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Historical Article

Did Ludwig's angina kill Ludwig?

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Abstract

Wilhelm Frederick von Ludwig first described in 1836 a potentially fatal, rapidly spreading soft tissue infection of the neck and floor of the mouth. The condition was later named 'Ludwig's angina', a term which persists in medicine to this day. A gold medallist at 19 and professor at 25, Ludwig also served as president of the Württemberg Medical Association and chief physician to the royal family. His outstanding contribution to medicine was rewarded with the title Excellence upon retiring in 1855. Ludwig died at the age of 75, ironically, days after developing an inflammation of the neck. Could it be that Ludwig died of his own condition? This article combines a biography of Wilhelm Frederick von Ludwig with an overview of his eponymous condition and its management.

Key words: Ludwig's Angina; History of Medicine

Introduction

Numerous conditions and syndromes in modern medicine bear the name of the physician who first recognized their characteristic signs and symptoms. All too often, little is known of the history behind these eponyms and of the physician after whom they are named. One such term is Ludwig's angina, a severe and potentially fatal infection of the neck and floor of the mouth. The condition was first described in 1836 by the president of the Württemberg Medical Association, Wilhelm Frederick von Ludwig.

Wilhelm Frederick von Ludwig

The son of a clergyman, Wilhelm Frederick von Ludwig was born in Uhlbach, near Stuttgart, Württemberg, on 16 September 1790. Ludwig became interested in medicine as a teenager and studied medicine, obstetrics and surgery at the University of Tübingen. He proved to be an exceptional student and in 1809, at only 19 years of age, was awarded a gold medal by King Frederick I for the advancement of surgery. Ludwig graduated two years later, receiving his doctorate on 16 July 1811.

Württemberg was an ally of France from 1802 to 1813 and King Frederick I of Württemberg joined Napoleon against Prussia, Austria and Russia. During the 1812 campaign in Russia, Ludwig was called up for military service. He served as physician to the Third Infantry at the Schorndorff garrison and later commanded the Württemberg field hospital at Smolensk. One year on, Ludwig fell ill with typhus and, during the retreat to Vilna, was captured by the Russians and transported to Pensa, Russia. Ludwig recovered and became a personal physician to a Russian countess until he was eventually freed in 1814 after King Frederick I joined the coalition against Napoleon. He returned to Germany to direct a typhus hospital in Hohenheim and, in 1815, completed his military service after accompanying the Württemberg troops to the Rhine.

In the time following his military service, Ludwig made a five-month tour of the surgical and obstetrical clinics in München, Salzburg, Wien, Erlangin, Bamberg and Würzburg. He later arrived at Tübingen to direct the obstetric and surgical clinic and there was granted professorship of surgery and obstetrics in June 1815. Ludwig set about purchasing new materials for the Tübingen clinic, funded by his own personal salary, but money soon dried up. He petitioned King Frederick I for an increase in salary to improve his clinic, and his wish was granted by appointment as one of the king's personal physicians. Ludwig continued to direct the Tübingen clinic until a successor was appointed in 1817. In May 1817, Ludwig returned to Stuttgart to serve as personal physician to King William I, son of and successor to King Frederick I, and was quickly promoted to chief physician for the royal family.

Between 1835 and 1846, Ludwig served as director of the medical college, president of the Württemberg Medical Association and chairman of the medical section at the first Stuttgart scientific congress. It was in 1836, as president of the Württemberg medical association, that Ludwig presented a paper to colleagues describing a fast-spreading, potentially

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fatal gangrenous induration of the connective tissues of the neck and floor of the mouth. Ludwig outlined specific characteristics: a peculiar hardness of the involved tissue; hard swelling beneath the tongue; well defined, firm oedema of the neck; and absence of glandular involvement. He observed that the condition progressively worsened, with death occurring within 10 to 12 days.[1] The paper was published untitled in the medical association's journal. It was editorials appearing in the *Gazette Médicale de Paris* and *Schmidt's Jahrbücher*, two prominent publications at that time, that spread word of Ludwig's condition.[2–4] One year later, a colleague suggested that this condition be named 'angina Ludovici' (Ludwig's angina), derived from the Latin 'angere', meaning 'to strangle'. The eponym persists in medicine to this day.

Ludwig retired in 1855, honoured with the title Excellence and a painting of his portrait. Retirement was unfortunately fraught with illness. Ludwig developed a cataract, and in his seventies a bladder stone was removed. Months later, Ludwig died at the age of 75; ironically, the fatal illness he had earlier described was possibly his cause of death. It is claimed that, on 7 December 1865, Ludwig developed an inflammation of the neck and, on the morning of 14 December 1865, he died suddenly. However, his obituaries did not state that Ludwig died of Ludwig's angina.

