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LUDWIG'S ANGINA: A NARRATIVE REVIEW OF AETIOLOGY, PATHOPHYSIOLOGY, DIAGNOSIS AND CONTEMPORARY MANAGEMENT, WITH IMPLICATIONS FOR DENTAL PRACTICE

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ABSTRACT

Objective: To provide a contemporary narrative review of Ludwig's angina, a rapidly progressive and potentially fatal cellulitis of the floor of the mouth, with emphasis on aetiology, pathophysiology, diagnosis, airway management and treatment, and to highlight implications for dental practitioners.

Methods: The English-language literature was searched in PubMed/MEDLINE, Scopus and Google Scholar. Case series, cohort studies, reviews and clinical reports addressing the epidemiology, microbiology, diagnosis and management of Ludwig's angina and related deep neck space infections were synthesised narratively.

Results: Odontogenic infection, particularly of the mandibular second and third molars, accounts for most cases. Infection spreads bilaterally through the sublingual, submandibular and submental spaces, elevating the floor of the mouth, displacing the tongue and progressively compromising the airway. Infections are usually polymicrobial, with streptococci and oral anaerobes predominating. Diabetes mellitus, immunosuppression and HIV infection are important comorbidities. Securing the airway is the first priority, and awake fiberoptic intubation or an early surgical airway is favoured when obstruction is threatened. Treatment combines empirical broad-spectrum intravenous antibiotics, elimination of the odontogenic source and surgical drainage where collections or clinical deterioration are present. Contemporary mortality is low with early recognition but rises with delayed airway control, mediastinitis or septic shock.

Conclusion: Ludwig's angina remains a dental and medical emergency. Early recognition, immediate airway assessment, prompt referral and control of dental sepsis are essential to prevent morbidity and death.

Keywords: Ludwig's Angina; Deep Neck Space Infection; Odontogenic Infection; Airway Management; Floor Of Mouth Cellulitis; Dental Emergency

1 INTRODUCTION

Ludwig's angina is a rapidly spreading, diffuse cellulitis involving the submandibular, sublingual and submental spaces bilaterally. It takes its name from the German physician Wilhelm Friedrich von Ludwig, who described the condition in

1836 [1,2]. The term “angina” derives from the Latin for strangling, reflecting the characteristic threat to the airway. Before the antibiotic era the condition was often fatal, with mortality in early series approaching 50% [2,3]. Modern reports place mortality considerably lower, generally below about 10%, although it remains substantial when the airway is lost or infection descends into the mediastinum [1,4].

Most cases arise from odontogenic infection, which places dental practitioners at the point where the disease is most likely to be recognised early and, ideally, prevented [5,6]. Dental sepsis remains common in many settings, including in parts of Africa where access to timely dental care is limited and comorbid conditions such as diabetes and HIV infection may modify the course of disease [5]. The condition is uncommon, so many clinicians will encounter it rarely, yet delay in diagnosis or in securing the airway is associated with poor outcomes [4,7].

The research problem addressed here is that available evidence on Ludwig’s angina is dominated by case reports and retrospective series, and that practical guidance is fragmented across dental, otolaryngological, anaesthetic and emergency medicine sources. The objective of this review is to synthesise the current literature on the aetiology, pathophysiology, clinical presentation, diagnosis and management of Ludwig’s angina, and to draw out the implications for prevention and early referral in dental practice.

2 MATERIALS AND METHODS

2.1 Study design

This is a narrative review. It was designed to integrate evidence from heterogeneous sources rather than to pool quantitative data, since the literature on Ludwig’s angina consists mainly of case reports, single-centre retrospective series and expert reviews, and is not suited to formal meta-analysis.

2.2 Search strategy

PubMed/MEDLINE, Scopus and Google Scholar were searched using combinations of the terms “Ludwig’s angina”, “deep neck infection”, “deep neck space infection”, “odontogenic infection”, “floor of mouth cellulitis”, “airway management” and “mediastinitis”. Reference lists of key articles were screened for additional sources. [Author to confirm database search dates before submission.]

