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Interprofessional Management of Submandibular Space Infections (Ludwig's Angina): Nursing, Radiology, Pharmacy, Dental, and Emergency Perspectives

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Abstract

Background: Ludwig's angina is a rapidly progressive, bilateral cellulitis of the submandibular, sublingual, and submental spaces, most commonly originating from infections of the mandibular second or third molars. It is characterized by aggressive soft-tissue edema, airway compromise, and continuity of spread along deep cervical fascial planes rather than lymphatic pathways, making rapid recognition and management essential.

Aim: This article aims to provide a comprehensive, interdisciplinary overview of the clinical presentation, anatomical considerations, diffusion mechanisms, pathophysiology, evaluation strategies, and evidence-based management of Ludwig's angina.

Methods: A narrative, multidisciplinary review was conducted using current clinical knowledge from nursing, emergency medicine, dentistry, radiology, anesthesia, and surgery. The review synthesizes anatomical descriptions, pathophysiological progression, diagnostic findings, and treatment modalities.

Results: Findings highlight that Ludwig's angina typically originates from odontogenic infections (over 90% involving second/third molars) and spreads contiguously across fascial planes, often leading to severe airway obstruction. Early airway control—preferably via awake fiberoptic intubation—is critical, while surgical drainage is reserved for cases with abscess formation or failure of medical therapy. Broad-spectrum IV antibiotics targeting aerobic and anaerobic organisms remain essential, along with prompt extraction of the causative tooth. Multidisciplinary coordination significantly reduces mortality from historical rates of nearly 50% to less than 5% in modern practice.

Conclusion: Ludwig's angina remains a medical and surgical emergency requiring rapid recognition, airway protection, early antibiotic therapy, and targeted surgical intervention. A coordinated, interdisciplinary approach is key to improving outcomes and preventing life-threatening complications.

Key Words: Ludwig's angina, submandibular space infection, airway management, odontogenic infection, deep neck spaces, cellulitis, multidisciplinary care.

Introduction

Ludwig's angina is a rapidly progressive cellulitis that involves the submandibular, sublingual, and submental spaces bilaterally, often originating from infections of the mandibular second or third molars [1]. Unlike other deep neck infections, Ludwig's angina typically does not present with abscess formation or lymphadenopathy, which distinguishes it clinically from other odontogenic or cervical infections. The condition is named after Karl Friedrich Wilhelm von Ludwig, who first described the aggressive, gangrenous nature of this infection affecting the floor of the mouth and adjacent neck spaces [2]. The primary concern with Ludwig's angina is airway compromise. Swelling of the soft tissues of the floor of the mouth elevates and displaces the tongue posteriorly, significantly narrowing the oropharyngeal airway. Historically, the mortality rate

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of untreated Ludwig's angina approached 50%, reflecting the severity of airway obstruction and systemic infection [3]. With the advent of broad-spectrum antibiotics, advanced imaging modalities, and timely surgical interventions, mortality has decreased to approximately 8% in modern clinical practice [2,3,4]. Despite this improvement, airway obstruction remains the most critical and potentially fatal complication, requiring prompt recognition and intervention. Clinically, patients often present with a combination of submandibular and floor-of-mouth edema, tooth pain, or a history of recent dental procedures. Dysphonia, dysphagia, and dysarthria are reported in a smaller proportion of patients, while signs of respiratory distress, including dyspnea, tachypnea, and stridor, appear in roughly one-third of cases [5]. Examination typically reveals firm, tender swelling in both submandibular regions, often described as "woody" or brawny, accompanied by erythema of the overlying skin. A high or protruding tongue is noted in over 95% of patients, which serves as a key diagnostic indicator [3,5]. Prompt recognition of these characteristic signs and symptoms is critical for early intervention, as delayed diagnosis can lead to life-threatening airway compromise and systemic infection.

Related Anatomy

A comprehensive understanding of the anatomy of the face and deep neck spaces is essential for diagnosing and managing infections such as Ludwig's angina. The neck is organized by fascial planes that define discrete compartments, which both contain and limit the spread of infections. These planes form potential spaces through which odontogenic or soft tissue infections can rapidly propagate, resulting in serious complications [6,7]. The cervical region consists of superficial and deep fascial layers. The superficial cervical fascia lies directly beneath the dermis and envelopes the muscles of facial expression. It extends from the base of the skull to the upper thorax and shoulders, incorporating the superficial musculoaponeurotic system (SMAS). Beneath this layer are fat, lymphatic channels, and neurovascular bundles. While clinically important for surgical and aesthetic procedures, the superficial fascia does not participate in the containment or spread of deep neck infections [6]. The deep cervical fascia, by contrast, is critical in understanding the pathways of deep neck infections. It consists of three layers: superficial (investing), middle (visceral/muscular), and deep (prevertebral/alar). The investing layer surrounds the neck globally, enclosing structures such as the submandibular and parotid glands, masticatory muscles, and sternocleidomastoid muscle. Its superior boundaries include the mandible, zygomatic arch, mastoid process, nuchal ridge, and hyoid bone, while inferiorly it is limited by the clavicles, sternum, scapula, and acromion. This layer contributes to the formation of the carotid sheath laterally and creates the floor of the submandibular space as it courses from the hyoid to the medial aspect of the mandibular ramus, enclosing the anterior belly of the digastric muscle. It also defines the lateral margins of the parotid and masticator spaces [6].