Ludwig left his remaining fortune to found a hospital for the poor in Württemberg, which was opened in 1874. Originally built for 50 patients, this hospital was later greatly expanded.[1]

Ludwig's angina and its management

Ludwig's angina is a severe, rapidly spreading cellulitis involving the submandibular, sublingual and submental spaces. It is uncommon but of clinical importance due to its potential for airway obstruction and death by asphyxiation. Aetiology is odontogenic in 85 per cent of cases.[5] Less common causes include: peritonsillar or parapharyngeal abscess; sialadenitis; epiglottitis; and penetrating injuries to the floor of the mouth.[5–7]

The most common pathogens involved are alpha haemolytic streptococci, staphylococci and bacteroides, and patients with an impaired immune function are at a greater risk of developing the condition.[6,8]

To understand the pathogenesis of Ludwig's angina, one must appreciate the anatomy of the submandibular space. It is bounded above by the mucous membrane of the floor of the mouth and tongue and below by the superficial layer of the deep cervical fascia, which extends from the hyoid to the mandible. The submandibular space is divided into two spaces by the mylohyoid muscle: the sublingual space superiorly and the submaxillary space inferiorly. The apices of the second and third lower molars extend below the mandibular insertion of the mylohyoid muscle. Periapical dental abscesses of these molars penetrate the thin inner cortex of the mandible and infection of the submaxillary space ensues. Communication around the posterior margin of the mylohyoid muscle leads to rapid involvement of the sublingual space. With the unyielding presence of the mandible, hyoid and cervical fascia, cellulitis and swelling displace the tongue superiorly and posteriorly, culminating in airway obstruction and asphyxiation if left untreated.[6,9]

Symptoms include toothache, mouth pain, neck swelling, fever and malaise. In more advanced cases, patients can experience odynophagia, dysphagia and drooling.[5,10] Classical signs are fever, tachycardia and a characteristic brawny, tender induration of the submandibular space, with elevation of the tongue. Associated carious molar teeth suggest an odontogenic source of infection, and trismus indicates direct irritation of the masticatory muscles. If a patient develops dyspnoea, tachypnoea, inspiratory stridor and cyanosis, these are indicative of progressive supraglottic oedema which, if untreated, will precipitate airway occlusion and death.[5,10,11] Other complications of Ludwig's angina include mediastinitis, subphrenic abscess, pleural effusion, empyema, osteomyelitis of the mandible and infection of the carotid sheath, with possible rupture of the carotid artery.[11]

The diagnosis of Ludwig's angina is essentially a clinical one and, given the potentially fatal consequences of the disease, investigations should not delay treatment. However, imaging is useful to gauge the extent and severity of infection. Plain radiographs of the neck and chest may demonstrate the extent of soft tissue swelling and would suggest anaerobic infection if gas is present in the soft tissues.[7,11] Ultrasonography will identify collections of pus and any metastatic abscess formation.[11] Computed tomography (CT) is excellent in evaluating deep neck and mediastinal collections. Magnetic resonance imaging produces higher resolution images but imaging time is longer, so CT is the investigation of choice.[12]

Treatment must be prompt and involves three steps. Most important is assessment and maintenance of a patent airway. The second step is aggressive antibiotic therapy and the third is surgical evaluation and, if required, operative decompression.[10] Airway management options include observation, fibre-optic nasotracheal intubation and tracheostomy under local anaesthetic. Although essential, the method and timing of airway management is a matter of ongoing debate. Observation is acceptable in less advanced infections with no evidence of airway compromise.[13] Awake fibre-optic nasotracheal intubation is widely recommended as a first-line approach in early upper airway compromise. Under no circumstances should blind nasotracheal intubation be attempted as it risks damage to the swollen pharyngeal mucosa and may precipitate complete airway obstruction.[14] In the emergency scenario of severe submandibular swelling, trismus and airway compromise, tracheostomy should be performed to provide the patient with a definitive airway.[11,14] The use of intravenous dexamethasone, given for 48 hours, has been shown to reduce oedema and enable intubation to be carried out under more controlled conditions. Steroids also enhance antibiotic penetration.[6]

The introduction of antibiotics in the 1940s has significantly decreased the incidence and mortality of Ludwig's angina.[5] Penicillin is the first-line antibiotic of choice, targetting gram-positive cocci. In patients sensitive to penicillin, clindamycin is recommended as a substitute. With the increasing prevalence of beta-lactamase-producing bacteroides species, metronidazole should be used in combination with penicillin.[10,11] In all patients, bacteriological investigations must be requested and antibiotic regimes adjusted according to the culture and sensitivities.

Surgical decompression of the submandibular space is indicated as an early procedure in suppurative infection or if there is no clinical improvement within the first 24 hours.[10,11] Absolute indications include fluctuance, crepitus, soft tissue air and the presence of a purulent needle aspirate. The goal of surgical drainage is the evacuation of pus and decompression of all closed fascial spaces of the neck. If an infected lower molar is present, it must be extracted to ensure complete decompression and drainage.[11]

Conclusion

In the pre-antibiotic era, Ludwig's angina carried a mortality rate in excess of 50 per cent.[1] Today, aggressive management with respect to airway protection, intravenous antibiotics and surgical intervention has dramatically reduced mortality to 4 per cent.[15] However, credit must be given to Wilhelm Frederick von Ludwig, whose recognition back in 1836 of a potentially fatal condition improved its subsequent diagnosis and treatment.

- **Ludwig's angina is a potentially fatal, rapidly spreading cellulitis involving the submandibular, sublingual and submental spaces**
- **The condition was first described in 1836 by Wilhelm Frederick von Ludwig, who, ironically, died at 75 after developing an inflammation of the neck**
- **It is possible that Ludwig died of Ludwig's angina**

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