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Chew on This: Emergency Imaging of the Oral Cavity and Salivary Glands—What the Clinician Really Needs to Know

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Nontraumatic emergencies of the oral cavity, sublingual and submandibular spaces, and salivary glands are common and those requiring imaging will most often be infectious in nature.[1,2] However, noninfectious pathologies such as sialolithiasis, autoimmune sialoadenitis, and soft tissue swelling due to angioedema or hemorrhage are also important conditions that radiologists must be familiar with in order to inform the clinician of critical imaging findings that can have a profound impact on patient outcomes. To understand these pathologies, familiarity with the anatomy of these spaces is essential.
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Relevant Anatomic Considerations

The oral cavity extends from the lips anteriorly, to the circumvallate papillae and anterior tonsillar pillars posteriorly, and to the hard palate superiorly. Inferiorly, the hammock-like mylohyoid muscle divides the sublingual space, part of the oral cavity, from the submandibular space. The major salivary glands consist of the sublingual, submandibular, and parotid glands within their respectively named spaces. The parotid and submandibular spaces are enclosed by reflections of the superficial layer of the deep cervical fascia. The sublingual space extends from the mylohyoid laterally to the geniohyoid/genioglossus complex medially and communicates across midline via a narrow isthmus anteriorly. The submandibular and sublingual spaces also communicate posteriorly at the free-margin of the mylohyoid, where a small lip of the submandibular gland often bridges the 2 spaces.

The submandibular (Wharton's) duct is contained within the sublingual space, coursing between the mylohyoid and hyoglossus muscles to its orifice anteriorly under the tongue. The parotid (Stensen's) duct passes through the subcutaneous tissue of the cheek to pierce the buccinator muscle and enter the oral cavity near the second maxillary molar. The sublingual glands drain into the floor of mouth through approximately 20 short independent ducts.

Multiple intraglandular lymph nodes are encapsulated within the parotid gland during development, but no lymph are nodes normally found in the submandibular or sublingual glands. There are multiple lymph nodes outside of the glands in the submandibular space.

Surgical Implications/Pearls[3]

- Fascial planes contain abscesses and infections within compartments, but also form routes of spread once breeched
  - Sublingual space infections may spread to the submandibular space through the mylohyoid-hyoglossus cleft
  - Submandibular space abscesses can spread to the parapharyngeal space and subsequently, retropharyngeal space

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Oral Cavity and Submandibular Space Infection and Abscess

Infections and abscesses in the oral cavity and submandibular space are most often secondary to dental infection or infection of the salivary ducts/glands.[4] Rarely infections in the oral cavity and submandibular space can arise from hematologic seeding (such as in intravenous drug users), suppurative submandibular lymph nodes, or trauma (such as lacerations of the tongue and oral mucosa). Patients with infections in the oral cavity, sublingual space, and/or submandibular space can present with symptoms of pain, swelling, poor control of secretions, dysphagia, and occasionally systemic symptoms. Much of the excitement surrounding infection in these spaces surrounds the possibility of tissue swelling leading to acute airway compromise. Not all infections require imaging, but in cases of suspected abscess or deep spread of infection cross sectional imaging is critical for diagnosis and management decisions.

In adults, contrast-enhanced CT is the modality of choice due to its accessibility, speed of scanning and proficiency in delineating abscess formation, bone erosion, and calculi.[1] Metal artifact from dental amalgam can significantly limit CT assessment in this region. If possible, tilting the gantry and rescanning can help. MRI is also susceptible to metal artifact, but coronal plane sequences are often useful. In adults with contraindications to iodinated contrast and in children it is reasonable to consider MRI as a first modality. Nuclear scintigraphy has been successful for troubleshooting in difficult cases but is rarely used. An abscess will appear as a single or multi-loculated area that is centrally hypodense on CT, and T2 hyperintense, T1 hypointense and diffusion restricting on MRI. Both modalities will demonstrate peripheral rim enhancement. Edema manifesting as fat stranding on CT and T2 hyperintensity on fat suppressed MRI is usually evident in the surrounding tissues. Regional lymph node enlargement is common.

Surgical Implications/Pearls[5]

- Presence of gas should alert clinicians to the possibility of necrotizing fasciitis, anaerobic or spore-forming (eg, Clostridium) infections which require more urgent surgical management and appropriate antibiotic therapy

Odontogenic Infection

Amongst adults dental infection is the most common cause of abscess in the head and neck.[4] During 2011 in the US diseases of the teeth and supporting structures led to approximately 2 million emergency room visits amongst 15-64-years old patients, comprising around 1% of all emergency room visits.[6]

Odontogenic infection often arises from a carious lesion of the dental enamel extending through the pulp to form a periapical cyst or abscess at the apex of the tooth. Alternatively, infection can arise from gingival disease surrounding the tooth and extend along the periodontal ligaments toward the tooth apex. In either case, osteomyelitis may develop in the bone surrounding the tooth and infection can perforate the bone to form a soft tissue abscess. In its earliest form the abscess will be subperiosteal. However, if it remains untreated, further extension into the oral cavity, submandibular space, and/or deep neck can occur.

Abscesses in the oral cavity/sublingual space and submandibular space require close inspection of the teeth with a CT window width sufficient to delineate the enamel, dentin, and pulp (Fig. 1). On many image viewing software systems the optimal windows are greater in width than default bone windows. When examining the teeth at CT, it is important to look for defects in the enamel (caries) and hypodensity extending through the pulp. Carious teeth should be examined closely for periapical lucency, which in the setting of a diseased symptomatic tooth is suggestive of periapical abscess. A perforation of the mandibular/maxillary cortex between a periapical abscess and an adjacent peripherally enhancing soft tissue collection is usually diagnostic of an odontogenic source of abscess (Fig. 2).