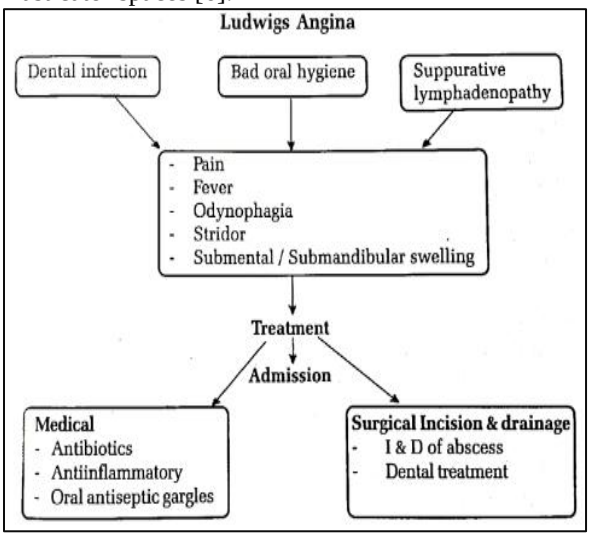

Fig. 1: Ludwigs Angina.

The middle layer of the deep cervical fascia has muscular and visceral components. The muscular portion encases the strap muscles (sternohyoid, sternothyroid, thyrohyoid, omohyoid) and the adventitia of major vessels. The visceral portion forms the buccopharyngeal fascia and anterior wall of the retropharyngeal space, surrounding the pharyngeal and esophageal constrictor muscles. The carotid sheath is a composite structure of these middle layer components, enclosing the carotid artery, internal jugular vein, and vagus nerve. The larynx, trachea, and thyroid gland are also contained within this intermediate layer. The prevertebral and alar layers constitute the deepest fascial structures. The prevertebral layer lies anterior to the vertebral bodies and extends laterally to the transverse processes, providing a supportive framework for the deep neck muscles. The alar fascia, located between the prevertebral and visceral layers, forms the posterior boundary of the retropharyngeal space and contributes to the carotid sheath [6]. The submandibular space, which is directly implicated in Ludwig's angina, is bounded inferiorly by the superficial layer of the deep cervical fascia, laterally by the mandible, and superiorly by the floor of the mouth mucosa. The mylohyoid muscle divides this space into sublingual and submaxillary regions. The sublingual compartment contains the sublingual glands, hypoglossal nerve, and Wharton duct, while the submaxillary region is accessible to infection through the posterior margin of the mylohyoid. The anterior belly of the digastric further partitions the submaxillary region into central submental and lateral submaxillary spaces. Odontogenic infections, trauma, or sialadenitis can introduce pathogens into these

compartments, which then spread rapidly along fascial planes rather than lymphatics [6].

In Ludwig's angina, infection of the submandibular space leads to firm, brawny swelling of the floor of the mouth. This may elevate or displace the tongue, producing airway compromise. The absence of a discrete abscess does not preclude the diagnosis. Common clinical manifestations include drooling, trismus, pain, dysphagia, submandibular enlargement, and dyspnea. Bilateral involvement is typical, and airway management, often via tracheostomy, is paramount due to the high risk of obstruction. Historically, mortality approached 50% before antibiotics; with modern interventions, this has decreased to approximately 5% [6]. Adjacent deep neck spaces, such as the parapharyngeal and retropharyngeal spaces, are vulnerable to secondary infection originating from the submandibular space. Understanding the anatomy and fascial continuity of these spaces is essential for planning surgical drainage, imaging interpretation, and predicting potential complications associated with the spread of Ludwig's angina [6].