2.3 Eligibility and selection

Articles were eligible if they were published in English and addressed the epidemiology, aetiology, microbiology, diagnosis, airway management, medical or surgical treatment, complications or outcomes of Ludwig’s angina or closely related deep neck space infections. Peer-reviewed case series, cohort studies, narrative and evidence-based reviews and clinical reports were included. Purely animal or laboratory studies and articles without accessible full text were excluded. Foundational historical papers were retained where they defined the clinical entity.

2.4 Data synthesis

Information was extracted thematically under the headings of epidemiology, anatomy and pathophysiology, aetiology and risk factors, microbiology, clinical features, investigations, airway management, medical and surgical treatment, complications and prognosis. Findings were compared across studies and synthesised narratively. No formal risk-of-bias assessment was undertaken, and the strength of conclusions is limited accordingly.

2.5 Ethical considerations

This review used published literature only and involved no patients or identifiable data. Ethical approval and informed consent were therefore not required.

3 RESULTS AND DISCUSSION

3.1 Definition and diagnostic criteria

Classical criteria describe Ludwig’s angina as a bilateral infection of the submandibular space, with gangrenous or serosanguinous, putrid infiltration but little frank pus, involving connective tissue, fascia and muscle but sparing glandular structures, and spreading by continuity rather than by the lymphatics [2]. In practice the diagnosis is clinical, and the term is used for a bilateral, rapidly progressive cellulitis of the floor of the mouth with tongue elevation and airway threat [1,8]. The distinction from a localised unilateral abscess is important, because cellulitis of this type is not readily drained and behaves more aggressively.

3.2 Epidemiology

Ludwig's angina accounts for a minority of deep neck space infections. In large series of deep neck infection, the submandibular space is commonly involved and Ludwig's angina represents a small but disproportionately dangerous subset [9,10]. Adults are most often affected, and a male predominance has been reported in several series [5,11]. The incidence has fallen since the introduction of antibiotics and improved dental care, but it has not disappeared, and case series continue to document severe disease in patients with delayed presentation or major comorbidity [5,12].

3.3 Anatomy and pathophysiology

The floor of the mouth is divided by the mylohyoid muscle into the sublingual space above and the submandibular space below, and the submental space lies anteriorly. These spaces communicate around the posterior free border of the mylohyoid, so infection can readily involve all three compartments [13,14]. Inflammatory oedema and induration raise the floor of the mouth and push the tongue upward and backward, narrowing the oropharyngeal airway, and the rigid, woody swelling of the submandibular region limits external access [8,15].

Further spread occurs along fascial planes into the parapharyngeal and retropharyngeal spaces, and from there towards the carotid sheath and the mediastinum. Descending necrotising mediastinitis is among the most feared complications and carries a high mortality [16,17]. Airway compromise arises from a combination of tongue displacement, pharyngeal and laryngeal oedema, trismus and pain, and can progress over hours.

3.4 Aetiology and risk factors

An odontogenic source is identified in the majority of cases, most often infection of the mandibular second or third molars, whose root apices commonly lie below the mylohyoid attachment, allowing periapical or pericoronal infection to perforate the lingual cortex directly into the submandibular space [4,5,15]. Other reported causes include peritonsillar abscess, mandibular fractures, oral or tongue piercings, floor-of-mouth lacerations, sialadenitis, and instrumentation or intubation trauma, though these are much less frequent [1,8].

Host factors influence both susceptibility and severity. Diabetes mellitus, malignancy, alcohol misuse, immunosuppressive therapy and HIV infection are repeatedly associated with more severe or atypical presentations [4–6]. In a South African cohort, comorbid systemic disease was an important feature of patients with Ludwig's angina, underscoring the need for a low threshold for admission and investigation in such patients [5]. Delay in seeking care, antibiotic non-completion and untreated dental disease are recurrent themes in the literature [18,19].

