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A Case of Ludwig's Angina: Life Threatening Deep Neck Infection Presenting with Unusual Symptoms of Chest Pain

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Authors' contributions

Author NHBW wrote the case study and literature review. Author MB finalized the manuscript and literature review.

Cash Study

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ABSTRACT

Ludwig's angina is a potentially lethal deep neck infection associated with rapid airway obstruction due to swelling of the neck, tongue and submandibular areas. Despite the name, it has no relation to angina pectoris. We report a case of an 80-year-old gentleman who presented with the unusual symptom of chest pain and previous history of diabetes mellitus and treated lymphoma, who was later diagnosed with Ludwig's angina. Despite of early intravenous antibiotics, airway support and intensive care treatment patient died within 24 hours.

This case highlights the need for a high index of suspicion for Ludwig's angina in patients with chest pain and neck swelling, as although it's lethal condition but early, rapid and aggressive intervention can save lives.

Keywords: Ludwig angina; Deep neck infection.

1. INTRODUCTION

Ludwig's angina is an aggressive soft tissue infection of the submandibular and sublingual spaces [1]. First described by Wilhelm Freidrich von Ludwig in 1836 [2] (the name derives from the Latin word *angereor* "to strangle"). Incidence of Ludwig's angina was high in pre-

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antibiotic era [3] but now it accounts for about 13% of all the deep neck tissue abscesses [4]. Although Ludwig angina is uncommon now but still lethal due to the potential for rapid airway obstruction.

The majority of cases of Ludwig's angina arise from dental infection [1], with the second mandibular molar being the most common source. Other causes include trauma, oral piercings, lymphadenitis, surgery and malignancy distorting the local anatomy and subsequently increasing the risk of infection. Following the initial infection in the submandibular space, the disease can spread along the tissue planes as far as the mediastinal space. Because of this manner of spread, the infection often results in symmetrical disease of the affected spaces.

The infection is often polymicrobial in origin, reflecting the mixed organisms found inside the human mouth. Common pathogens include group A *Streptococcus*, *Staphylococcus*, *Bacteroides*, *Haemophilus* and *Fusobacterium* [1,5]. Other risk factors for developing Ludwig's angina include immunosuppression, alcoholism and diabetes mellitus.

Patients with Ludwig's angina commonly present with unilateral or bilateral neck swelling, pain, dysphagia, tongue enlargement, drooling and trismus. Features of systemic illness, e.g. fever, malaise, rigors may also occur. As the infection worsens and airway compromise develops, the patient will show signs of respiratory distress. If allowed to progress, the infection may spread into the bones leading to osteomyelitis. Cases of Ludwig's angina complicated by necrotising fasciitis have also been reported [6]. Examination findings include tenderness and swelling of the tongue, neck and submandibular areas, lymphadenopathy, tachypnoea and stridor. These features are illustrated in Fig. 1.



Fig. 1. Extensive neck and submandibular swelling secondary to Ludwig's angina

The diagnosis of Ludwig's angina is generally made clinically. CT scanning can assist in identifying which spaces are infected and detect abscess formation in affected areas, and is recommended in patients requiring operative management [7]. Blood tests will generally show abnormalities consistent with infection.

The first priority in managing Ludwig's angina is ensuring a secure airway. Tracheal intubation through the oral route may prove difficult due to upward displacement of the tongue from infection, bleeding from inflamed tissue, and purulence [8]. Blind nasotracheal intubation may be successful, but risks causing further damage to already friable tissue and possible abscess rupture [8]. Advanced airway control techniques, such as fibre-optic intubation, upright positioning or retrograde intubation may be beneficial, and the condition can be managed without the need for a surgical airway [8]. Should these methods fail, however, cricothyrotomy or tracheostomy can be used to provide a definitive airway for the patient.

In the absence of airway compromise, the patient's airway should be monitored closely and secured should any deterioration occur. Intravenous broad-spectrum antibiotics must be administered promptly. Options include penicillin combined with beta-lactamase inhibitors, carbapenems, clindamycin and macrolides with metronidazole for penicillin-allergic patients (1). Abscesses should be surgically drained.

The mortality of Ludwig's angina is currently estimated at 0-8.5% [8]. Before antibiotic therapy was widely available, mortality was considerably higher, in the region of over 50% [1].

2. CASE REPORT

We report a case of an 80-year-old gentleman, referred to hospital by his GP with a 12-24 hour history of central chest pain. This pain was constant, pleuritic and associated with sweating and vomiting. He had recently been diagnosed with shingles on the right side of his neck, for which he was taking acyclovir. Patient also complained of increasing neck swelling over the last several days and a cough with white sputum. He was afebrile and denied rigors. His past medical history included nodular sclerosing Hodgkin's lymphoma affecting his neck (treated with local radiotherapy with no recurrence), a urinary bladder tumour for which he was receiving chemotherapy and radiotherapy, and type 2 diabetes mellitus. His medications at the time of presentation included pioglitazone, paroxetine, finasteride, tolterodine and acyclovir.