Diffusion Mechanisms

Ludwig's angina predominantly originates from odontogenic infections, with over two-thirds of cases linked to the second or third mandibular molars [7,8]. The roots of these teeth lie inferior to the attachment of the mylohyoid muscle, rendering the submandibular space the primary site of infection. Additional etiologies include trauma to the mandible or floor of the mouth, sialolithiasis, infections of the submandibular or sublingual salivary glands, peritonsillar abscesses, and suppurative parotitis, all of which can propagate infection into the submandibular and adjacent deep neck compartments [7,8,9]. The anatomical positioning of the mandibular molars, combined with the thin lingual cortical plates of the alveolar bone, facilitates medial extension of infection into the sublingual and submental spaces, frequently affecting both sides of the neck simultaneously. This bilateral involvement helps distinguish Ludwig's angina from lymphatic spread, which would typically present unilaterally [1]. Less commonly, infections arising from premolars or injuries to the floor of the mouth may propagate similarly into the sublingual region. Once established, infection in Ludwig's angina progresses rapidly due to the continuity of fascial planes in the submandibular region. The floor of the mouth becomes tense and indurated, and tongue edema may result in a striking anterior and superior displacement. The tongue can swell to two to three times its normal volume, occupying the oral cavity, compressing the hypopharynx, and contacting the palate, often producing the clinical appearance described as a "double tongue" [10]. Posterior involvement extends to the epiglottis, which is anatomically contiguous with the oropharyngeal and hypopharyngeal spaces. The buccopharyngeal space serves as a critical conduit for infection between the submandibular and parapharyngeal compartments, posing a significant risk for airway compromise. The styloglossus muscle, originating from the tongue and traversing between the middle and superior constrictor muscles to insert on the styloid process, provides a pathway for cellulitis from the submandibular space into the parapharyngeal space. From there, the infection may advance into the retropharyngeal space and extend inferiorly to the superior mediastinum [11]. This pattern of diffusion underscores the life-threatening potential of Ludwig's angina and highlights the importance of timely recognition and intervention. Rapid clinical progression necessitates vigilant monitoring and early airway management, as the infection exploits the anatomical continuity of the deep cervical fascial spaces to spread extensively within hours to days.

Pathophysiology

Ludwig's angina originates primarily from odontogenic infections, particularly involving the second and third mandibular molars, including partially erupted third molars [12]. The infection usually begins in the subgingival pockets and progresses to the muscles of the floor of the mouth. Because the roots of the second and third molars extend below the mylohyoid line, the infection can easily invade the sublingual space. The thin lingual cortical plate of the mandible provides minimal resistance, favoring medial spread over buccal diffusion. Once the infection breaches the lingual cortex, it infiltrates the sublingual space and subsequently progresses into the submandibular space, which constitutes the hallmark anatomical site for Ludwig's angina [13].

The condition is typically polymicrobial, reflecting the complex oral flora. Aerobic and anaerobic bacteria are frequently implicated, with Staphylococcus, Streptococcus, Peptostreptococcus, Fusobacterium, Bacteroides, and Actinomyces being the most commonly isolated organisms. Immunocompromised individuals, including those with diabetes, HIV, or on immunosuppressive therapy, are at higher risk of developing more severe infections. These patients may also harbor atypical pathogens such as Pseudomonas, Escherichia coli, Klebsiella, Enterococcus faecalis, Candida, or Clostridium species, which can complicate clinical management [13]. The pathophysiology of Ludwig's angina involves rapid spread along fascial planes rather than lymphatic pathways. The infection leads to diffuse cellulitis, edema, and induration of the floor of the mouth, elevating and posteriorly displacing the tongue. This process is often bilateral and can compromise the airway within hours. Tissue edema and inflammation extend along the submandibular, sublingual, and submental spaces, sometimes progressing to the parapharyngeal and retropharyngeal compartments, which increases the risk of mediastinal involvement. The combination of polymicrobial infection, fascial plane continuity, and potential systemic

immunocompromise underpins the rapid progression and high morbidity associated with Ludwig's angina.

Etiology

The submandibular space is the principal site affected in Ludwig's angina [14,15]. The mylohyoid muscle anatomically divides this region into sublingual and submaxillary compartments, dictating the spread of odontogenic infections. Most cases have a clear dental origin, with infections of the second and third mandibular molars accounting for over 90% of presentations [12]. These infections exploit the anatomical relationship of the roots to the mylohyoid line, facilitating direct invasion into the sublingual and submaxillary spaces. Once established, the infection may extend contiguously into the pharyngomaxillary and retropharyngeal regions, creating a circumferential involvement of the airway, which represents the principal life-threatening complication [4]. Although odontogenic infections are overwhelmingly the cause, other etiologies have been documented, including mandibular fractures, trauma to the neck or floor of the mouth, sialadenitis of the submandibular or sublingual glands, malignancies, and parapharyngeal space infections [2,3,15]. In over half of patients, cultures reveal polymicrobial infections dominated by Staphylococcus, Streptococcus, and Bacteroides species [15]. Atypical pathogens are more frequently isolated in immunocompromised populations, increasing the complexity and severity of the condition. Opportunistic bacteria and fungi in these populations can exacerbate tissue destruction, delay recovery, and necessitate broader-spectrum antimicrobial therapy [15].