Infection tends to perforate the bone where it is thinnest. Anteriorly this is usually at buccal margin for both the mandible and maxilla. Posteriorly this is usually at the buccal margin for the maxilla and lingual margin for the mandible.[2] Some authors also believe that infection

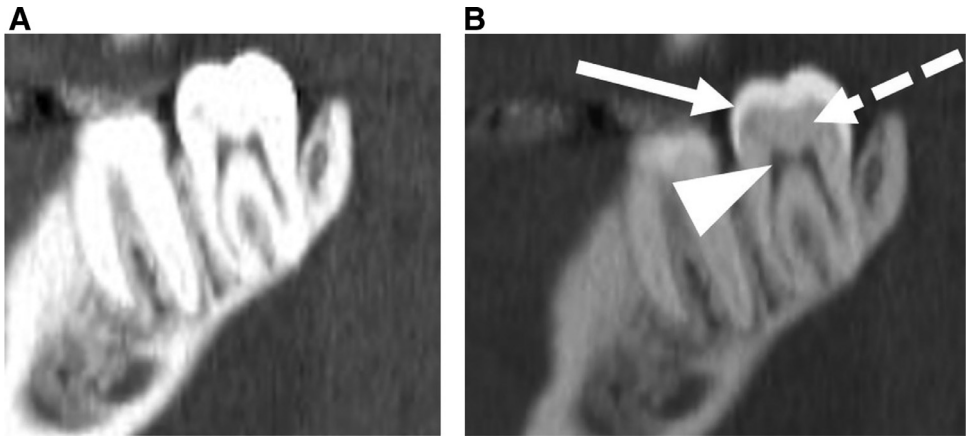

Figure 1 Evaluation of the teeth on CT. Default bone windows (A) vs bone windows for evaluation of teeth (B). Enamel (solid arrow), dentin (broken arrow) and pulp (arrowhead) are better demonstrated with the greater window width (B).

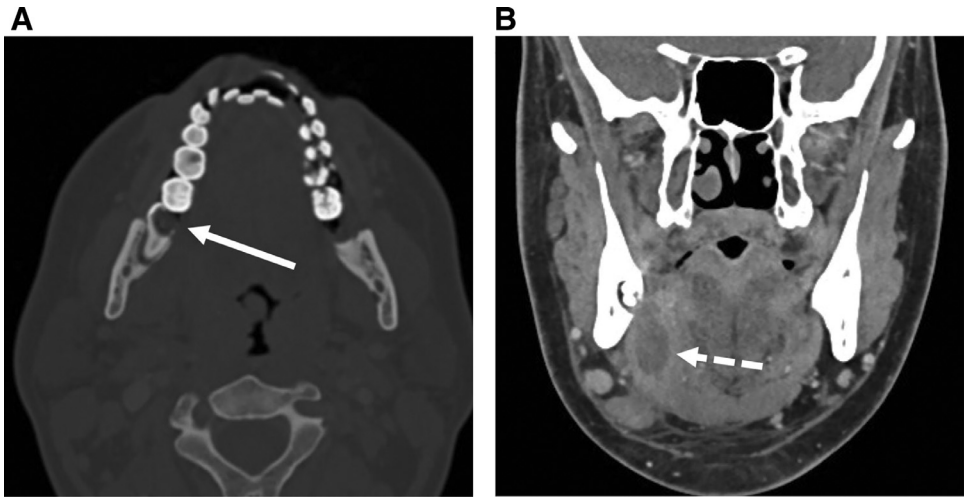

Figure 2 A 26-year-old man presenting with mouth pain and trismus. Axial CT image in bone algorithm (A) demonstrates a large carious lesion in the crown of the right third mandibular molar (solid arrow). Coronal CT image with intravenous contrast in soft tissue algorithm (B) demonstrates a periperhally enhancing abscess extends from the infected tooth into the adjacent mylohoid muscle (broken arrow), at the border of the submandibular and sublingual spaces.

perforating the buccal plate outside the attachment of the buccinator will extend extraorally while perforation inside the buccinator attachment will extend intraorally.[2] Similarly, many sources suggest lingual perforation of infection in the mandible will present in the sublingual space if the apex of the involved teeth lie above the mylohyoid muscle (ie, the premolars and first molar) and in the submandibular space if the apex of the involved teeth lie below the mylohyoid muscle (ie, the second and third molars). However, some feel this is not a reliable predictor of the pattern of spread and may be of questionable utility given free communication of the sublingual and submandibular spaces posteriorly.[1]

Treatment of odontogenic infection typically involves extraction of the offending teeth, antibiotics, and surgical irrigation/debridement or supportive care depending on the extent of disease. Odontogenic infection can also extend into the maxillary sinus, resulting in odontogenic sinusitis, which can progress to orbital cellulitis (Fig. 3). Although rare in the current era of antibiotics and surgery, inadequately-treated infection in the oral cavity and submandibular space can extend directly inferiorly into the mediastinum causing suppurative mediastinitis. Rarely, untreated infection can spread superiorly in the masticator space bordering the skull base and result in intracranial infection (Fig. 4).

Surgical Implications/Pearls[3]

- Limited floor of mouth or sublingual space abscesses close to the mesial inferior alveolar ridge are easily addressed at the time of dental extraction via a gingival or mucosal incision
- Infections that have extended inferiorly into the submandibular space, or laterally to the masseteric space will require transcervical incision and drainage
  - Infections limited to the suprahyoid neck can be approached by an incision 2-finger breaths below the mandible
  - Infections involving the infrahyoid neck or retropharyngeal space require a more inferior incision centered over the anterior border of the sternocleidomastoid muscle and may also require combined intra-oral drainage

Ludwig's Angina

Ludwig's angina is a life-threatening infection of the floor of mouth. The classic description is of a rapidly progressive indurated cellulitis originating in the submandibular space, involving the sublingual and submandibular spaces bilaterally, without abscess or lymphadenopathy.[7] On contrast enhanced CT this will appear as soft tissue thickening and fat stranding in the floor of mouth and submandibular space, possibly with areas of complex fluid but without an organized drainable fluid collection (Fig. 5).

In the preantibiotic era, mortality approached 50%, although with modern medical and surgical management mortality is less than 8%.[8] Odontogenic infection is the most common cause of Ludwig's angina, but it can be secondary to peritonsillar abscess, parapharyngeal abscess, epiglottitis, suppurative sialadenitis, or penetrating injuries to the floor of mouth.[9,7] Rapid enlargement of the tongue with displacement of the tongue base posteriorly and superiorly or soft tissue swelling of the pharynx and larynx can prompt urgent surgical intervention due to the potential for deadly airway obstruction.

