

Ludwig's Angina: Diagnosis and Treatment

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Arare disorder, Ludwig's angina is a serious, potentially life-threatening infection of the neck and the floor of the mouth (Table 1). Originally described by Wilhelm Frederick von Ludwig in 1836,¹ this condition is notorious for its aggressiveness, rapid progression to airway compromise, and high mortality when not treated promptly.²⁻⁶ This article discusses the pathophysiology, clinical manifestations, diagnosis, and treatment of this dangerous condition. Two cases are described to illustrate typical presentations of the disorder.

ILLUSTRATIVE CASE PRESENTATIONS

Patient 1

A 39-year-old man with type 1 diabetes mellitus comes to the emergency department reporting a 3-day history of fever, chills, and facial swelling. He also reports difficulty swallowing. Examination shows an acutely ill-appearing man with evident facial edema but no acute respiratory distress (Figure 1).

Vital signs include an oral temperature of 38.7°C (101.7°F), a heart rate of 140 bpm, and a respiratory rate of 18 breaths/min. Examination of the oral cavity reveals numerous carious teeth, dry oral mucosae, and woody, tender edema of the floor of the mouth and anterior neck. Plain radiographs of the neck show evidence of gas in the soft tissues (Figure 2), a finding that is confirmed by a computed tomography (CT) scan (Figure 3). Leukocyte count is $36 \times 10^3/\text{mm}^3$, and anion gap measurement indicates metabolic acidosis.

After stabilization by administration of intravenous fluids and antibiotic agents, the patient is transferred to the operating room, where a formal tracheostomy is performed. An additional drainage procedure is later performed because of an infected fluid collection, and complete dental extraction is also necessary.

Patient 2

A 53-year-old woman with type 1 diabetes mellitus reports a 2-day history of a worsening sore throat that has resulted in an inability to swallow her saliva. She has a productive cough and says that her face has become swollen from her neck to the lower part of her ears, bilaterally. The patient sits upright with her

mouth open and her tongue protruding slightly (Figure 4).

Vital signs include an oral temperature of 37.1°C (98.8°F), a pulse of 110 bpm, and a respiratory rate of 24 breaths/min. Physical examination reveals no respiratory distress. Oral examination reveals a foul breath odor, trismus, and a brawny submandibular swelling that elevates the tongue. Examination of the lungs reveals right posterior rales. Results of laboratory testing include a leukocyte count of $17,500 \times 10^3/\text{mm}^3$, a serum glucose level of 285 mg/dL, and a serum bicarbonate level within normal limits. Radiographs of the soft tissue of the neck show supraglottic edema (Figure 5), and a CT scan confirms supraglottitis and soft-tissue gas (Figure 6). A chest radiograph shows a right-sided infiltrate, consistent with aspiration pneumonitis (Figure 7).

The patient receives antibiotics, intravenously, and is transferred to the operating room for fiberoptically guided orotracheal intubation. However, intubation is unsuccessful because of airway distortion. A surgical airway is then established.

PATHOPHYSIOLOGY

Ludwig's angina is a rapidly progressing polymicrobial cellulitis of the sublingual and submandibular spaces that can result in life-threatening airway compromise. The bacteriology of Ludwig's angina is polymicrobial and predominantly involves the oral flora. The organisms most often isolated in patients with the disorder are *Streptococcus viridans* and *Staphylococcus aureus*. Anaerobes also are frequently involved, including bacteroides, peptostreptococci, and peptococci. Other gram-positive bacteria that have been isolated include *Fusobacterium nucleatum*, *Aerobacter aeruginosa*, spirochetes, and *Veillonella*, *Candida*, *Eubacteria*, and *Clostridium* species. Gram-negative organisms that have been isolated include *Neisseria* species, *Escherichia coli*, *Pseudomonas* species, *Haemophilus influenzae*, and *Klebsiella* species.

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Table 1. Clinical Pearls Concerning Ludwig's Angina

Ludwig's angina is rare and sometimes fatal.
Morbidity and mortality primarily result from airway compromise from swelling.
Etiology usually involves an odontogenic infection.
Streptococci and staphylococci are the most common bacteria associated with Ludwig's angina.
Early recognition, prompt airway control, and antibiotic administration are the keys to optimum outcome.
Early consultation with an anesthesiologist and an ear-nose-throat surgeon are strongly encouraged.

