No Tooth Sleuth: A Common Cause of Facial Swelling with an Unusual History
TaReva Warrick-Stone DO and Darragh Cullen DO
Department of Emergency Medicine, Jefferson Health - Northeast

Chief Complaint

Facial Swelling

History of Present Illness

An 86-year-old female with history of DVT (on Eliquis), HTN, HLD, osteoporosis (on alendronate) and IDDM, presented to the ED from home for evaluation of facial swelling and pain worsening over 2 days. She reported that she woke up yesterday and noticed a small amount of swelling of the right cheek and chin, which had quickly progressed to a very large area of painful swelling to the right side of her face making it difficult to speak or eat. She denied any trauma to the area and denied recent dental work. She denied any drainage or foul taste. She reported no similar instances to this in the past. She wears full upper and lower dentures because all her teeth were removed several years ago. She admits to some limited ability to open her mouth but does not report any difficulty swallowing or breathing. She admits to loss of appetite and intermittent episodes of nausea but states she has been able to drink liquids without difficulty and denies fevers, chills, or vomiting.

Pertinent Physical Findings

Vital signs: Temp(F) 97.9 deg, Temp(C) 36.6 deg, HR 84 bpm, RR 18 bpm, BP 153/65, SPO2 96 %, FIO2 room air.

Physical exam: large focal swelling and induration to the right mandible extending into the submandibular region, firm to touch, painful to palpation, warm, erythematous, no trismus, normal phonation. Edentulous, floor of mouth soft with questionable right sublingual elevation causing leftward deviation, multiple small hemorrhagic bullae to the oral mucosa, frank spontaneous purulent drainage from the right sublingual area, posterior oropharynx without edema or erythema.

Pertinent Laboratory Data

Glucose 109 (mg/dL)
WBC 15.0 (K/UL)
HGB 10.7 (g/dL)
Lactic Acid 0.9 (mmol/L)

Questions

1. What is the most likely cause of this patient’s facial swelling?  
2. If this diagnosis is not addressed, what dangerous complication could develop?

Pearls

- Recognizing potential airway emergencies is a necessary skill for the emergency medicine physician, one of which is Ludwig’s Angina.  
- Ludwig’s Angina is a polymicrobial cellulitis of the submandibular space that is often caused by a tooth abscess, usually of the mandibular molars, including partially erupted ones as in this patient. Usually seen in people with poor dentition, patients present with swelling and pain in the submandibular area and in the floor of the mouth under the tongue, but can include trismus, odynophagia, dysphagia, and dysarthria.  
- Other, less common, etiologies include injury or laceration to the floor of the mouth, mandible fracture, tongue injury, oral piercing, osteomyelitis, peritonsillar abscess, submandibular sialadenitis, and infected thyroglossal duct cysts. Predisposing factors include diabetes, oral malignancy, dental caries, alcoholism, malnutrition, and immunocompromised status.  
- The infection is rapidly progressive leading to airway obstruction. A clinical diagnosis should be made based on the presentation. If Ludwig’s angina is diagnosed, the patient should be electively intubated immediately. The safest manner to secure the airway is via awake fiberoptic intubation. CT scan of the soft tissue of the neck with IV contrast can be used to evaluate the severity of the infection after securing the airway.  
- Early broad-spectrum IV antibiotics are essential to management. Antibiotics should cover gram-positive bacteria, gram-negative bacteria, and anaerobes. Ampicillin/sulbactam or clindamycin are reasonable choices for immunocompetent patients, broadening to cover pseudomonas in immunocompromised patients. IV steroids are controversial, though several case reports have shown a decrease in the need for airway management with the use of steroids.

Case Discussion

Answers:
1. Cellulitis and submandibular abscess.  
2. Ludwig’s angina.

While the patient’s cellulitis and large submandibular abscess were evident, the underlying cause of the abscess was unclear. Our differential included obstructive sialolithiasis, osteonecrosis of the jaw due to her use of a bisphosphonate, and periapical disease, though this seemed unlikely as the patient reported having all teeth removed. Submandibular abscesses can range from mildly symptomatic to airway compromising. In rapidly progressing cases such as this, the presentation can mimic emergent oropharyngeal processes, primarily Ludwig’s angina. The patient was quickly evaluated at bedside by the resident and attending and her airway was confirmed to be patent. However, due to risk of progressive swelling, she was given IV dexamethasone in addition to IV ampicillin/sulbactam for polymicrobial coverage and fentanyl for pain control prior to pursuing CT imaging.

CT imaging of the neck with IV contrast showed that the patient was almost edentulous. There was a single unerupted right mandibular molar with a small rim of lucency and a large fluid collection (5.3 x 3.6 x 4.0 cm) abutting the mandible on the right.

OMFS evaluated the patient at bedside and found that although the impacted tooth (#32) was not clinically visible due to macroglossia and active purulent drainage, on palpation there appeared to be a defect in the soft tissue overlying the site. The patient was admitted for continued antibiotics, as well as incision, drainage and extraction of tooth in the operating room with general anesthesia the following day. The patient did well post-operatively and the drains were removed on post-op day #2. Surgical cultures grew back normal skin flora and the patient was transitioned to amoxicillin/clavulanic acid upon discharge on post-op day #5 to complete 10 total days of antibiotics.