Surgical Implications/Pearls[3]

- Patients with true Ludwig's angina often require urgent airway management (ie, awake fiberoptic intubation or

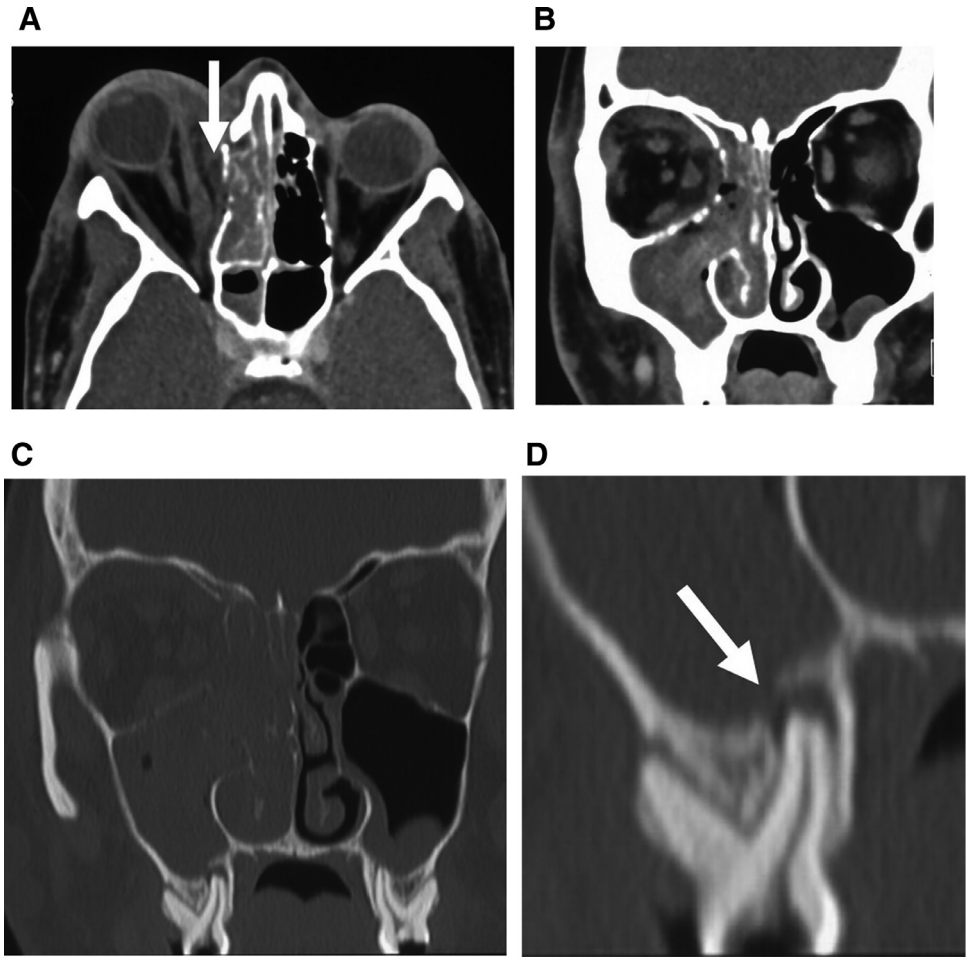

Figure 3 Odontogenic sinusitis and orbital cellulitis. Axial (A) and coronal (B) CT images in soft tissue algorithm demonstrate extensive inflammatory change in the right orbit, with infiltration of the extraconal orbital fat, a small subperiosteal abscess (arrow, A) in the medial extraconal right orbit bordering the ethmoid and marked proptosis with tenting of the posterior globe at the optic nerve (A). Complete opacification of the right paranasal sinuses is also seen (B and C), and this patient was diagnosed with orbital cellulitis secondary to acute sinusitis. However, on bone algorithm coronal images (C and D), the source of the infection becomes apparent, as there is a periapical lucency involving a right maxillary molar that is dehiscent (arrow, D) with the adjacent opacified right maxillary sinus.

awake tracheostomy) which should not be deferred by imaging

- Patients with limited superior mediastinal involvement by abscess can be drained through a transcervical approach. However, patients with low-mediastinal collections or complicated empyema will require thoracic surgery consultation for a combined approach

Acute Sialadenitis

Infection is the most common acute pathology involving the salivary glands and can be bacterial or viral.[1] It typically presents with pain, swelling, and tenderness of the infected gland.

Bacterial infections usually ascend from the oral cavity. Patients with salivary stasis and poor dental hygiene are predisposed to infections of this type. Decreased salivary flow can be caused by severe dehydration, surgery, irradiation, prior infection, ductal obstruction due to stenosis or calculus, and some medications. Acute bacterial/suppurative sialadenitis is more common in the parotid than submandibular glands, likely due to increased incidence of Stensen's duct orifice trauma by cheek biting and dental prostheses, the larger orifice of Stensen's duct, and antibacterial components of mucoid secretions present in higher concentrations in submandibular gland secretions.[10] When involving the submandibular gland, bacterial sialadenitis is usually associated with a calculus.[11] If left untreated, bacterial sialadenitis can lead to abscess formation and/or extend into the deep spaces of the neck.

Mumps is the most common cause of viral sialadenitis and within the general population it is the most common cause of all salivary gland disease.[1] Prevalence of the disease is decreasing in developed countries due to widespread vaccination, but outbreaks still occur. A prodrome of low-grade fever, myalgia, malaise, and anorexia followed by nonsuppurative parotitis characterized by painful gland enlargement is pathognomonic.[12] Bilateral parotid involvement is most common, but the other major salivary glands can also rarely be involved. Diagnosis can be confirmed serologically.