ANATOMY

In order to appreciate the potential of this infection to spread from the floor of the mouth to the neck and mediastinal structures, a brief review of the involved anatomy is helpful (**Figure 8**). The submandibular space comprises part of the space above the hyoid bone. The total space is divided into the sublingual space superiorly and submandibular space inferiorly. The former, located between the geniohyoid and mylohyoid muscles, and the latter, located between the mylohyoid muscle and the superficial fascia and skin, communicate around the free posterior border of the mylohyoid muscle.

Once established in the submandibular space, the infection can then spread to adjacent structures. Typically affected structures, in order of most frequent contamination, are the anterior neck, the pharyngomaxillary space, the retropharynx, and the superior mediastinum.

EPIDEMIOLOGY

Most cases of Ludwig's angina occur in previously healthy persons. Predisposing conditions include diabetes mellitus, neutropenia, alcoholism, aplastic anemia, glomerulonephritis, dermatomyositis, and systemic lupus erythematosus (**Table 2**). Most affected patients are between age 20 and 60 years, although an age range from 12 days to 84 years has been reported.⁶ There is a male predominance (3:1 to 4:1)⁶ of the disorder.

ETIOLOGY

Ludwig's angina usually originates from an odontogenic infection, especially from the second or third lower molars. These teeth have roots that lie at the level of the mylohyoid muscle, and abscesses here can spread to the submandibular space. Other less commonly reported causes of Ludwig's angina include sialadenitis, peritonsillar abscess, open mandibular



Figure 1. Appearance of patient 1.

fracture, infected thyroglossal duct cyst, epiglottitis, intravenous injections of drugs into the neck, traumatic bronchoscopy, endotracheal intubation, oral lacerations, tongue piercing, upper respiratory infections, and trauma to the floor of the mouth.

Ludwig's angina can be thought of as a cellulitis of the submandibular space that spreads to the structures of the anterior neck and beyond via connective tissue, muscle, and fascial planes rather than by the lymphatic system. For this reason, adenopathy is not associated with the disorder. Cellulitis, rather than abscess formation, is the most common early presenting finding. As the infection progresses, edema of the suprahyoid tissues and supraglottic larynx elevate and posteriorly displace the tongue, resulting in life-threatening airway narrowing. In advanced infection, cavernous sinus thrombosis and brain abscess, in addition to airway compromise, have been described. Other reported complications of Ludwig's angina include carotid sheath infection and arterial rupture, suppurative thrombophlebitis of the internal jugular vein, mediastinitis, empyema, pericardial and/or pleural effusion, osteomyelitis of the mandible, subphrenic abscess, and aspiration pneumonia.^{5,7-10}

CLINICAL MANIFESTATIONS

Patients with Ludwig's angina typically have a history of recent dental extraction or of poor oral hygiene and dental pain. Clinical findings are consistent with sepsis and include fever, tachypnea, and tachycardia. Patients may be anxious, agitated, and confused. Their symptoms

