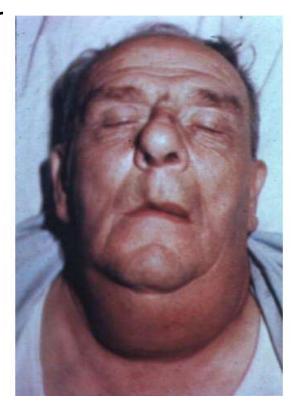
LUDWIGS ANGINA

Ludwigs angina

- Ludwig's angina is a serious, potentially lifethreatening infection of the neck and the floor of the mouth
- Originally described by Wilhelr
 Ludwid





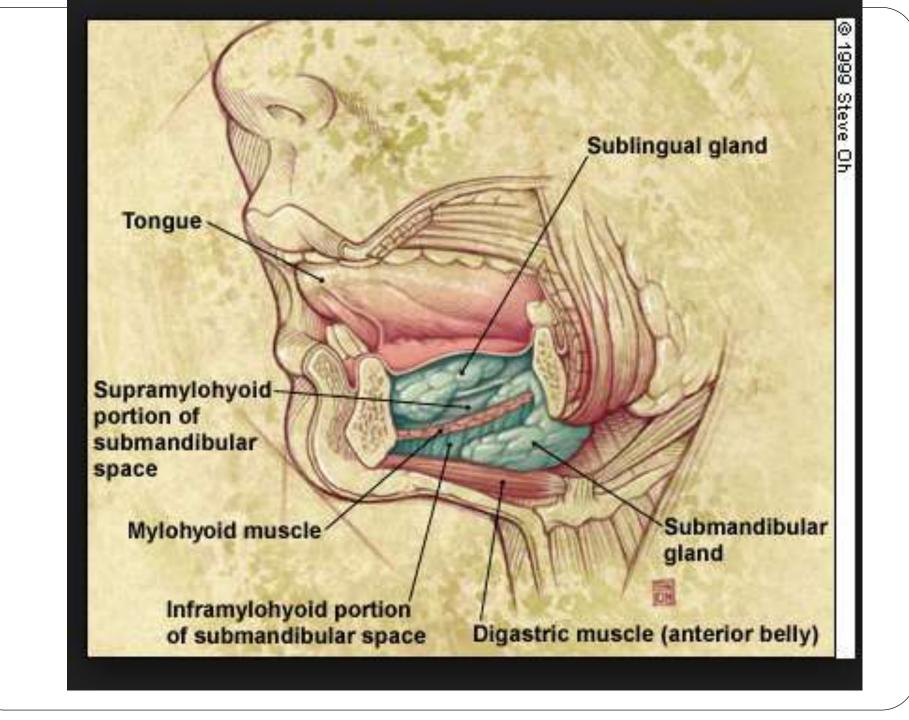
PATHOPHYSIOLOGY

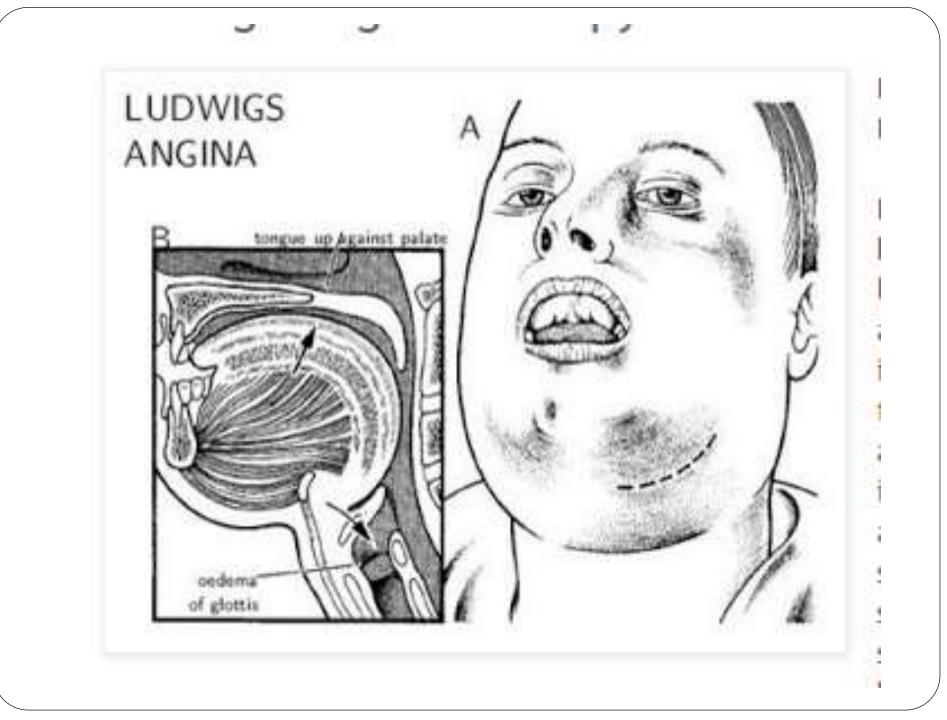
- Ludwig's angina is a rapidly progressing polymicrobial cellulitis of the sublingual and submandibular spaces
- Results in life threatnening air way compromise
- The organisms most often isolated in patients with the disorder are Streptococcus viridans and Staphylococcus aureus
- Anaerobes also are frequently in bacteroides, peptostreptococci, fusiform bacilli, diptheroids.
- Non specific mixed infection