On examination, he appeared unwell, sweaty and clammy, with an irregularly irregular pulse of 133bpm, respiration rate of 28 per minute, temperature of 36.6 and blood pressure of 122/77. Aside from a swollen and erythematous right neck with two vesicles, the rest of the examination was unremarkable. An ECG showed atrial fibrillation with fast ventricular response. His chest x-ray was normal, but arterial blood gases demonstrated a metabolic acidosis with respiratory compensation.

Blood tests revealed a markedly raised white cell count ($51 \times 10^9/l$), a C-reactive protein of 395 mg/l, and impaired renal function (urea 12.2 mmol/L and creatinine 203 mmol/L). Liver function tests were normal. Blood cultures grew *Streptococcus* species.

Initial treatment for this man consisted of fluid resuscitation and IV amiodarone. IV benzyl penicillin was prescribed, but not administered due to a suspected allergy. In view of his neck swelling, an ENT opinion was sought and patient was transferred to the high dependency unit. As airway started to compromise, the patient was transferred to ENT theatre for emergency intubation under general anaesthetic. Glottic narrowing and displacement was noted during this and a rigid laryngoscope was needed for the procedure.

Empirical treatment with clindamycin, gentamicin and metronidazole was started, along with hydrocortisone, vasopressors and dobutamine.

Despite the intensive therapy received patient continued to deteriorate over the next twelve hours. His metabolic acidosis and renal failure persisted despite renal replacement therapy and developed type 2 respiratory failure and refractory hypotension. Patient died within 24 hours of admission to the intensive care unit.

3. DISCUSSION

Our patient's presentation was unusual for several reasons. His presenting complaint was severe chest pain, which, despite the name of the condition, is not usually associated with Ludwig's angina, although it has been reported previously [9]. This is likely to have been caused by the infection spreading into the mediastinal spaces, which corresponds to our patient who appeared profoundly septic on admission. Despite the presence of multiple "classical" features of cardiac chest pain in the history, this was ruled out by our subsequent investigations. Mediastinal involvement occurs when the infection spreads through the buccopharyngeal gap, a space between the styloglossus muscle and middle and superior constrictor muscles of the pharynx [10] (Fig. 2). This gap provides a conduit between the submandibular and lateral pharyngeal spaces.

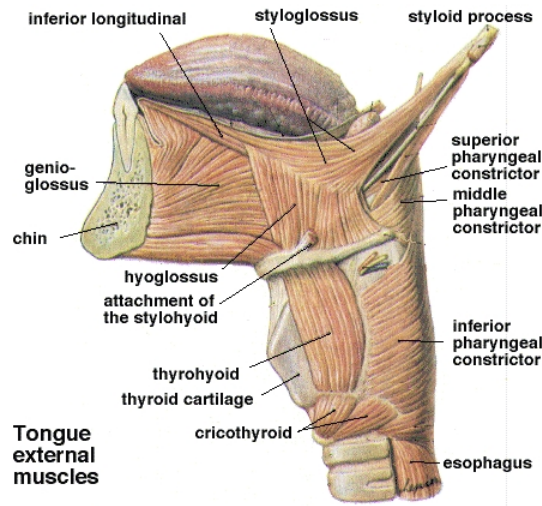


Fig. 2. Infection spreading between the styloglossus and pharyngeal constrictors into the mediastinum can cause presentations similar to our case

There was no history of preceding dental work with our patient but given his history of haematological malignancy and its location and diabetes, he was relatively immunosuppressed and undoubtedly had risk factors for developing the illness. The preceding shingles infection on his neck may have been complicated by a secondary bacterial infection leading to his presentation.

In most cases of Ludwig's angina, the cause of death is airway obstruction. However, in this man's case, his airway was managed with intubation once symptoms developed, and he died of septic shock. In view of the clinical picture, this outcome was not unexpected, but this

case nonetheless highlights the importance of careful airway management and early antibiotic therapy in patients with suspected Ludwig's angina. Furthermore, it serves as a reminder that not all cases of chest pain are cardiac in nature.

4. CONCLUSION

Ludwig's angina is rare and lethal condition but early recognition and multidisciplinary approach involving emergency medical team, anaesthetists, and ENT team for airway management can help save lives. Early intravenous antibiotics and airway support is key in managing such cases.

Also its important to recognize that Ludwig angina can present as chest pain especially if associated with neck swelling inimmuno-suppressed patients.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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