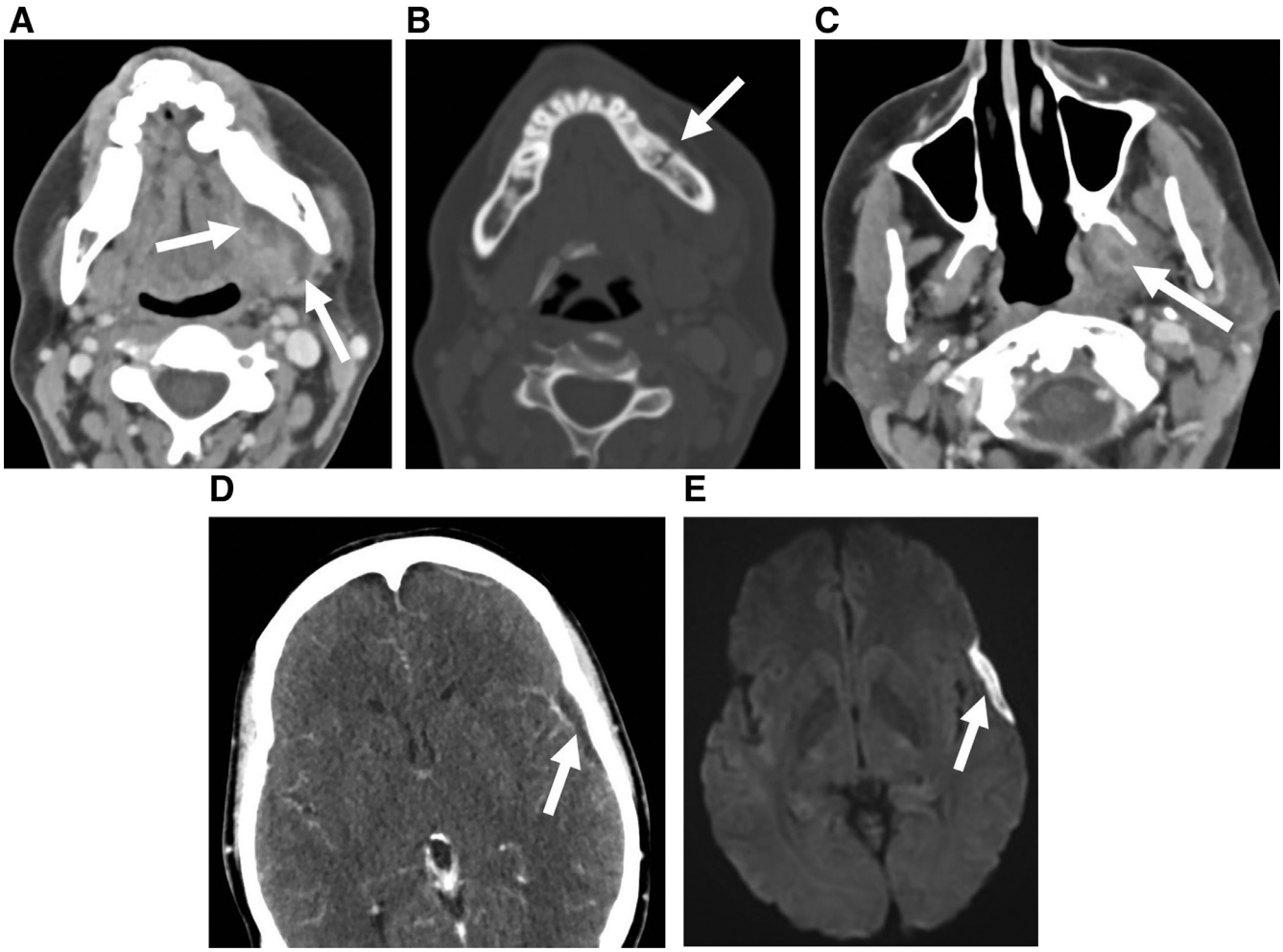

Figure 4 Odontogenic subdural empyema. A 50-year-old woman presented with trismus, left jaw pain and submandibular swelling. Axial CT with intravenous contrast in soft tissue (A) and bone windows (B) demonstrates a rim-enhancing collection along the lingual aspect of the left mandible (arrow, A) adjacent to a periapical lucency involving a left mandibular molar, with periosteal reaction and cortical breakthrough of the adjacent buccal cortex (arrow, B). The infection spread superiorly into the masticator space with a small rim-enhancing abscess involving the left medial pterygoid muscle (arrow, C). The infection ultimately gained access to the intracranial space via the left foramen ovale, and the patient developed a subdural empyema along the left temporal convexity, seen as a hypoattenuating extra-axial collection on the axial contrast enhanced CT (arrow, D), with corresponding restricted diffusion on axial DWI MR imaging (arrow, E).

Mumps may also be subclinical manifesting as otherwise asymptomatic parotid enlargement. Approximately 75% of cases are uncomplicated, but epidymo-orchitis, meningoencephalitis, pancreatitis, and thyroiditis are well-described potential complications. Other viral causes of sialadenitis include but are not limited to: Epstein Barr Virus, cytomegalovirus, and human herpes virus.

Suppurative parotitis of the neonate is a well-described entity that also warrants mention. It usually affects premature infants of 7-14 days of age who are suffering from dehydration and typically presents with parotid swelling and sometimes skin discoloration.

Autoimmune sialadenitis is most commonly from Sjögren's syndrome (SS). SS is an autoimmune disorder of the exocrine glands most commonly affecting the salivary and lacrimal glands. It can occur as an isolated disease (primary SS) or in association with other connective tissue diseases such as lupus (secondary SS). Patients classically experience xerostomia and xeropthalmia, but also commonly have extrasalivary manifestations such as polyarthritis. Although it is a chronic condition characterized by progressive glandular dysfunction, acute episodes of glandular tenderness and/or swelling are common. Therefore, recurrent bilateral sialadenitis or acute sialadenitis in the setting of a connective tissue disorder should raise the possibility of SS. Serologic testing and satisfaction of clinical criteria are required for diagnosis. Fluoroscopic sialography (now rarely performed) or MRI sialography demonstrate an appearance varying from multiple small globular collections in early disease to large collections and central ductal dilatation in advanced disease. Eventually, fatty glandular replacement can occur. SS significantly increases the risk of salivary gland lymphoma.

Other causes of chronic sialadenitis such as chronic sclerosing sialadenitis and juvenile idiopathic sialadenitis are less common but can also present with acute glandular

