Ludwig’s Angina and Treatment Considerations in the COVID-19 Pandemic

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Abstract

Introduction
Ludwig’s angina is a type of severe cellulitis that spreads rapidly and carries a significant risk of airway compromise. Previous complications with COVID-19 are poorly described within the literature.

Case Presentation
This case report describes the complication of COVID-19 infection with suspected Ludwig’s angina 2 days after admission, resulting in awake fibroscopic endotracheal intubation. Emergent treatment and establishing a secure airway are paramount in these cases. We discuss the role of antibiotics and adjunct treatment in these cases of potential airway compromise.

Conclusion
Limited data demonstrate simultaneous infection of COVID-19 with these types of submandibular soft tissue infections in the literature. Previous explorations into this subject are limited, as COVID-19 is a relatively new condition with its own treatment guidelines. We discuss specifically the role of corticosteroid use and surgical intervention in these cases. We wish to highlight awareness and treatment considerations for COVID-19 patients with superimposed Ludwig’s angina.

Keywords
Ludwig’s angina; COVID-19; airway management; awake fiberoptic intubation

Introduction
Ludwig’s angina is a rare subtype of cellulitis that presents with acute onset in the submandibular and sublingual areas. This condition was named after Dr. Wilhelm von Ludwig, who may have died from the infection that bears his name. The nidi for Ludwig's angina are typically from odontogenic infections (>75%) in the molar region, with no significant gender bias.

Presentation typically involves a rapid progression of gangrenous cellulitis and edema into the neck’s soft tissues. The most dangerous complication of Ludwig’s angina is airway obstruction due to edematous swelling of the soft tissues and posterior displacement of the tongue. Treatment involves emergent airway management and empirical antibiotic therapy. Surgical incision and drainage are performed in cases of severe airway compromise. Due to the low incidence of Ludwig’s angina, limited data demonstrate simultaneous SARS-CoV-2 (COVID-19) infection in the literature.

We present the case of a patient with COVID-19 complicated by Ludwig’s angina to highlight complications in the traditional treatment of COVID-19 pneumonia.

Case Description
Our patient was a 72-year-old female with past medical history inclusive of obesity, prediabetes, obstructive sleep apnea, and coronary artery disease who presented to the emergency department with chief complaint of right ear pain.
department with a chief complaint of shortness of breath.

No odontogenic infection or other dental pathology was noted, and our patient did not report any oral or tooth pain. She did not receive a COVID-19 vaccine. Her symptoms began 3 days before admission, and she believes she was in contact with someone who had also contracted COVID-19. A COVID-19 polymerase chain reaction test was positive. Imaging revealed bilateral pulmonary infiltrates consistent with COVID-19 infection. Remdesivir and dexamethasone were initiated, and the patient was started on 4 L of O₂ nasal cannula. Her vital signs were stable at this time.

On the second day of admission, our patient noticed significant edema and erythema over the left submandibular area with severe pain. This swelling extended over to the anterior neck and down to the clavicular and chest area the following day, with ecchymotic expansion and severe tenderness to palpation. The patient also noted a severe hoarse voice at this time. A computed tomography (CT) scan revealed significant swelling of the left oropharyngeal mucosa with simultaneous swelling of the uvula and tonsillar prominence (Figure 1). The swelling proceeded to expand into the upper chest area the next day. The patient was admitted to the ICU for further management and consented to intubation. She was intubated via awake fibroscopic intubation in the operating room. A surgical tracheostomy team was on standby in case of intubation failure. Antibiotic therapy with intravenous ampicillin-sulbactam 3 g was started.

The patient remained intubated and sedated with propofol and fentanyl. She was extubated after 8 days. Antibiotic therapy was changed to intravenous ceftriaxone 2 g and clindamycin 900 mg on day 6 per recommendations from the hospital’s infectious disease department. Submandibular swelling improved clinically without surgical intervention. Dexamethasone was discontinued on day 10, and our patient was discharged from the hospital on day 14.

**Discussion**

There are 3 primary aspects to treating Ludwig’s angina: airway management, empiric broad-spectrum antibiotic therapy, and incision and drainage when appropriate.

This case highlights the importance of urgent airway management on suspicion of Ludwig’s angina. Our case is a typical presentation. However, the onset is atypical due to no evident

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**Figure 1.** This CT image reveals swelling of the uvula, oropharyngeal, and hypopharyngeal mucosa with narrowing of the hypopharyngeal airway, demonstrating concerns for airway compromise and the necessity of emergent intervention.
nidus of odontogenic infection. Our case is further complicated by a simultaneous COVID-19 infection.

Intubation for hypoxic respiratory failure secondary to COVID-19 is only indicated when all other respiratory support modalities have failed, and respiratory arrest is imminent (as dictated by the persistent need for high-flow oxygen or rapid progression of dyspnea). In contrast, awake fiberoptic intubation is preferred in advanced Ludwig’s angina cases when antibiotic therapy has failed. Patients are usually intubated awake as induction of general anesthesia may precipitate airway closure. If intubation measures fail, preparations for a surgical tracheostomy must always be in place. Our patient was electively intubated despite having adequate O2 saturation on a high-flow nasal cannula, and intervention with a surgical tracheostomy was not necessary.

Corticosteroid treatments are controversial in the setting of Ludwig’s angina. Certain studies have shown decreased need for airway management with steroid use, while others show minimal benefit.3 Our patient was started on IV dexamethasone 4mg for 10 days for COVID-19. There have been relatively few studies investigating the utility of dexamethasone for Ludwig’s angina; further investigation is required.

Direct surgical intervention via incision and drainage is essential when antibiotic therapy fails in cases of severe infections. Oral anaerobes and Streptococcus species are the most common bacteria from deep neck infections. Empiric treatment in immunocompetent patients with normal renal function includes ampicillin-sulbactam, ceftriaxone, metronidazole, clindamycin, or levofloxacin for coverage of gram-positive, gram-negative, and anaerobic bacteria.4 Antibiotic therapy is not indicated for the treatment of COVID-19, as bacterial superinfection is not typically considered a prominent feature of COVID-19 infection. Our patient was started on a regimen of ampicillin-sulbactam and transitioned to ceftriaxone and clindamycin per recommendations from our infectious diseases department. Neck swelling resolved with antibiotic therapy alone, and surgical intervention with incision and drainage was not necessary.

**Conclusion**
The causal relationship between COVID-19 and Ludwig’s angina remains unclear. Superimposed COVID-19 infection can limit or change treatment modalities by adding proper antibiotic therapy and increasing the dose of steroids while continuing current therapy for COVID-19. Understanding different options for securing the airway in light of concomitant COVID-19 infection can guide treatment in the future.

**Patient Consent**
Written consent was obtained from the patient; details of the case were explained to the patient; the patient demonstrated understanding and was agreeable.

**Conflicts of Interest**
The authors declare that they have no conflicts of interest.

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