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Ludwig's Angina

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ABSTRACT

Ludwig's angina is a bacterial infection of the tongue and floor of the mouth. It can be life-threatening if not treated promptly. Treatment includes IV antibiotics and, in some cases, surgical intervention. In this report, we describe a case with an unusual presentation. Early recognition of the condition in such cases is vital to prevent potential complications.

KEYWORDS

Ludwig's angina, bacterial infection, airway obstruction

LEARNING POINTS

- Ludwig's angina, even though a well-established clinical entity, can be hard to recognize clinically.
- Early recognition of the condition is vital for the prevention of complications.
- A low threshold for surgical treatment should be maintained when surgery is indicated.

INTRODUCTION

Ludwig's angina is characterized by diffuse cellulitis involving the floor of the mouth, submandibular region and neck that can be potentially life-threatening due to its tendency to cause airway obstruction. This condition is usually polymicrobial, with the most common culprit microorganisms being *Streptococcus*, *Bacteroides* and *Staphylococcus*. Treatment of Ludwig's angina varies depending on the patient's clinical presentation and can range from intravenous antibiotics and supportive measures to airway intervention in the setting of more advanced disease with airway compromise.

We present the case of a 58-year-old man who presented to the Emergency Room (ER) complaining of odynophagia, dysphagia, and bilateral neck swelling and tenderness.

CASE DESCRIPTION

A 58-year-old African American man with a past medical history of hypertension, diabetes, dyslipidaemia and obstructive sleep apnoea on nightly CPAP presented to the ER complaining of dysphagia and odynophagia. He had a 3-day history of difficulty swallowing liquid and solids. He denied fever, chills, shortness of breath, chest pain, or any purulent secretions. He had not been in contact with anybody who was sick and was in a monogamous relationship.

Upon physical examination in the ER, the patient was found to be hypertensive, tachycardic, afebrile, and saturating 98% on room air. He was comfortable sitting in bed and was not in distress. Very poor dentition was noted, with anterior tongue protrusion which precluded proper characterization of the oropharynx for evidence of secretions or erythematous changes. Oedema was seen bilaterally in the submental region with mild tenderness upon gentle palpation. The patient had bilateral clear lungs on auscultation.

Laboratory findings on admission included WBC 9 K/µl, glucose 235 mg/dl with HbA1c 7.3%, total cholesterol 224 mg/dl, and LDL 138 mg/dl. EKG showed sinus tachycardia with possible left atrial enlargement and right superior axis deviation. A chest x-ray showed cardiomegaly without radiographic evidence of acute pulmonary disease. A CT scan of the neck and soft tissue with contrast showed moderate left peritonsillar and retropharyngeal oedema and associated phlegmon likely infectious in aetiology without evidence of abscess. However, there was narrowing of the oropharyngeal airway with mild reactive left cervical lymphadenopathy.

In the ER, the patient received one dose of piperacillin-tazobactam 3.375 g and 125 mg of SOLU-MEDROL IV push. An ENT specialist was consulted and recommended monitoring the patient in ICU and started him on 12 mg of IV dexamethasone every 8 hours. The patient was also started on vancomycin 1.5 g IV every 12 hours and ampicillin-sulbactam 3 g every 6 hours in the setting of a possible infectious aetiology. During his ICU stay, the patient remained haemodynamically stable, and was able to transition from 'nothing by mouth' to a heart-healthy diet. WBC trended up from 9 to 12.4 K/µl with 84.3% polymorphonuclear cells. The patient was successfully discharged on amoxicillin-clavulanate 875 mg-125 mg by mouth twice a day, and Medrol Dosepak, with follow-up with an ENT specialist within 1–2 weeks after discharge.

DISCUSSION

Although uncommon, Ludwig's angina is a serious complication of an infection of the lower molar teeth that can quickly develop into lethal upper airway obstruction. Odontogenic infections are responsible for most cases, with a few rare instances caused by tongue piercings, mandibular fracture, otitis media, and sialolithiasis of the submandibular glands [1]. The cause of Ludwig's angina in our patient remains unclear; however, very poor dentition was noted on physical examination, which is a major contributing factor. Symptoms predominantly include acute onset of dysphagia and odynophagia, as in our patient, as well as facial and upper neck swelling, difficulty breathing, sore throat, and a change in voice due to narrowing of the airways. On physical examination, tenderness and erythema are commonly noted over the submandibular and/or submental regions. The diagnosis of Ludwig's angina is primarily based on clinical examination of the pharynx, which typically demonstrates an erythematous, oedematous neck, and anterior tongue protrusion.

Most cases are polymicrobial in nature, involving a combination of Gram-positive, Gram-negative and anaerobic microorganisms [2]. *Streptococcus viridans* and *Staphylococcus aureus* are the most common organisms isolated as these are frequently associated with oral and skin flora. However, it is important to also perform anaerobic cultures, even though it is not routine, as anaerobic microorganisms are likely inhabitants of the oral cavity, and can help guide treatment without delay.

Most patients require intensive care as the potential for the condition to rapidly deteriorate is high. In their retrospective study, Lin et al. found Ludwig's angina could have serious complications, particularly in those with descending necrotizing mediastinitis, who displayed longer ICU and hospital stays and a high mortality rate of 37.5%. Those with comorbidities have a more severe course of Ludwig's angina due to their immunocompromised state and hence, a higher mortality rate [3]. However, our patient, who had comorbidities, did not have a complicated course or a prolonged ICU stay. This is largely because he did not require endotracheal intubation or a tracheostomy, which significantly reduced the length of stay.

It is unclear if more conservative treatment (i.e., intravenous antibiotics without any surgical management) is the best option for managing the early stages of Ludwig's angina. A retrospective cohort study by Ekaniyere et al. reported that there was a higher incidence of airway compromise in patients who were treated with antibiotics alone versus surgical debridement plus antibiotics [4]. In contrast, our patient was managed with antibiotic therapy alone and showed daily improvement while in hospital. Moreover, it is worth noting that the administration of antibiotics when the patient is first admitted to hospital versus ICU admission may also improve outcomes. Further study is needed to examine the association between antibiotic administration upon hospital admission compared with antibiotics administration upon ICU admission after several hours, and how this may reduce the length of ICU stay and possibly the mortality rate.

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