

Letter to the Editor: Role of Early Imaging in a Case of Ludwig's Angina

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Dear Editor,

Ludwig's angina is a rapidly progressive deep neck space infection with a high risk of airway compromise [1].

We present a case highlighting the importance of early imaging, airway assessment and appropriate intervention. A 68-year-old female presented with swelling below the chin [Table/Fig-1] for five days, which was insidious in onset and progressively increasing in size, associated with pain, difficulty in mouth opening (trismus), dysphagia, and odynophagia. She was a known case of diabetes mellitus, hypertension, and rheumatoid arthritis, and was on anti-tubercular therapy for the past nine months.



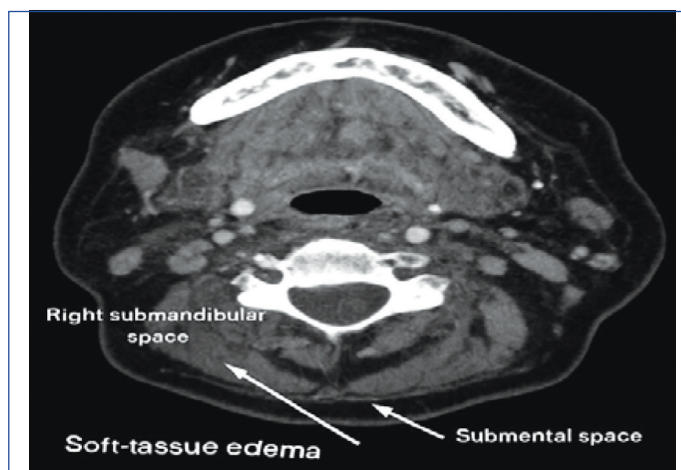
[Table/Fig-1]: Clinical photograph showing diffuse swelling in the submental region.

On examination, the patient was febrile (38.5°C), tachycardic (140 beats/min) normotensive (Blood pressure: 110/80 mmHg), and maintaining an oxygen saturation of 99% on room air. Local examination revealed diffuse, tender, indurated swelling in the submental and submandibular regions. Oral examination revealed multiple carious teeth, primarily involving the bilateral mandibular second and third molars, suggesting an odontogenic source of infection. Elevation of the floor of the mouth and restricted tongue mobility were noted. Although no stridor was present, clinical features suggested impending airway compromise.

Laboratory investigations revealed elevated total leukocyte count and elevated inflammatory markers, indicative of an acute infectious process. Blood cultures were obtained prior to initiation of antibiotic therapy; however, no growth was observed.

Contrast-enhanced CT of the neck demonstrated diffuse soft-tissue oedema involving bilateral submandibular (right > left), submental, and right sublingual spaces with associated fascial plane involvement and airway narrowing, consistent with Ludwig's angina [Table/Fig-2,3].

The patient was started on empirical broad-spectrum intravenous antibiotics (piperacillin-tazobactam combined with metronidazole). Airway status was closely monitored throughout hospitalisation, and no immediate airway intervention was necessary.



[Table/Fig-2]: Axial contrast-enhanced CT Neck: soft-tissue oedema involving the right submandibular and submental spaces with diffuse fat stranding, consistent with early Ludwig's angina. No drainable abscess or gas formation is noted.



[Table/Fig-3]: Sagittal contrast-enhanced CT neck: sagittal CT shows vertical extension of inflammatory oedema into the anterior cervical soft-tissues with effacement of the fascial planes of the floor of the mouth. Upper airway remains patent but narrowed due to soft-tissue swelling.

Surgical incision and drainage were performed within 24 hours of admission under general anaesthesia. Culture from the drained pus revealed polymicrobial growth, predominantly viridans streptococci and anaerobic organisms, consistent with an odontogenic source of infection. Postoperatively, the patient demonstrated gradual clinical improvement with a reduction in neck swelling and resolution of symptoms. She was transitioned to oral antibiotics after clinical stabilisation and discharged on the 8th day of hospitalisation in a stable condition.

Ludwig angina most commonly originates from dental infections involving the mandibular molars, particularly the second and third molars, which account for 90% of cases [1]. Infection is often polymicrobial, with *Streptococcus pyogenes*, Peptostreptococci, and Bacteroides being the most commonly implicated organisms [2].

In a case report by Candamourty R et al., a 25-year-old male presented with trismus, mandibular pain, neck swelling, fever (101.8°F), and respiratory distress. Examination revealed a grossly decayed lower right second molar with pus discharge and bilateral indurated submandibular and sublingual space swelling, consistent with Ludwig's angina. The patient received intravenous cefotaxime, gentamicin, metronidazole, and short-course dexamethasone. Drains and the infected tooth were removed after 36 hours. Postoperative recovery was uneventful with satisfactory clinical improvement [3]. Our case similarly established successful management without airway intervention, emphasising individualised clinical assessment.

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