Ludwig angina

Marina Kobayashi MD, Kenta Watanabe MD PhD

■ Cite as: *CMAJ* 2017 February 13;189:E246. doi: 10.1503/cmaj.160279

A previously healthy 28-year-old man presented to the hospital with a sore throat and neck swelling that had progressively worsened over the previous three days. He also had difficulty with opening his mouth, moving his tongue and swallowing. There was firm swelling in the right oral floor (Figure 1) that was spreading to the caudal side of the tongue, lateral wall of the mesopharynx and submandibular area. Fibre optic laryngoscopy showed laryngeal edema. The patient had no caries and reported no recent history of dental care. Although he had a body temperature of 38.9°C, an elevated leukocyte count (19.9 [normal 3.4–9.2] × 10^9/L) and an elevated serum C-reactive protein level (2333.38 [normal ≤ 28.57] nmol/L), his general condition was stable enough to tolerate radiologic examinations. Computed tomography (CT) showed air locules along the Wharton duct in the oral floor accompanied by sialolithiasis at the boundary between the duct and the right submandibular gland (Appendix 1, available at www.cmaj.ca/lookup/suppl/doi:10.1503/cmaj.160279/-/DC1). We diagnosed Ludwig angina.

Figure 1: Swelling in the right oral floor of a 28-year-old man with Ludwig angina.

The patient was admitted to hospital and underwent surgical incision, lavage of the tissue of the oral floor and tracheostomy on the same day. Malodorous purulent discharge was drained from the incision site, and several sialolith fragments were extirpated (Appendix 1). α-Hemolytic *Streptococcus*, *Staphylococcus* species, *Neisseria* species, *Hemophilus parainfluenzae,* and anaerobic gram-positive and gram-negative bacilli were detected in cultures of the discharge.

The patient recovered and was discharged two weeks later after receiving intravenous antibiotics and closure of the tracheostoma. At a follow-up visit three months later, he was in good condition, and no evidence of sialolith residue was found.

Ludwig angina is a bacterial cellulitis at the oral floor that rapidly spreads to the adjacent structures beyond the mylohyoid muscle.[1,2] It requires prompt treatment with airway management, intravenous antibiotics and, on occasion, surgical drainage.[2,3] Airway obstruction may occur because of edema of the suprahyoid tissue.[3]

Mortality caused by Ludwig angina was greater than 50% in the preantibiotic era.[1] The patient should be referred urgently to a head and neck specialist when Ludwig angina is diagnosed.[4] If the patient is sufficiently stable to allow radiologic investigations, CT is useful to evaluate deep neck and mediastinal conditions.[5]

The most common cause of Ludwig angina is infection in odontogenic tissue.[1,3] In this case, however, we presumed that the condition originated from submandibular sialadenitis caused by sialolithiasis.

References

1. Kremer MJ, Blair T. Ludwig angina: forewarned is forearmed. *AANA J* 2006; 74:445-51.
2. Hartmann RW Jr. Ludwig's angina in children. *Am Fam Physician* 1999;60: 109-12.
3. Moreland LW, Corey J, McKenzie R. Ludwig's angina. Report of a case and review of the literature. *Arch Intern Med* 1988;148:461-6.
4. Costain N, Marrie TJ. Ludwig's angina. *Am J Med* 2011;124:115-7.
5. Wasson J, Hopkins C, Bowdler D. Did Ludwig's angina kill Ludwig? *J Laryngol Otol* 2006;120:363-5.

Competing interests: None declared.

This article has been peer reviewed.

The authors have obtained patient consent.

Affiliation: Department of Otolaryngology, Toranomon Hospital, Tokyo, Japan

Correspondence to: Marina Kobayashi, marinakobayashi@nms.ac.jp