3.5 Microbiology

Odontogenic deep-space infections are typically polymicrobial, involving oral streptococci (including the *Streptococcus anginosus* group), staphylococci and obligate anaerobes such as *Prevotella*, *Fusobacterium* and *Peptostreptococcus* species [18,20]. Cultures from Ludwig's angina series broadly reflect this mixed flora, and anaerobes are frequently under-recovered when samples are not handled appropriately [4,6]. Methicillin-resistant *Staphylococcus aureus* and resistant Gram-negative organisms may be relevant in patients with recent hospitalisation, prior antibiotic exposure or immunocompromise, and should influence empirical selection in such cases [4,19].

3.6 Clinical presentation

Patients typically present with a history of dental pain or recent dental procedure, followed by rapidly increasing swelling beneath the chin and jaw, floor-of-mouth pain, dysphagia and odynophagia. Trismus, drooling, muffled or "hot potato" voice, and difficulty handling secretions suggest impending airway loss [1,8]. Examination shows bilateral, firm, tender submandibular and submental swelling, an elevated and indurated floor of the mouth, and a protruding or displaced tongue. Fever, tachycardia and other markers of systemic inflammatory response are common [4,15].

Stridor, tripod posture, an inability to lie flat, cyanosis and altered consciousness are late and ominous signs of airway obstruction. Because deterioration can be abrupt, the presence of any of these features should prompt immediate escalation rather than further diagnostic work-up [21,22].

3.7 Diagnosis and investigations

The diagnosis is clinical, and investigations must never delay airway management. Laboratory tests typically show leukocytosis and raised inflammatory markers, and blood glucose, renal function and lactate assist risk stratification. Blood cultures should be taken before antibiotics where possible, and pus or tissue obtained at drainage should be sent for aerobic and anaerobic culture [4,23].

Contrast-enhanced computed tomography is the imaging modality of choice for defining the extent of infection, identifying collections and detecting extension to the parapharyngeal, retropharyngeal and mediastinal spaces [23,24]. It should be undertaken only when the patient's airway is secure or the patient is stable enough to lie flat and be monitored during transfer. Plain radiographs, including orthopantomography, can help identify the dental source but add little to the management of the acute emergency.

3.8 Airway management

Airway management is the central determinant of outcome. Where airway compromise is present or anticipated, the airway should be secured early, in a controlled setting, by the most experienced operator available, with a surgical airway team ready [21,22,25]. Distorted anatomy, trismus and tongue elevation make direct laryngoscopy hazardous, and induction of anaesthesia or paralysis without a secured plan can convert a difficult airway into a failed one [22,25]. Table 1 summarises the principal approaches.

Table 1: Airway management options in Ludwig's angina

Approach	Typical indication	Key considerations
Awake fiberoptic nasotracheal intubation	Threatened airway in a cooperative patient with a stable airway	Widely favoured first-line technique; requires expertise, topical anaesthesia and minimal sedation; secretions and bleeding may impair vision [21,25]
Awake video laryngoscopy or other awake techniques	Where fiberoptic equipment or expertise is limited and mouth opening is adequate	Trismus and tongue displacement limit success; prepare for immediate conversion to a surgical airway [7,22]
Surgical airway (tracheostomy or cricothyroidotomy)	Impending obstruction, failed intubation, uncooperative patient or lack of expertise	Local anaesthetic tracheostomy remains the definitive rescue; neck swelling can make landmarks difficult, so early involvement of a surgeon is advised [7,22]
Close observation in a high-dependency setting	Early disease with a patent airway and no progression	Only with continuous monitoring, immediate access to airway expertise and a low threshold for intervention [1,4]

Comparative studies of intubation versus tracheostomy in deep neck infection show that both are used, that the choice depends on local expertise and disease severity, and that tracheostomy is more often required when the airway is already critically compromised [7]. There is no randomised evidence to prescribe a single technique, so decisions should reflect the patient's presentation and the skills available.