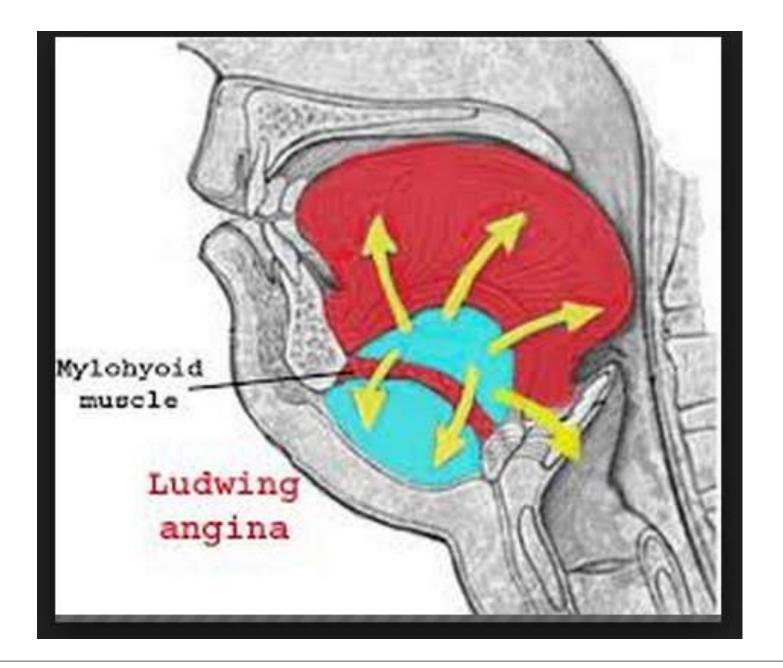


Anatomy

- 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
- Ludwigs angina begins in the submaxillary space and secondarily involves submental and sublingual space
- Typically affected structures, in order of most frequent contamination, are the anterior neck, the pharyngomaxillary space, the retropharynx, and the superior mediastinum.

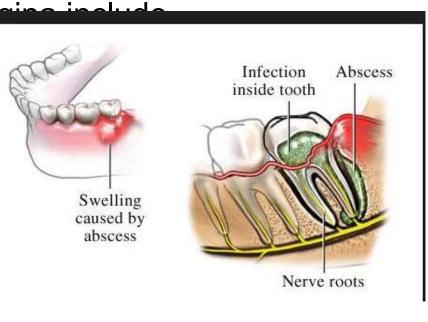




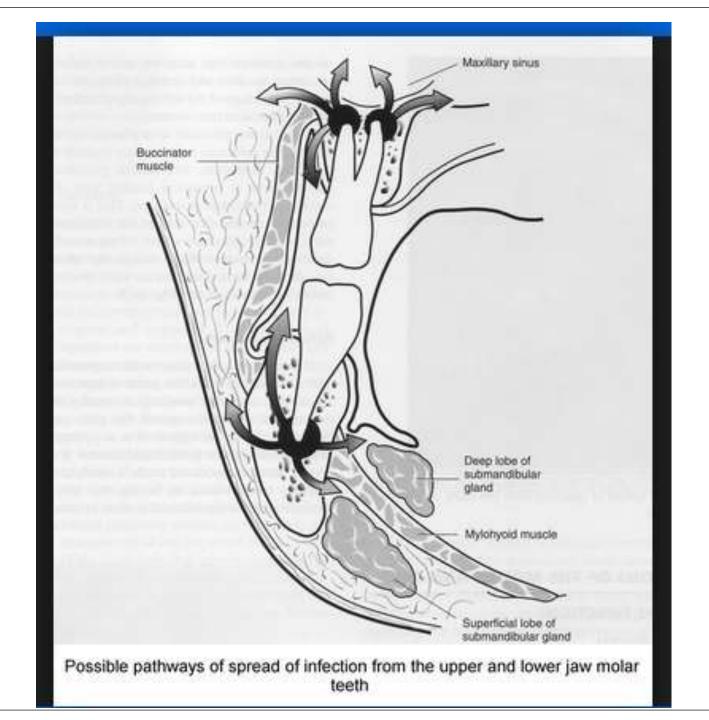


Etiology

- 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 causes of Ludwig's an
- Sialadenitis
- Peritonsillar Abscess
- Open Mandibular Fracture
- Infected Thyroglossal Duct C
- Epiglottitis
- Oral Lacerations
- Tongue Piercing
- Upper Respiratory Infections
- Trauma To The Floor Of The Mouth.