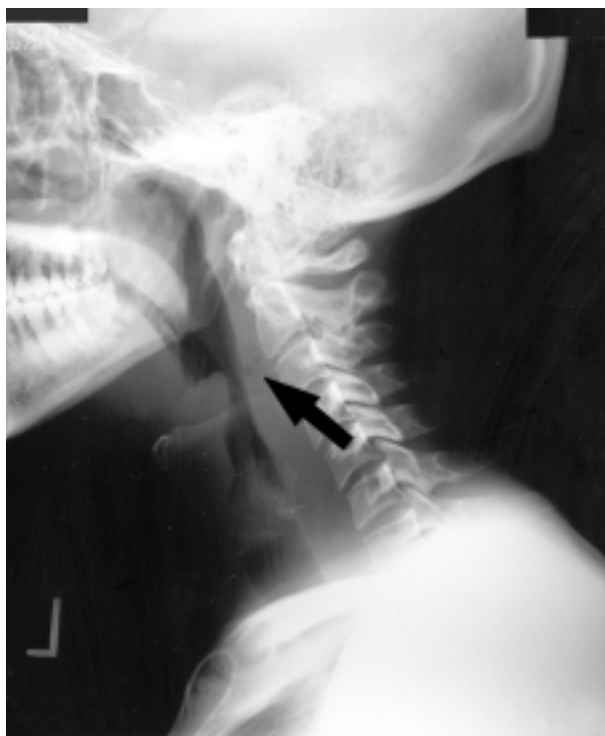


Figure 2. Soft-tissue radiograph of patient 1 showing the presence of gas (arrow).

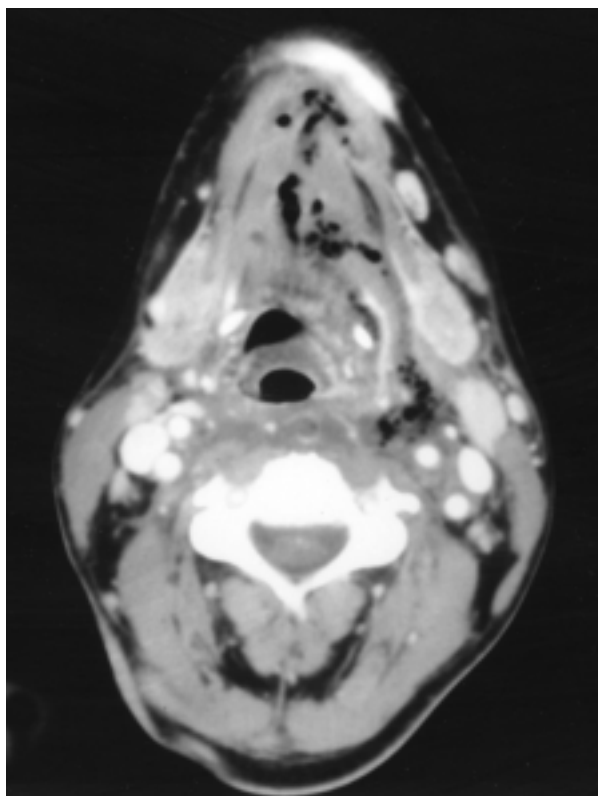


Figure 3. Computed tomography scan of patient 1.

can include swelling and pain in the floor of the mouth and anterior neck, fever, dysphagia, odynophagia, drooling, trismus, toothache, and fetid breath. Hoarseness, stridor, respiratory distress, decreased air movement, cyanosis, and a “sniffing” position (ie, the characteristic posture assumed by patients with impending upper airway compromise consisting of an upright posture with the neck thrust forward and the chin elevated) are all signs of impending airway catastrophe. Patients may exhibit dysphonia. More specifically, they may have a muffled tone at higher registers (ie, a “hot potato” voice) caused by edema of the vocal apparatus; this finding should be a warning to clinicians of potentially severe airway compromise.

On oral examination, elevation of the tongue, woody, brawny induration of the floor of the mouth and anterior neck, and nonfluctuant suprahyoid swelling typify the disease process. There is typically a bilateral submandibular edema, with marked tenderness on palpation and, occasionally, subcutaneous emphysema. The swelling of the anterior soft tissues of the neck above the hyoid bone sometimes leads to the characteristic “bull’s neck” appearance of affected patients. Adenopathy and fluctuance are not usually seen in patients with Ludwig’s angina.

DIAGNOSIS

Awareness and recognition of the possibility of Ludwig’s angina is the first and most essential step in the diagnosis and management of this serious condition. There are 4 cardinal signs of Ludwig’s angina: (1) bilateral involvement of more than a single deep-tissue space; (2) gangrene with serosanguinous, putrid infiltration but little or no frank pus; (3) involvement of connective tissue, fasciae, and muscles but not glandular structures; and (4) spread via fascial space continuity rather than by the lymphatic system.^{6,9} The presence of brawny induration of the floor of the mouth in a suggestive clinical presentation should prompt the clinician to move rapidly toward airway stabilization first, followed by further diagnostic confirmation. Unnecessary perturbation of the patient, such as forcing him or her into a supine position or prematurely attempting endotracheal intubation, might result in laryngospasm and complete upper airway obstruction and thus should be avoided.