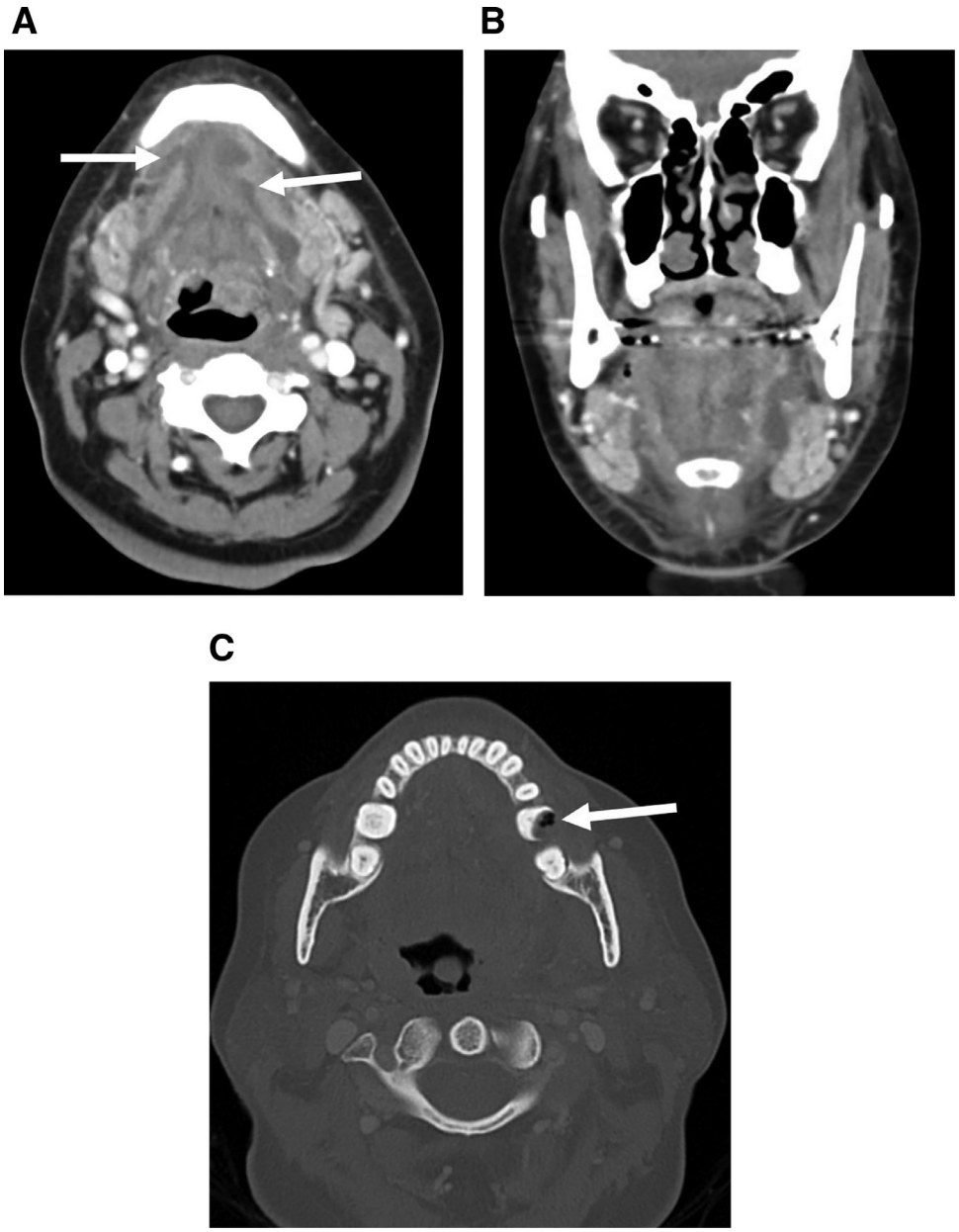

Figure 5 Ludwig's angina. A 62-year-old women with poorly controlled diabetes mellitus and a 3-day history of progressive painful submandibular swelling presented to the emergency room after she developed dysphagia and difficulty breathing. Of note, she had been seen by a dentist for a painful left mandibular molar 1 week prior to presentation and expert consultation had been advised. Axial (A) and coronal (B) contrast-enhanced CT in soft tissue windows demonstrated extensive edema in the floor of mouth and sublingual spaces, as well as extensive inflammatory changes in the submandibular spaces including reticulation of the fat, thickening of the platysma and enlargement and hyperenhancement of both submandibular glands. Fluid was seen throughout the sublingual spaces (arrows, A), without a well-defined rim-enhancing drainable fluid collection. On axial CT in bone windows (C) a grossly carious left mandibular molar is apparent (arrow). The patient was intubated for airway protection and treated with intravenous antibiotics. She ultimately recovered after a prolonged ICU course.

inflammation and pain. The imaging appearance varies widely from diffusely hypoattenuating to hyperattenuating and diffusely enlarged to normal sized or smaller than normal glands.[13]

In adults, contrast-enhanced CT is the favored modality for assessment of acute sialadenitis due to its accessibility, speed of scanning and proficiency in delineating abscess formation, bone erosion, and calculi.[1] Conventional fluoroscopic sialography is contraindicated in cases of acute sialadenitis as retrograde contrast injection can drive infected secretions back into the gland worsening infection. The typical imaging appearance of acute sialadenitis is characterized by enlarged and heterogeneously enhancing glands (Fig. 6) that are often T1 hypointense and T2 hyperintense.[14] Treatment of bacterial sialadenitis will include antibiotics and, if required for abscess control or calculus removal, surgery.

Figure 6 Acute sialadenitis of the submandibular glands. Axial (A) and coronal (B) contrast-enhanced CT in soft tissue windows demonstrates enlargement and heterogeneous enhancement of the glands with surrounding fat stranding (solid arrows). No calculi or ductal dilation was seen. Mumps testing was negative. Although a specific causative organism was not identified, the patient improved with routine antibiotics covering oral flora.

Treatment of autoimmune sialadenitis typically involves supportive therapy and immunotherapy.

Surgical Implications/Pearls[3]

- Treatment for acute sialadenitis is almost always medical and supportive. Clinicians should consider ordering serologic testing for viral etiologies, autoimmune disorders, or sarcoidosis in patients with multi-gland involvement.
- Acute parotitis complicated by abscess formation may require a modified parotidectomy incision and requires care to not injure the facial nerve.

Sialolithiasis

Sialolithiasis is a relatively common condition, with postmortem studies suggesting up to 1.2% prevalence of salivary stones amongst the general population and annual symptomatic incidence of 1 per 10,000-30,000 individuals.[15] Patients with acutely symptomatic sialolithiasis will often present to emergency rooms. Between 80% and90% of salivary stones occur in the submandibular gland, 10% and 20% in the parotid, and only a small number in the sublingual glands. The thicker and higher pH secretions, upward course of the duct, and small orifice are likely reasons for this predilection of the submandibular duct.[1] Strictures such as those developing as sequela of prior trauma, surgery, or infection will predispose to stone formation. Symptomatic stones occur more commonly in the ducts, but small asymptomatic calculi are also often seen within the glands. When stones are obstructing patients typically present with pain and intermittent glandular swelling. Obstruction can lead to recurrent or chronic bacterial infection, duct rupture, and or abscess formation. If left untreated chronic obstruction may eventually lead to gland atrophy.

Most (80%-90%) calculi are thought to be radiopaque,[13] although there is discrepancy in the literature with some sources stating that up to 80% of sialoliths are actually radiolucent.[16] This disagreement may be due inclusion of both calculi and mucus plugging as "sialolithiasis" in some studies. About 20% of radiopaque submandibular stones and 40% of radiopaque parotid stones can be seen on routine radiography, but CT has as much as 10-fold higher sensitivity and is typically the test of choice.[1] CT or MRI can show ductal dilation proximal to the stone as well as hyperenhancement and heterogeneity of the gland indicative of associated sialadenitis (Fig. 7). A peripherally enhancing periductal or intraglandular fluid collection suggests abscess (Fig. 8). Treatment includes either periodic dilation or surgical repair, as well as antibiotics in cases complicated by infection.

