A Case of Ludwig's Angina Following Influenza in a 16-Year-Old Male

CHUKWUMA E. ONUCHUKWU, MD, MPH; NIVEDITA MURALIDHAR, MD

ABSTRACT

This case report illuminates an instance of Ludwig's Angina in a 16-year-old male patient with concurrent Influenza A. Ludwig's Angina, a potentially fatal, rapidly spreading cellulitis, primarily affects the submandibular and sublingual spaces. This case emphasizes the importance of early detection, immediate intervention, and multidisciplinary management to prevent life-threatening complications. The patient presented with fever, sore throat, swelling under the tongue and submandibular region, and difficulty breathing following a family trip to Puerto Rico. Despite initial respiratory distress and severe soft tissue swelling, the patient responded positively to broad-spectrum antibiotics, with clinical improvement leading to discharge after five days. The report discusses the challenges in diagnosing and managing Ludwig's Angina, the potential role of the patient's environment in disease presentation, and the relevance of historical medical literature in contemporary clinical practice. This case underscores the need for vigilance in patients with influenza for potential secondary bacterial complications and the necessity of expedited airway management in patients with Ludwig's Angina.

KEYWORDS: Ludwig angina, Influenza A, neck induration, difficulty breathing

INTRODUCTION

Ludwig's Angina is a potentially life-threatening, rapidly spreading cellulitis that primarily involves the submandibular and sublingual spaces, resulting from an infection in the floor of the mouth.[1] Despite its rarity, it is a critical condition for providers to be aware of due to the risk of rapid airway obstruction. Without antibiotic therapy, it often progresses rapidly and results in life-threatening upper airway obstruction with a mortality exceeding 50%.[2] It was first described in 1836, and before the widespread use of antibiotics, more than 50% of the cases were fatal.[3] The usual cause of Ludwig Angina is a mixture of aerobic and anaerobic bacteria including predominately normal oral flora.[4,5] Early recognition of this disease process can prevent a lethal outcome due to airway compression and obstruction.[4] This case report aims to highlight the importance of early recognition, immediate intervention, and multidisciplinary management to prevent lethal complications of Ludwig's Angina. The report will delve into the clinical presentation, diagnosis, and antimicrobial therapy, providing valuable insights for physicians in managing this severe condition.

CASE PRESENTATION

The patient, a 16-year-old male with a history of ADHD, presented with worsening neck swelling in the context of sore throat and fever. His symptoms began after a family trip to Puerto Rico, where he frequently swam in the ocean and pool. A friend who accompanied the family on the trip had similar symptoms and an ear infection but recovered after treatment with amoxicillin. He developed a sore throat, fatigue, and subjective fevers four days prior to presentation. One day prior, he developed swelling under the tongue and in the submandibular region, which gradually increased in size, extending bilaterally, and causing difficulty breathing. His condition worsened, necessitating presentation to the Emergency Department where he was intubated because of severe respiratory distress and inability to manage secretions. He did not receive any medical care or antibiotics prior to ED presentation and managed his symptoms at home with acetaminophen.

Upon arrival, he was saturating in the 80s, had difficulty managing secretions, and presented a muffled voice. Labs were remarkable for a WBC of 10.5 and CRP of 8.6 mg/L. He tested positive for influenza A. For airway protection, he was intubated after receiving Ketamine and Rocuronium. A CT neck scan showed diffuse soft tissue swelling, with severe edema of the structures in the neck noted by the reading radiologist. After admission, the patient remained afebrile, with fluctuating blood pressure, heart rate, and respiratory rate. He was initiated on piperacillin-tazobactam and vancomycin for broad empiric coverage of causative pathogens including oral aerobes, anaerobes, and MRSA. (**Figures 1–3**).

Examination showed an intubated child with swelling to left and right anterolateral neck region with overt fluctuance, induration, erythema, left worse than right, mildly tender. Repeat CT soft tissue neck with contrast after 48 hours showed enlarged palatine tonsils with heterogeneity

Figure 1. Sagittal view, enlargement and elevation of tongue

Figure 2. Axial view, diffuse soft tissue edema

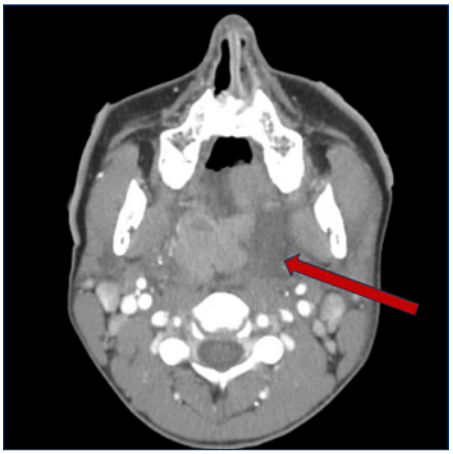

Figure 3. Coronal view, edema and adenopathy

anteriorly with lobulated tracking fluid and edema in the dorsal aspect of the sublingual space with no drainable collection.