3.9 Medical management

Empirical intravenous antibiotics should be started without delay after cultures are obtained, and should cover streptococci, oral anaerobes and, where indicated, *Staphylococcus aureus*. Commonly used regimens include a beta-lactam with a beta-lactamase inhibitor, or a third-generation cephalosporin combined with metronidazole, with clindamycin as an alternative in penicillin allergy; vancomycin or an equivalent agent is added when MRSA is a concern, and therapy is then adjusted to culture results and clinical response [4,18,19]. Local antimicrobial guidelines and resistance patterns should guide the final choice.

Supportive care includes intravenous fluids, analgesia, glycaemic control in diabetic patients and monitoring for sepsis. Corticosteroids have been used by some clinicians to reduce airway oedema, but evidence from controlled studies is lacking, and their use is not a substitute for airway protection [4]. Antibiotics alone rarely suffice when airway compromise is evident, and antibiotic therapy should be considered an adjunct to source control rather than a replacement for it [13,18].

3.10 Surgical and dental management

Removal of the causative tooth or drainage of the odontogenic focus is a key element of treatment and should occur as soon as the airway and physiological condition permit [13,19]. Surgical decompression of the affected spaces is indicated when there is a demonstrable collection, crepitus suggesting gas-forming infection, failure to improve within 24 to 48 hours of appropriate antibiotics, or progression of swelling [4,14]. Some authors describe successful conservative management of carefully selected early cases without external drainage, but this requires close

observation and should be led by a specialist team [4,9]. Involvement of an oral and maxillofacial or head and neck surgeon is therefore advisable from the outset.

3.11 Complications and prognosis

Recognised complications include airway obstruction, aspiration pneumonia, septic shock, jugular vein thrombosis, carotid sheath involvement, descending necrotising mediastinitis, pericarditis, empyema and death [12,16,17]. Mediastinal extension has historically carried mortality of 25% or more and requires multidisciplinary management including thoracic surgical drainage [16,17]. In contrast, contemporary series in which the airway is secured early and source control is achieved report low mortality [1,10,11]. Prognosis is worse in older patients, in those with diabetes or other significant comorbidity, and where presentation is delayed [5,9].

3.12 Implications for dental practice

Dental practitioners have a vital role in preventing Ludwig's angina by managing odontogenic infection promptly and definitively, particularly in the lower molar region. Practitioners should recognise features that indicate spread beyond the alveolus, including floor-of-mouth swelling, tongue elevation, trismus, dysphagia, voice change and systemic toxicity, and should regard them as an indication for urgent hospital referral rather than outpatient antibiotics [18,19]. Patients with diabetes, immunosuppression or HIV infection deserve a lower threshold for referral. Referral letters should convey the urgency, and the patient should be transported in a position that does not worsen airway obstruction, with escort where possible. Patient education on completing antibiotic courses, seeking early care for dental swelling and avoiding delay is likewise important [19,20].

Limitations and future directions

The conclusions of this review are limited by the nature of the evidence base, which is largely retrospective and heterogeneous, by the use of a narrative rather than systematic method without formal quality appraisal, and by the restriction to English-language sources. Prospective multicentre registries, standardised reporting of airway strategy and outcomes, and region-specific data, including from low- and middle-income settings, would improve understanding of the true burden and best practice.

4 CONCLUSION

Ludwig's angina is a bilateral, rapidly progressive cellulitis of the floor of the mouth that arises predominantly from odontogenic infection and threatens life through airway obstruction and descending infection. Outcomes have improved markedly since the pre-antibiotic era, but the disease remains dangerous when recognised late. The available evidence supports early airway assessment and control, broad-spectrum antibiotics, elimination of the dental source and timely surgical drainage in a multidisciplinary setting. For dental practitioners, the key messages are prevention through definitive management of dental sepsis, vigilance for signs of deep space spread, and urgent referral when airway compromise is suspected.

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