Surgical Implications/Pearls

- Distal stones that are greater than 4 mm in diameter are unlikely to pass spontaneously and will usually require transoral extraction.[17]
- Stones that are proximal to the hilum are usually not amenable to endoscopic or transoral approaches and will need to be removed in combination with submandibular gland excision transcervically.[18]
- Stones situated posterior to the first molar, proximal to the "comma area" (where the submandibular duct courses inferiorly at the posterior boarder of the mylohyoid), are difficult to reach transorally and risk injury to the lingual nerve.[3]

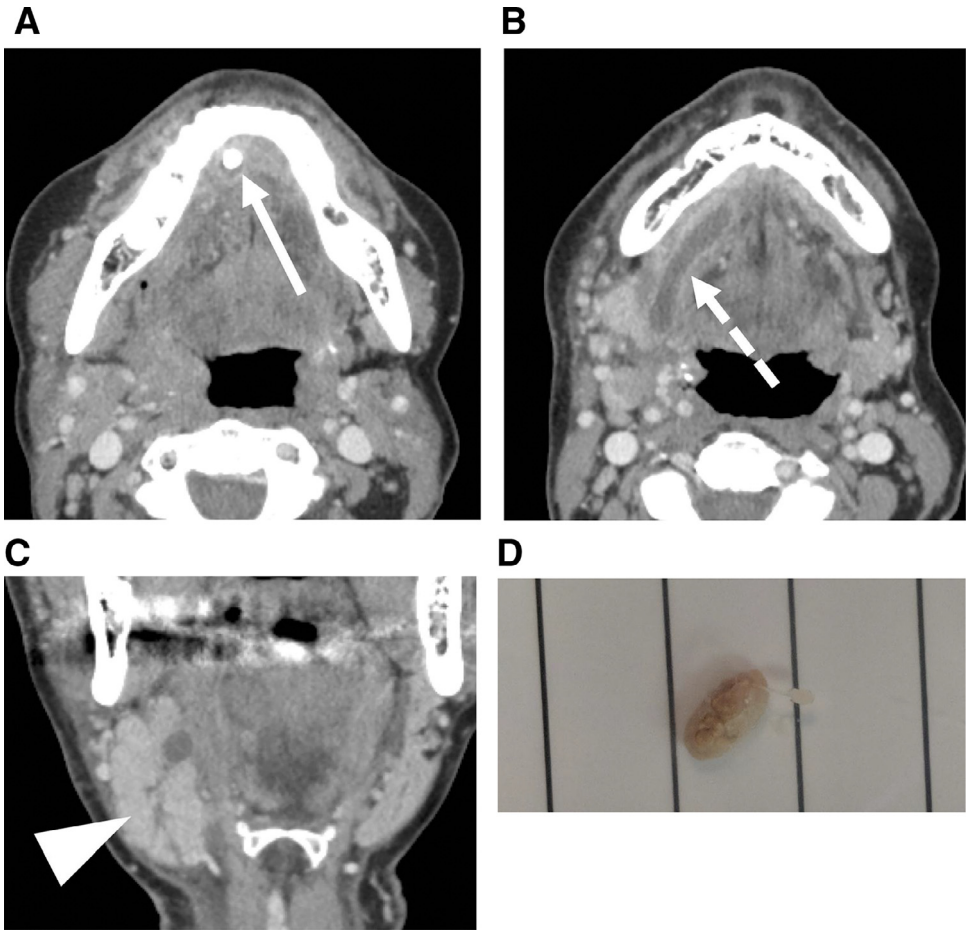

Figure 7 Sialolithiasis with acute sialadenitis. A 71-year-old woman with a known prior history of sialolithiasis presented with new submandibular swelling and pain. Axial contrast-enhanced CT images in soft tissue windows (A and B) demonstrates a calculus within the right submandibular duct just proximal to its orifice under the tongue (solid arrow, A) with upstream dilation of the duct (broken arrow, B). Enlargement and hyperenhancement of the submandibular gland is seen on the coronal contrast-enhanced CT image (arrowhead, C), in keeping with associated sialadenitis. While reviewing the results of the scan with the patient, the calculus expressed spontaneously (D). She experienced immediate relief in symptoms. (Color version of figure is available online.)

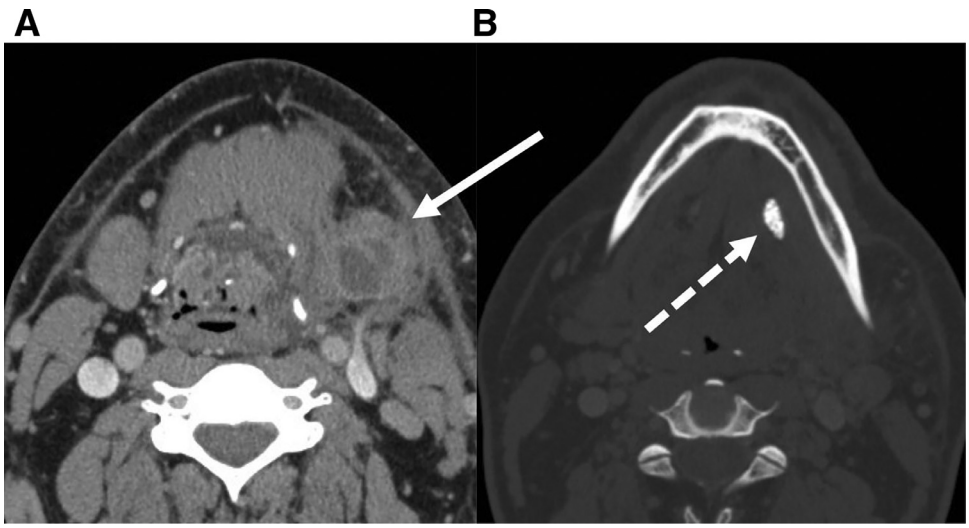

Figure 8 Abscess within the submandibular gland. A 76-year-old man presented with pain and submandibular swelling. Axial contrast-enhanced CT in soft tissue windows (A) demonstrates a peripherally enhancing abscess within the left submandibular gland (arrow). An associated radiopaque calculus is seen on the axial CT image in bone windows (B), lodged within the mid-portion of the submandibular duct (broken arrow).