Plain radiographs of the neck and chest often show soft-tissue swelling, the presence of gas, and the extent of airway narrowing. Sonography has been used to identify fluid collections in the soft tissues, as has gallium citrate



Figure 4. Appearance of patient 2.



Figure 5. Soft-tissue radiograph of patient 2 showing supra-glottic swelling (arrow).

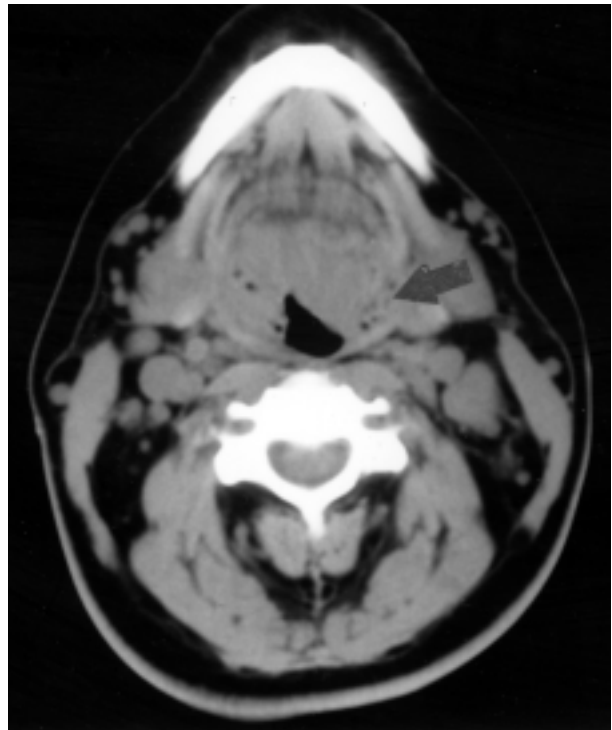


Figure 6. Computed tomography scan of patient 2, showing supraglottitis and soft-tissue gas (arrow).

Ga-67 scanning. Panoramic radiographic views of the jaw may show a dental focus of infection. After the airway patency is assured, CT scanning is a valuable modality to show the extent of soft-tissue swelling, the presence of gas, fluid collection, and airway compromise. Magnetic resonance imaging is another elegant modality that can be considered in some patients.⁶

The differential diagnosis of Ludwig's angina includes angioneurotic edema, lingual carcinoma, sublingual hematoma (following anticoagulation), salivary gland abscess, lymphadenitis, cellulitis, and peritonsillar abscess (Table 3).

MANAGEMENT

A suggested algorithm for the diagnosis and management of Ludwig's angina is provided in Figure 9. Because morbidity and mortality in Ludwig's angina are primarily related to loss of airway patency, protection of the airway takes highest priority in the initial management of affected patients. Early consultation with an anesthesiologist and an otolaryngologist is prudent, and transfer of the patient to the operating room should be strongly considered before manipulation of the airway begins. Patients who do not require immediate airway



Figure 7. Chest radiograph of patient 2 showing a right-sided infiltrate.

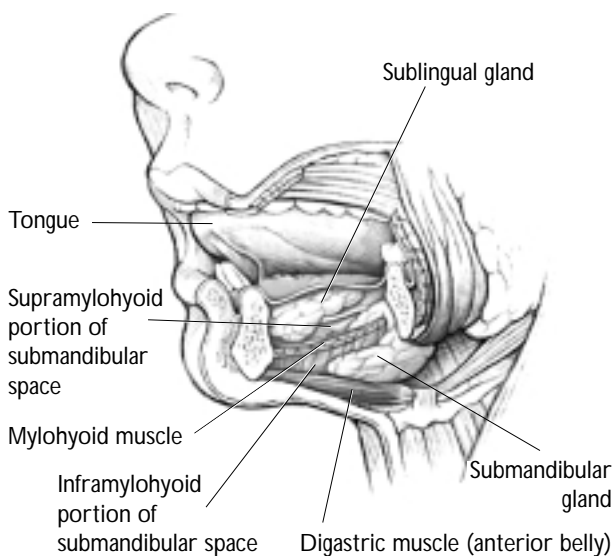


Figure 8. Anatomy of the mandibular space. (Adapted from Hartmann RW Jr. Ludwig's angina in children. *Am Fam Physician* 1999;60:109–12. Used with permission of Steve Oh.)

control should receive continuous monitoring for signs of impending respiratory distress.