- Ludwig's is 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.
- 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 lifethreatening airway narrowing.
- In advanced infection, cavernous sinus thrombosis and brain abscess, in addition to airway compromise, have been described.



- Predisposing factors include:
- A. Dental Carries
- B. Recent Dental Treatment
- C. Systemic Illnesses Such As Diabetes Mellitus
- D. Malnutrition
- E. Alcoholism
- F. Compromised Immune System Such As AIDS
- G. Organ Transplantation And Trauma

Clinical manifestations

- Poor oral hygeine, tooth pain
- Tachypnea, and tachycardia and fever
- Swelling and pain in the floor of the mouth
- Dysphagia, odynophagia, drooling, trismus, and fetid breath
- Hoarseness, stridor, respiratory decreased air movement, cyand
- Patients may exhibit dysphonia.



On Oral examination

- Board like swelling of floor of mouth
- Elevation of the tongue
- Nonfluctuant suprahyoid swelling typify the disease process. There is typically a bilateral submandibular edema,
- 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

Tongue protrusion culminating in rapid and progressive airway obstruction.



- As disease continues swelling of neck , edema of glottis occur
- Causes serious risk of suffocation
- Infection may spread to parapharyngeal spaces, carotid sheath and pterygopalantine fossa
- Cavernous sinus thrombosis is a sequela to this infection
- Other complications such as descending necrotising mediastinitis usually occur through the retropharyngeal space (71%) and the carotid sheath (21%)

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 bacte-

ria associated with Ludwig's angina.

Early recognition, prompt airway control, and antibiotic administration are the keys to optimum outcome.

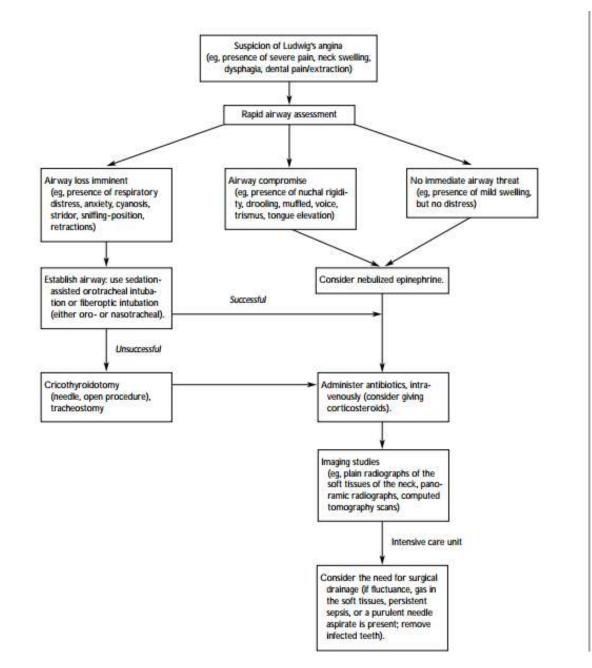
Early consultation with an anesthesiologist and an ear-nosethroat surgeon are strongly encouraged.

Diagnosis

- 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

Differential diagnosis

- Angioneuretic edema
- Cellulitis
- Lingual carcinoma
- Lynmphadenitis
- Peritonsillar abscess
- Salivary gland abscess
- sublingual haematoma



Suggested algorithm for diagnosis and management of

Management

- Protection of the airway takes highest priority in the initial management of affected patients.
- Immediate air way mangement is with cricothyroidotomy or formal tracheostomy
- When severe swelling or trismus prevents orotracheal intubation, formal tracheostomy remains the gold standard for securing the airway
- Administration of nebulized epinephrine has been suggested as a possible adjunct prior to airway manipulation

Tracheostomy and drainage





Once the airway is secured, aggressive intravenous administration of antibiotic agents should begin

are

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.

Management protocol

- The main requirements are:
- immediate admission to hospital
- procurement of a sample for culture and sensitivity testing
- aggressive antibiotic treatment
- securement of the airway by *tracheostomy* if necessary, and drainage of the swelling to reduce pressure.