health facilities early because Ludwig's angina is a rapidly progressing disease with fatal consequences. Reasons for this delay in presentation might be attributed to the user fee-based health system with few on health insurance and long distance from the rural referring centers. Similarly, self-medication and abuse of antibiotics, ignorance, and patronage of unorthodox medical practitioners are other reasons. It is, therefore, imperative that measures should be taken to prevent dental caries and its sequelae by incorporating oral health into existing primary health centers. Furthermore, expanding existing oral health services and manpower in the rural and urban populace is imperative.¹⁸ Ludwig's Angina tends to affect a wide age range. The most affected age range in our study was the 4th and 7th decade of life (23.07%). This is similar with findings by Huang and colleagues in their study who reported 52.4% and 34.1% of

patients being in the 5th and 7th decade respectively. Huang *et al.* attributed it to an increase in the population of the elderly due to the progress of medical care in their environment which is a developed one.¹⁹The finding of anaerobic bacteria in dentoalveolar abscesses is of importance because of the association of anaerobes with many serious infections of odontogenic origin such as bacteremia, endocarditis, sinusitis, meningitis, subdural empyema, brain abscess, and pulmonary empyema. The results of the present study support those of previous studies, which showed that dentoalveolar abscesses are polymicrobial infections in which obligate anaerobes play an important role. The ratio of anaerobes to other isolates has been ~75% in 10 studies in which 289 dentoalveolar abscesses were investigated. *Prevotella* or *Porphyromonas* species were the most common isolates (26%); the second most frequently isolated genus was *Peptostreptococcus* (18%), and the third most common bacterial group recovered were the alpha-hemolytic streptococci (13.9%).^{20,21} For treatment oral antibiotics are prescribed for dentoalveolar abscess and in serious cases IV antibiotics. Ludwig's angina is treated with the help of iv antibiotics, tracheostomy. But judicious use of antibiotics is necessary as the lesions become resistant to common antibiotics, which can be based upon culture of organisms from lesional aspirates.

CONCLUSION

It is imperative to be equipped with the knowledge related to these two spreading odontogenic infections which spreads to fascial spaces, so that proper and early management can be carried out by dental surgeons.

REFERENCES

1. Dar-Odeh NS, Abu-Hammad OA, Al-Omiri MK, Khraisat AS, Shehabi AA. Antibiotic prescribing practices by dentists: A review. *Ther Clin Risk Manage* 2010;6:301.
2. Longman LP, Preston AJ, Martin MV, Wilson NH. Endodontics in the adult patient: The role of antibiotics. *J Dent* 2000;28:539-48.
3. Öcek Z, Sahin H, Baksi G, Apaydin S. Development of a rational antibiotic usage course for dentists. *Eur J Dent Educ* 2008;12:41-7.
4. Dhanvanth M et al. Choice of antibiotics in the management of dentoalveolar abscess among dental practitioners. *Drug Invention Today* 2018;10(12):2390-2394.
5. Murphy SC. The person behind the eponym: Wilhelm Frederick von Ludwig (1790-1865). *J Oral Pathol Med* 1996;25:513-5.
6. Wasson J, Hopkins C, Bowdler D. Did Ludwig's angina kill Ludwig? *J Laryngol Otol* 2006;120:363-5.
7. Barakate MS, Jensen MJ, Hemli JM, Graham AR. Ludwig's angina: Report of a case and review of management issues. *Ann Otol Rhinol Laryngol* 2001;110 (5 Pt 1):453-6.
8. Honrado CP, Lam SM, Karen M. Bilateral submandibular gland infection presenting as Ludwig's angina: First report of a case. *Ear Nose Throat J* 2001;80:217-8, 222-3.
9. Wong TY. A nationwide survey of deaths from oral and maxillofacial infections: The Taiwanese experience. *J Oral Maxillofac Surg* 1999;57:1297-9.
10. Williams AM, Southern SJ. Body piercing: To what depths? An unusual case and review of associated problems. *Plast Reconstr Surg* 2005;115:50e-4e.
11. Perkins CS, Meisner J, Harrison JM. A complication of tongue piercing. *Br Dent J* 1997;182:147-8.
12. Hartmann RW Jr. Ludwig's angina in children. *Am Fam Physician* 1999;60:109-12.
13. Doldo G, Albanese I, Macheda S, Caminiti G. Ludwig angina: A disease of the past century. Case report. *Minerva Anestesiol* 2001;67:811-4.
14. Iwu CO. Ludwig's angina: Report of seven cases and review of current concepts in management. *Br J Oral Maxillofac Surg* 1990;28:189-93.

15. HardielM. Dental and oral infection. In: Duerden BI, Drasar BS, eds. Anaerobes in human disease. London: Edward Arnold, 1991:245-67.
16. Sabiston CB Jr, Grigsby WR, Segerstrom N. Bacterial study of pyogenic infections of dental origin. *Oral Surg Oral Med Oral Pathol* 1976; 41: 430-5.
17. Doldo G, Albanese I, Macheda S, Caminiti G. Ludwig angina: A disease of the past century. Case report. *Minerva Anestesiol* 2001;67:811-4.
18. Ugboko VI, Ndukwe KC, Oginni FO. Ludwig's angina: An analysis of sixteen cases in a suburban Nigerian tertiary facility. *Afr J Oral Health* 2005;2:16-23.
19. Huang TT, Liu TC, Chen PR, *et al.* Deep neck infection: analysis of 185 cases. *Head & neck.* 2004;26(10):854-60.
20. Rams TE, Slots J. Systemic manifestations of oral infections. In: Slots I, Taubman MA, eds. Contemporary oral microbiology and immunology. St. Louis: Mosby Year Book, 1992:500-10.
21. Dahlen G, Moller AJR. Microbiology of endodontic infections. In: Slots J, Taubman MA, eds. Contemporary oral microbiology and immunology. St. Louis: Mosby Year Book, 1992:444-75.