For those patients who are in more immediate jeopardy, airway control is ideally achieved in the operating room, with surgical backup available, for performance of cricothyroidotomy or formal tracheostomy, if necessary. Endotracheal intubation of these patients can be difficult, and direct laryngoscopy may even precipitate the loss of the airway. Awake fiberoptic intubation is an attractive alternative and should be considered when

Table 2. Predisposing Conditions in Cases of Ludwig's Angina*

Diabetes
Neutropenia
Alcoholism
Aplastic anemia
Glomerulonephritis
Dermatomyositis
Systemic lupus erythematosus

*Entries are listed in order of decreasing frequency.

Table 3. Differential Diagnosis in Cases of Ludwig's Angina

Angioneurotic edema
Cellulitis
Lingual carcinoma
Lymphadenitis
Peritonsillar abscess
Salivary gland abscess
Sublingual hematoma

the degree of airway narrowing is severe. Blind nasotracheal intubation should be avoided because of the degree of airway distortion and because resulting trauma may further narrow an already tenuous airway or cause bleeding or rupture of an abscess.

When severe swelling or trismus prevents orotracheal intubation, formal tracheostomy remains the gold standard for securing the airway. This procedure is frequently complicated by extensive soft-tissue swelling of the anterior neck, and it risks spreading the infection to the mediastinum. In the setting of imminent loss of the airway, however, clinicians must not hesitate to attempt orotracheal intubation or needle or surgical cricothyroidotomy, even if conditions are sub-optimal. Administration of nebulized epinephrine has been suggested as a possible adjunct prior to airway manipulation, when time allows, in an attempt to reduce mucosal swelling.

Once the airway is secured, aggressive intravenous administration of antibiotic agents should begin (**Table 4**). Initial treatment is targeted at gram-positive organisms and anaerobes in the oral cavity. Administration of several antibiotic agents has been proposed, including high-dose penicillin G plus metronidazole,

