Nontraumatic Head and Neck Emergencies

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Abbreviation: ACE = angiotensin-converting enzyme

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Describe fascial planes and spaces of the neck and other relevant head and neck anatomic landmarks.
- Identify a wide variety of acute infectious and inflammatory diseases of the head and neck and their acute complications.
- Recognize acute complications of head and neck neoplasms that bring patients to the emergency department.

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Head and neck imaging is an intimidating subject for many radiologists because of the complex anatomy and potentially serious consequences of delayed or improper diagnosis of the diverse abnormalities involving this region. The purpose of this article is to help radiologists to understand the intricate anatomy of the head and neck and to review the imaging appearances of a variety of nontraumatic head and neck conditions that bring patients to the emergency department, including acute infectious and inflammatory diseases and acute complications of head and neck neoplasms. These conditions are presented in five sections on the basis of their primary location of involvement: the oral cavity and pharynx, neck, sinonasal tract, orbits, and ears. Important anatomic landmarks are reviewed briefly in each related section.

Online supplemental material is available for this article.

Introduction

Patients present to the emergency department with symptoms of a variety of infectious and inflammatory diseases and with acute complications of neoplasms of the head and neck. Infections are more common than the other types of diseases, and they usually are treated by primary care providers in the absence of serious complications (1). Although complications can occur in otherwise healthy individuals, older patients and those with diabetes or a compromised immune system are at increased risk for complications (1,2). Delayed or improper diagnosis can lead to substantial morbidity or mortality (3–7); therefore, radiologists should be familiar with these conditions and scrutinize all patients for related clinical and imaging findings. Specifically, radiologists should pay attention to airway patency; vascular complications such as thrombosis, pseudoaneurysm formation, and hemorrhage; and spread of diseases to the mediastinum, spine, or intracranial compartments.

In this article, we review the fascial planes and spaces of the neck. The fascial planes are important barriers to the spread of abnormalities. However, once a disease is established in the spaces of the neck, the fascial planes provide specific routes of dissemination of the disease. Although the fascial layers are not seen at imaging, radiologists should be aware of them for proper interpretation of images and detection of potential complications.

We review a variety of acute infectious and inflammatory diseases of the head and neck and potentially life-threatening acute complications of head and neck cancers. The diseases are organized into five sections according to their primary location of involvement: the oral cavity and pharynx, the neck, the sinonasal tract, the orbits, and the ears. Abnormalities of the neck are arranged according to location involved when possible, but they overlap, and sometimes multiple spaces are involved.

TEACHING POINTS

- Above the hyoid bone, the posterior aspect of the middle layer of the deep cervical fascia (or buccopharyngeal fascia) continues to the skull base and covers the pharyngeal constrictor muscles. However, above the carotid bifurcation, the contribution of the middle layer to the carotid sheath is inconsistent; it does not form a well-defined sheath around the internal and external carotid arteries and the internal jugular vein.
- The retropharyngeal space extends from the skull base to the thoracocervical junction as the alar fascia attaches to the buccopharyngeal fascia somewhere between the C6 and T4 vertebral levels.
- If the masticator space is involved, the infection has the potential to extend intracranially through the foramen ovale, along the course of the mandibular nerve (V3 branch of the trigeminal nerve).
- CT allows the radiologist to localize the infection accurately and distinguish a true abscess from retropharyngeal edema. Accurate diagnosis is important to avoid unnecessary and potentially harmful surgery for drainage of a reactive noninfected effusion. A true abscess appears as a rim-enhancing hypoattenuating collection that expands the retropharyngeal space and may contain foci of air.
- The degree of sinonasal mucosal thickening and opacification may be minimal and is not sensitive or specific for diagnosis of invasive fungal sinusitis in at-risk patients. Subtle stranding of the periantral fat can be the first imaging finding of vascular invasion and spread of infection beyond the sinuses.

Neck Anatomy

The neck is enclosed by the superficial and deep cervical fasciae. The superficial cervical fascia is not a true fascial sheath and is composed of fatty connective tissue between the skin and the superficial layer of the deep cervical fascia. It contains fat, the platysma muscle, nerve endings, lymph vessels, and portions of the anterior and external jugular veins (Fig 1a, 1b) (8). The deep cervical fascia is composed of three layers: the superficial, middle, and deep layers of the deep cervical fascia. These layers divide the neck into multiple spaces. The neck spaces are further divided into the suprahyoid and infrahyoid spaces, because the deep cervical fascial anatomy is slightly different above and below the level of the hyoid bone. However, some of these spaces cross the hyoid bone level and run throughout the entire neck (Table) (8).

Superficial Layer of the Deep Cervical Fascia

The superficial layer of the deep cervical fascia encases the entire neck deeper than the platysma. It splits and encloses the sternocleidomastoid and trapezius muscles in both the infrahyoid and suprahyoid neck and attaches posteriorly to the ligamentum nuchae and vertebral spinous processes (Fig 1a). In the suprahyoid neck, the superficial layer of the deep cervical fascia also splits, forming the masticator space and the submandibular and parotid gland capsules (Fig 1b). The masticator space contains the temporalis, masseter, and pterygoid muscles, the ramus of the mandible, the mandibular nerve (V3 branch of the trigeminal nerve), the pterygoid venous plexus, and the trigeminal fat pad (8).