Evaluation

The immediate assessment of a patient with suspected Ludwig's angina must prioritize airway security, as airway obstruction represents the most imminent threat [13]. Rapid swelling of the submandibular and sublingual spaces can quickly compromise oral and pharyngeal passages, necessitating early intervention. Transporting patients to radiologic imaging prior to securing the airway is discouraged due to the risk of sudden obstruction. The preferred initial airway management includes awake fiberoptic intubation, which allows for continuous monitoring while minimizing the risk of complete airway collapse. Emergent awake tracheostomy should be prepared as a contingency in case conventional intubation fails [13]. Once the airway is secured, imaging studies can be employed to delineate the extent of infection and identify any abscess formation. Computed tomography with intravenous contrast provides detailed visualization of the submandibular, sublingual, parapharyngeal, and retropharyngeal spaces, assessing soft tissue edema and fluid accumulation [16]. Magnetic resonance imaging may also be utilized to identify mediastinal involvement. Radiographs can demonstrate gas formation in anaerobic infections, while ultrasonography can detect pus collections and guide percutaneous drainage [17]. Laboratory investigations, including blood cultures, may aid in identifying systemic spread but are secondary to clinical assessment. Initial management and diagnosis remain primarily clinical, with imaging and laboratory studies serving to supplement treatment planning rather than guiding urgent intervention [13].

Treatment

Controlling the Airway

Management of Ludwig's angina begins with securing the airway, as airway compromise is the most immediate and life-threatening complication of this condition. The decision to intervene is guided by the clinician's assessment of respiratory distress, edema, and the patient's overall stability, as there is no universally accepted protocol for airway management in Ludwig's angina [18]. Airway obstruction may develop rapidly due to the progressive edema of the submandibular, sublingual, and submental spaces, which elevates and posteriorly displaces the tongue. Early recognition and intervention are critical to prevent hypoxia and asphyxiation. Orotracheal or fiberoptic nasotracheal intubation is generally recommended, as blind nasotracheal intubation carries a high risk of abscess rupture, hemorrhage, and laryngospasm [15,18,19]. Patients who are not intubated require emergency airway devices such as tracheostomy and cricothyroidotomy instruments [4]. In many cases, airway management may initially involve vigilant observation with intravenous antibiotics, but progression of cellulitis or worsening dyspnea necessitates prompt operative intervention. Mechanical control of the airway must be established before the onset of stridor, cyanosis, or severe hypoxia. Collaboration with otolaryngology and anesthesia specialists is recommended for patients at high risk of airway compromise. In critical scenarios, emergency tracheostomy may be the only viable option [1]. Fiberoptic nasal intubation under direct visualization is preferred in suspected airway compromise, as it allows the clinician to safely advance an endotracheal tube while continuously monitoring the airway [22]. Nasopharyngeal airways may provide temporary support, but advanced Ludwig's angina often precludes blind intubation due to the risk of inducing severe laryngospasm. In cases where fiberoptic intubation is unsuccessful, tracheostomy remains the definitive alternative [1].

Antibiotics

Prompt initiation of broad-spectrum intravenous antibiotics is a cornerstone of Ludwig's angina management. Empiric therapy should provide coverage against both aerobic and anaerobic pathogens, which are frequently polymicrobial in odontogenic infections. Commonly used regimens include combinations of penicillin, clindamycin, and metronidazole [3]. The choice of antibiotics may be

expanded in immunocompromised patients to include coverage for Gram-negative organisms and methicillin-resistant Staphylococcus aureus (MRSA) [13]. Corticosteroids have been suggested in case reports to reduce airway edema, potentially decreasing the need for urgent airway intervention [2,15,23]. However, randomized controlled trials have not demonstrated a clear benefit, and their use remains discretionary [4]. Antibiotic therapy should begin as soon as cultures are obtained, with treatment generally extending for approximately two weeks. Monitoring of white blood cell counts and body temperature is essential to assess therapeutic response. In odontogenic cases, extraction of the offending tooth is critical to remove the source of infection. Failure to improve on antibiotic therapy or imaging evidence of fluid collection necessitates needle aspiration or surgical drainage [13].