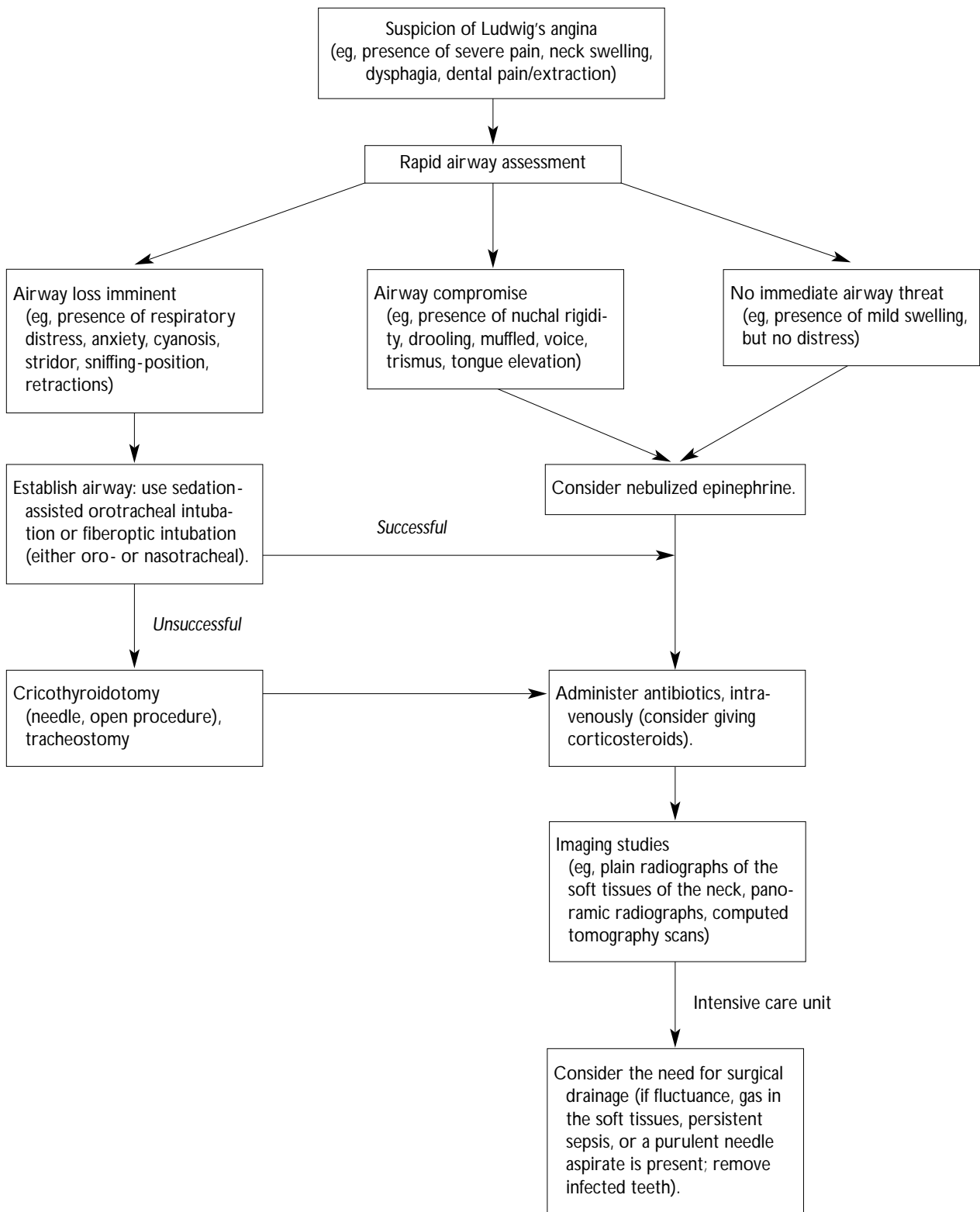


Figure 9. A suggested algorithm for the diagnosis and management of Ludwig's angina.

clindamycin, cefoxitin, piperacillin-tazobactam, amoxicillin-clavulanate, and ticarcillin-clavulanate. Although controversial, the use of dexamethasone to decrease edema and to improve antibiotic penetration has received some support.

Surgical drainage is indicated when there is suppurative infection, radiologic evidence of fluid collection or soft-tissue air, clinical fluctuance, crepitus, or a purulent needle aspirate. Drainage is also indicated when there is no clinical improvement after an initial course of antibiotic therapy. Removal of infected teeth is also essential to complete the drainage process.

The prognosis in Ludwig's angina depends primarily on immediate protection of the airway and then on prompt antibiotic—and possibly surgical—treatment of the infection. Mortality in the preantibiotic era was 50% but, with the advent of current therapies, has declined to less than 5%.^{7,8,10}

CONCLUSION

Ludwig's angina is a dramatic, life-threatening, soft-tissue infection of the floor of the mouth and neck. If vigilant for its clinical presentation and aware of its potential for rapid compromise of the patient's airway, clinicians can intervene early in order to prevent its most dire consequences.

Airway control is of paramount importance, and attention to this consideration, combined with antibiotic therapy, surgical drainage, and modern intensive care, have all contributed to a declining mortality. Early and liberal consultation with otolaryngology and anesthesiology services will assure the greatest hope for speedy airway control, prompt institution of intravenous antibiotic therapy, and an uncomplicated recovery from this rare and dreaded condition. **HP**

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Table 4. Commonly Used Antibiotic Agents in Cases of Ludwig's Angina

Ampicillin/sulbactam
Cefoxitin
Clindamycin*
Gentamicin
Penicillin G plus metronidazole
Piperacillin/tazobactam
Ticarcillin/clavulanate

*Administer if patient is allergic to penicillin.

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SUGGESTED READING

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