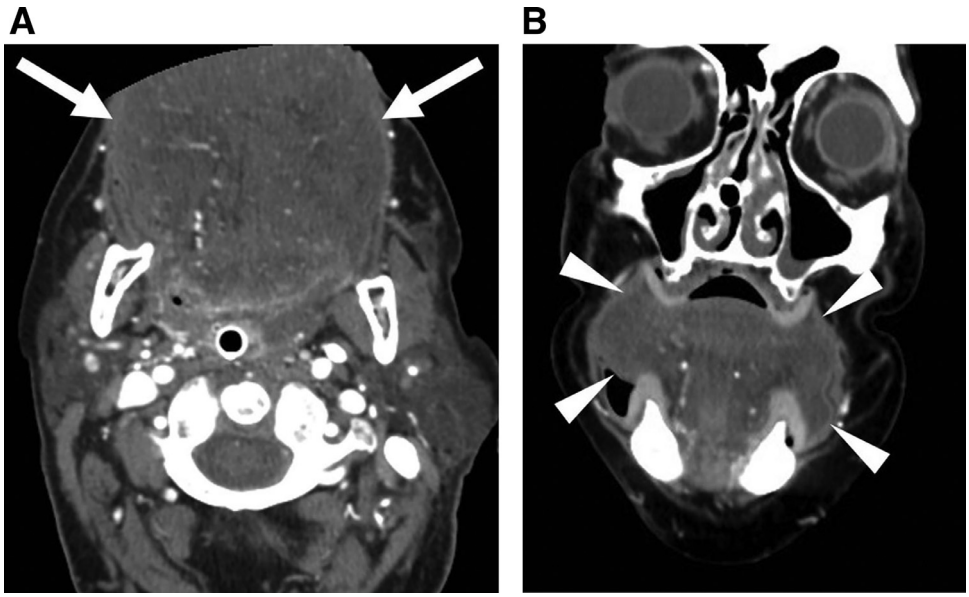

Figure 9 tPA-associated angioedema. An 84-year-old female presented with left hemiplegia. tPA was administered 90 minutes after symptom onset at an outside hospital after acute stroke workup. The patient was transferred for further stroke care and possible intra-arterial therapy and developed acute angioedema while in transit. Fibro-optic intubation was performed. Axial (A) and coronal (B) CT images from the CT angiogram that was performed on arrival demonstrates massive enlargement of the tongue (arrows, A) with diffuse low attenuation consistent with edema. The tongue protruded through the lips (not shown), and protruded laterally, filling the mandibular and maxillary vestibules bilaterally (arrowheads, B).

Other Emergencies: Nontraumatic and Noninfectious Causes of Acute Soft Tissue Swelling

Another important emergency of the oral cavity and submandibular space is that of noninfectious and nontraumatic soft tissue swelling that can lead to life threatening airway compromise. Angioedema and spontaneous hematoma are 2 important causes. It is critical for the radiologist to recognize these potentially life-threatening conditions, although in many cases urgent control of the airway necessitates treatment prior to imaging.

Angioedema is swelling of the subcutaneous and submucosal tissues caused by transient increase in capillary permeability. Patients can present with poor control of secretions, sublingual or submandibular swelling, difficulty swallowing, and/or difficulty breathing. Patients with allergic angioedema (histaminergic) present within an hour of the offending exposure with or without urticaria and respond to antihistamines and steroids. Food and drugs are the most common causes. Drug-related angioedema can also be bradykinin-mediated, classically associated with angiotensin-converting enzyme (ACE) inhibitor use. Bradykinin-mediated angioedema more commonly presents several hours after the exposure, never presents with urticaria,[19] and has symptoms resistant to antihistamines and steroids. The most important treatment for drug induced bradykinin-mediated angioedema is to discontinue the implicated drug. Recently intravenous tissue plasminogen activator (tPA) has been implicated as a cause of orolingual angioedema (Fig. 9), at least some cases of which appear to be bradykinin-mediated with documented increased risk amongst patients also on ACE inhibitors.[20] Hereditary angioedema is a less common condition, most often due to inherited C1 esterase inhibitor deficiency or dysfunction. Cross-sectional imaging will show diffuse soft tissue swelling without discrete fluid collection/abscess commonly involving the oral cavity and pharynx. Diffuse submucosal swelling may also extend into the pharynx and larynx. Laboratory and imaging tests are often not available early enough to guide treatment, but the radiologist should still be familiar with this important condition as he/she may be the first to suggest the diagnosis in complicated cases. Supportive care including intubation or tracheostomy may be required.

Surgical Implications/Pearls

- Patients with angioedema of the supraglottis, oral tongue, or floor of mouth may require urgent airway management (ie, awake fiberoptic intubation or awake tracheostomy) which should not be deferred by imaging.

Oral/sublingual and submandibular soft tissue swelling related to spontaneous hematoma is also well described. Spontaneous sublingual or submandibular hematoma is a rare but well documented complication of anticoagulant use and hypertension.[21,22] It has also been described in the setting of