Surgery

Surgical intervention is indicated for patients with abscess formation, necrotic tissue, or failed medical therapy. Early surgical decompression in the absence of purulence does not typically improve outcomes and is therefore not universally recommended [3]. However, early extraction of the causative tooth and collection of cultures are warranted in patients requiring intubation due to dental-origin infections. When abscess formation is confirmed by imaging or needle aspiration, incision and drainage are performed under general anesthesia with appropriate airway protection. The incision is generally placed perpendicular to the skin, approximately two fingerbreadths below the mandibular angle. During the procedure, the submandibular gland may be mobilized and the mylohyoid muscle divided to decompress the affected fascial compartments [17]. Abscesses may not develop until 24–36 hours after symptom onset, necessitating repeat imaging if the patient's response to antibiotics is inadequate or if physical examination reveals fluctuance. Computed tomography or ultrasonography can identify fluid collections requiring drainage. Needle aspiration may serve as both a diagnostic and therapeutic measure, allowing for Gram staining and culture of aerobic and anaerobic organisms [26]. Surgical drainage, along with continued broad-spectrum antibiotic therapy and removal of the source tooth, remains the definitive management approach for advanced or complicated cases [1]. In summary, treatment of Ludwig's angina involves a coordinated approach that prioritizes airway security, prompt empiric antimicrobial therapy, and surgical intervention when indicated. Airway management is the critical initial step, with fiberoptic intubation or emergency tracheostomy serving as essential life-saving procedures. Antibiotic therapy must be broad and initiated early, with adjustments based on culture results and patient response. Surgical drainage is reserved for patients with confirmed abscesses or failure of medical therapy, and the causative tooth should be removed to eliminate the nidus of infection. Coordination among otolaryngology, anesthesia, dental, and critical care teams ensures timely intervention, reduces morbidity, and significantly improves survival outcomes.

The interdisciplinary approach to Ludwig's angina exemplifies the importance of rapid decision-making, vigilant monitoring, and tailored therapy to address both the underlying infection and its potentially life-threatening complications. Continuous reassessment of airway status, infection progression, and response to antibiotics is critical, particularly in immunocompromised patients or those presenting with severe cellulitis. With the combined use of advanced imaging, broad-spectrum antimicrobials, and judicious surgical intervention, the mortality rate has decreased from nearly 50% in the pre-antibiotic era to less than 5% in modern practice [1,2,3,4]. Treatment planning should also incorporate preventive strategies. Dental evaluation and management of chronic odontogenic infections, along with patient education regarding oral hygiene, may prevent recurrence. Post-discharge follow-up should include monitoring for residual infection, assessment of airway patency, and evaluation of functional outcomes related to speech, swallowing, and mastication. Multidisciplinary involvement, including nursing staff for continuous monitoring, pharmacists for antibiotic stewardship and dosing optimization, dental specialists for source control, and radiologists for imaging-guided interventions, ensures comprehensive care. By integrating these strategies, patient morbidity is minimized, hospital stays are reduced, and the risk of long-term complications, including mediastinitis and septicemia, is substantially mitigated. Overall, successful management of Ludwig's angina relies on early recognition, rapid airway intervention, appropriate antibiotic coverage, timely surgical drainage, and multidisciplinary coordination. Clinicians must maintain a high index of suspicion for airway compromise and escalate care promptly to prevent fatal outcomes. The integration of surgical, medical, and supportive care, guided by clinical assessment and imaging, is essential for achieving favorable outcomes in this severe and rapidly progressing infection.

Conclusion:

Ludwig's angina continues to represent one of the most dangerous deep neck infections due to its rapid progression, bilateral involvement, and high risk of airway obstruction. The condition's hallmark features—severe soft-tissue edema, posterior tongue displacement, and contiguous spread across cervical fascial planes—make early clinical recognition vital to survival. Airway assessment and protection remain the most decisive steps in management, as delays can lead to sudden and fatal obstruction. Awake fiberoptic intubation is preferred, with emergency tracheostomy as a critical backup strategy in unstable patients. Effective treatment hinges on immediate initiation of

broad-spectrum IV antibiotics, targeting common aerobic and anaerobic oral flora while accounting for atypical pathogens in immunocompromised individuals. Surgical intervention becomes essential when abscess formation is present or when medical therapy fails to halt progression. Equally important is prompt extraction of the offending tooth to eliminate the infection source. Modern outcomes demonstrate a significant reduction in mortality thanks to advancements in imaging, antimicrobial therapy, and airway techniques. However, optimal results are only achieved through multidisciplinary teamwork involving emergency physicians, anesthesiologists, surgeons, radiologists, nurses, pharmacists, and dental specialists. Continued vigilance, rapid intervention, and coordinated care remain essential to preventing the life-threatening complications associated with this aggressive infection.

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