Blood culture remained negative. His ESR was marginally increased with a normal CRP. He was treated for influenza with oseltamivir for five days. With clinical improvement, negative MRSA nasal screen, we narrowed antibiotic coverage to ampicillin-sulbactam which covers most etiologic agents. He remained admitted on parenteral antibiotic therapy for five days and was transitioned to oral amoxicillin-clavulanate at discharge for 14 days of therapy. He remained stable and well-appearing on follow-up.

DISCUSSION

This case report highlights a rare and severe presentation of Ludwig's Angina in a young patient, who also tested positive for Influenza A. The concurrence of Ludwig's Angina and influenza is not common, but it is noteworthy due to the potential exacerbation of the patient's condition and the complexity it adds to clinical management. Interestingly, more than a century ago, a similar link between Ludwig's Angina and influenza was reported.[6] In 1905, during the pre-antibiotic era, a fatal case of Ludwig's Angina associated with influenza was documented. The patient developed a rapidly progressive infection following influenza, highlighting the potential for influenza to predispose individuals to severe bacterial infections.[7] In our case, the patient also presented with influenza, which may have compromised his immune system and contributed to the development of Ludwig's Angina. The patient's symptoms began after a trip to Puerto Rico. The initial failure to identify the causative organism in our patient underscores the challenges in diagnosing and managing Ludwig's Angina. The absence of a detectable organism in our patient's case could be attributed to various factors, including the initiation of empiric antibiotic therapy prior to obtaining cultures, the presence of non-culturable organisms, or the possibility of an atypical causative agent. Despite the advancements in diagnostic technologies, there are still cases where the causative organism is not identified. It is not uncommon for the causative agent to not be identified; this is the situation in up to 50% of cases,[5] and empiric antibiotic coverage is often used in these cases as its essential to initiate prompt treatment to prevent complications and improve outcome. The typical time course for development of Ludwig's angina can vary but often develops rapidly within hours or days and respiratory obstruction which can develop suddenly can be a presentation or complication of the condition.

The infection is odontogenic in 85% of cases, and other causes include peritonsillar abscess, parapharyngeal abscess, mandibular fractures, oral piercings or wounds, and submandibular sialadenitis.[8] Common bacteria that cause Ludwig angina include: *Streptococcus viridans, Staphylococcus aureus*, B-hemolytic streptococcus species, *Staphylococcus epidermidis, Bacteroides* genus, *Fusobacterium nucleatum, Peptostreptococcus,* and *Enterobacter aerogenes*.[9] Rare organisms that have been implicated as a cause include: *Proteus, Pseudomonas, Escherichia coli, Haemophilus influenza, Neisseria catarrhalis, Borellia vincenti, and Morganella morganii*.[10] *Klebsiella pneumoniae* in one study was identified in greater than 50% of diabetic patients with Ludwig angina[9] while *Candida albicans* has also been reported.[10] The source of infection in our patient was ultimately thought to be from left palatine tonsilitis, with no evidence of odontogenic infection on visual examination of the oropharynx or cross sectional imaging.

The empirical selection of broad-spectrum antibiotics, which covered most potential etiologic agents, seemed to be effective, as evidenced by the patient's clinical improvement. Despite the advancements in medical science since the 1905 case, the management of Ludwig's Angina remains

challenging due to its rapid progression and potential for airway obstruction. Early recognition and aggressive management, as exemplified in this case, are paramount in preventing fatal outcomes. The multidisciplinary approach involving emergency medicine, infectious diseases, and otolaryngology services was crucial in managing this complex case. The association of Ludwig's Angina with influenza underlines the importance of considering secondary bacterial infections in patients with viral illnesses. This case serves as a reminder to clinicians to maintain a high index of suspicion for serious bacterial infections in patients presenting with flu-like symptoms, especially in the presence of oral or neck symptoms suggestive of Ludwig's Angina.

In conclusion, this case underscores the importance of early recognition and aggressive management of Ludwig's Angina. It also reinforces the need for vigilance in patients with influenza for potential secondary bacterial complications. The parallels between our case and the 1905 report serve as a testament to the enduring relevance of historical medical literature in contemporary clinical practice.

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Authors

Chukwuma E. Onuchukwu, MD, MPH, Fellow, Pediatric Infectious Diseases, Hasbro Children's Hospital/The Warren Alpert Medical School of Brown University, Providence, Rhode Island.

NiveditaMuralidhar, MD, Attending Physician, Pediatric Infectious Diseases, Hasbro Children's Hospital; Assistant Professor of Pediatrics, The Warren Alpert Medical School of Brown University, Providence, Rhode Island.

Correspondence

Chukwuma Onuchukwu, MD
ndiboy@yahoo.com

Nivedita Muralidhar, MD
Physicians Office Building, Suite 018
593 Eddy Street, Providence RI 02904
401-444-8360
nivedita.muralidhar@lifespan.org