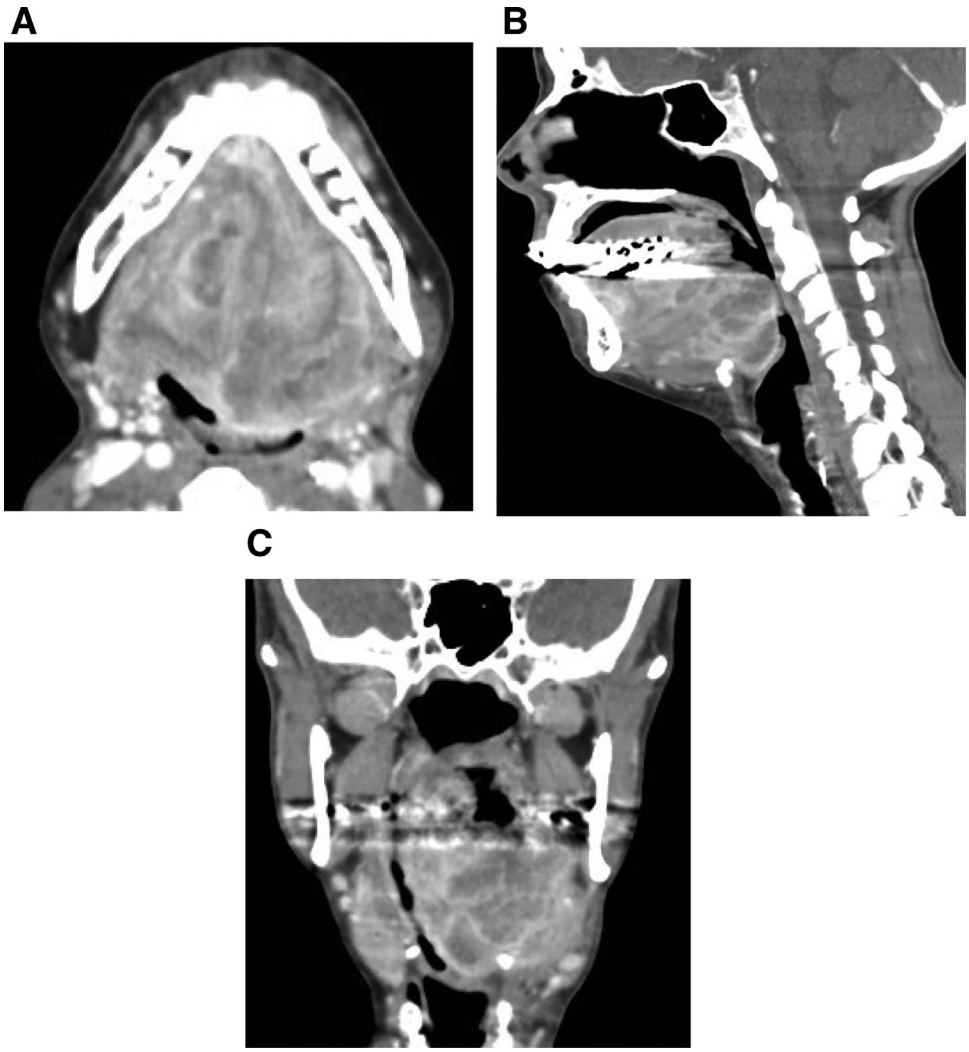

Figure 10 Oral cavity squamous cell carcinoma with hemorrhage resulting in airway compromise. A 64-year-old woman presented with a history of "bit tongue 10 days ago, query abscess." Axial (A), sagittal (B) and coronal (C) contrast-enhanced CT images demonstrate a large enhancing oral cavity mass with areas of central hypoattenuation and complex heterogeneous areas of enhancement and hyperattenuation. Multiple abnormal cervical lymph nodes were also present (not shown). The patient passed away shortly after from airway complications having refused tracheostomy. Biopsies taken before death demonstrated squamous cell carcinoma.

hemophilia[23] and rarely as a presentation of oral cavity carcinoma (Fig. 10). Presentation can be similar to angioedema and sometimes the 2 conditions can be confused.[24] However, discoloration of the affected tissue and hyperattenuating hemorrhage vs more diffuse and low-density edema on CT are important clues to the diagnosis. If imaging is acquired, CT is the preferred test due to its speed of acquisition and accuracy in detecting hemorrhage. The hematoma can spread to other deep spaces of the neck and even to the mediastinum. Experts suggest that if discovered patients should be admitted for observation and reversal of any anticoagulation. Intubation or tracheostomy may be required.[21]

A ranula is another potential cause of soft tissue swelling in the region of the oral cavity. It is a mucocele or mucous retention cyst of the floor of mouth and can be thought of as a saliva-containing pseudocyst, presumably arising from trauma or obstruction of the sublingual gland or minor salivary glands.[1] Most often a ranula presents with painless soft tissue swelling, but it can become secondarily infected leading to tenderness, acute swelling, and other infectious symptoms. Ranulas can be "simple" (confined to the sublingual space) or plunging/diving (extending into the submandibular space along the free margin of the mylohyoid or through a boutonnière defect). On CT and MRI they appear as a thin walled cystic structure that is fluid attenuating (Fig. 11) and T2 hyperintense with no diffusion restriction (if not secondarily infected). Usually there is no associated enhancement, although in cases of superimposed infection peripheral enhancement can occur. A more complex fluid collection with septal or internal enhancement, which may be better characterized on MRI, or with associated phleboliths on CT, suggests the possibility of a lymphatic or vascular malformation. Treatment of ranulas can include surgery and/or antibiotics as needed.

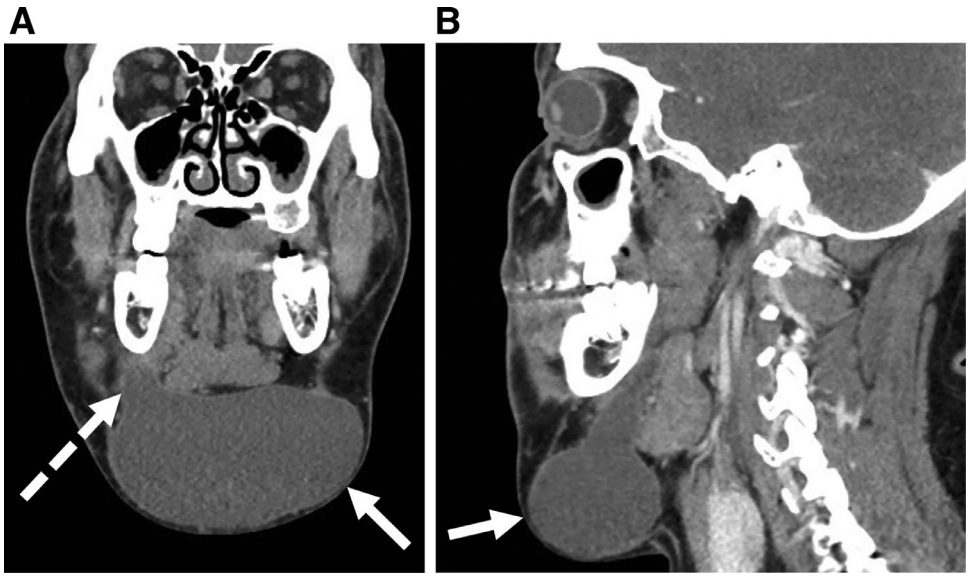

Figure 11 Ranula in the submandibular space. A 28-year-old man presented with submandibular swelling. Coronal (A) and sagittal (B) contrast-enhanced CT images demonstrate a large fluid attenuation lesion in the submandibular space (solid arrows, A and B) with thin, barely perceptible wall. On the coronal image the lesion is seen arising from a portion of the right sublingual gland which has herniated through a defect (or boutonnière) in the mylohoid muscle (broken arrow, A). Note the absence of a thick rim-enhancing wall as well as lack of surrounding inflammatory changes, consistent with a ranula rather than an abscess.

Surgical Implications/Pearls

- Due to drastically different surgical management options for ranulas (isolated floor of mouth or plunging) vs lymphatic or vascular malformations, surgeons rely on radiology input for accurate diagnosis.
- Ranulas that do not pierce the mylohoid and be aspirated transorally or marsupialized in the acute setting. However, recurrence rates are high and definitive surgical therapy usually requires excision of the sublingual gland as well as the pseudocyst.

Summary

Nontraumatic emergencies of the oral cavity, sublingual, and submandibular spaces are a frequent source of emergency room visits and often require imaging for diagnosis and treatment planning. Odontogenic infection is the most common acute pathology in this region. However, infectious and autoimmune sialadenitis, sialolithiasis, and noninfectious causes of soft tissue swelling such as angioedema are also regularly encountered in the emergency setting. Soft tissue swelling in the oral cavity and submandibular region can lead to airway compromise necessitating urgent airway management, which should not be delayed by imaging. Familiarity with the anatomy, as well as the clinical and surgical implications of nontraumatic emergencies in these spaces is important for the radiologist so that the imaging findings that impact timely patient management can be communicated effectively and efficiently.

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