Middle Layer of the Deep Cervical Fascia

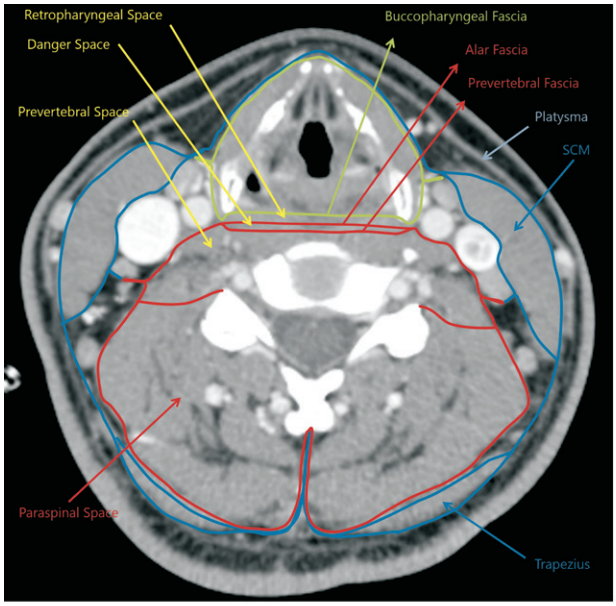
The middle layer of the deep cervical fascia encases all of the visceral organs and forms the visceral space in the infrahyoid neck. It contains the cervical esophagus, larynx, trachea, thyroid gland, parathyroid glands, visceral lymph nodes, and recurrent laryngeal nerves. In the infrahyoid neck, all three layers of the deep cervical fascia join to form the carotid sheath (Fig 1a). Above the hyoid bone, the posterior aspect of the middle layer of the deep cervical fascia (or buccopharyngeal fascia) continues to the skull base and covers the pharyngeal constrictor muscles. However, above the carotid bifurcation, the contribution of the middle layer to the carotid sheath is inconsistent; it does not form a well-defined sheath around the internal and external carotid arteries and the internal jugular vein (Fig 1b). Therefore, many experts recommend the use of the term *poststyloid* (or *retrostyloid*) *parapharyngeal space* instead of the terms *carotid sheath* and *carotid space* in the suprahyoid neck. The parapharyngeal space is divided into the prestyloid and poststyloid spaces by the tensor-vascular-styloid fascia, which runs approximately along a straight line from the styloid process to the medial pterygoid plate on each side (8).

Deep Layer of the Deep Cervical Fascia

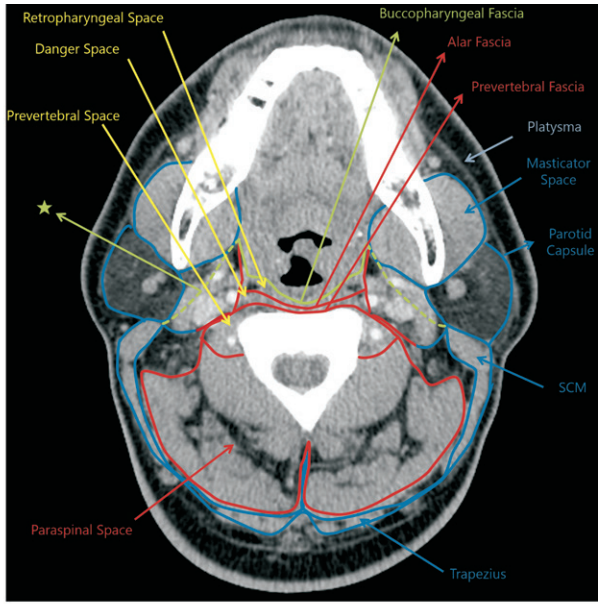
The deep layer of the deep cervical fascia encases the paravertebral muscles and forms the perivertebral space. The deep layer attaches to the ligamentum nuchae and vertebral spinous processes posteriorly, similar to the superficial layer (Fig 1a, 1b). On each side, a flap extends to the cervical transverse processes posterior to the scalene muscles and divides the perivertebral space into the prevertebral component (anterior) and the paraspinal compartment (posterior) (8).

Anterior and medial to the scalene muscles, the deep layer splits into two leaves (Fig 1). The dorsal leaf is the *prevertebral fascia*, which is against the prevertebral muscles and the anterior longitudinal ligament. The ventral leaf is called the *alar fascia*. The space between the prevertebral fascia and the alar fascia is called the *danger space*. The space between the alar fascia and the posterior aspect of the middle layer of the deep cervical fascia (or the *buccopharyngeal fascia*) is called the *retropharyngeal space*. Finally, the space between the prevertebral fascia and the spine is called the prevertebral space (8).

The retropharyngeal space extends from the skull base to the thoracocervical junction as the



a.



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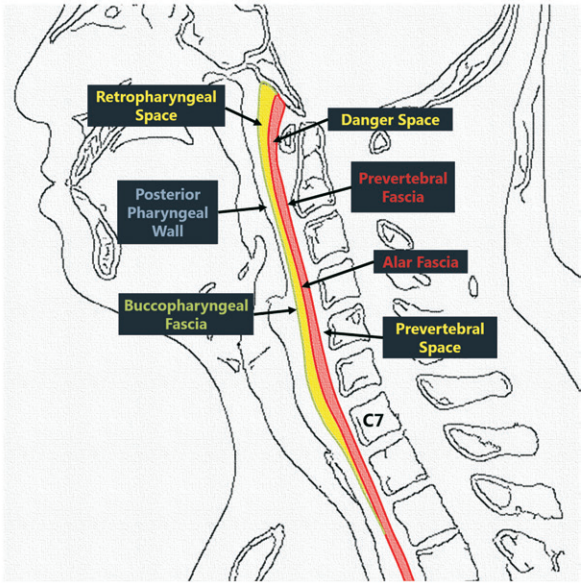


c.

Figure 1. Fascial sheaths and spaces. **(a, b)** Axial CT images show the fascial sheaths in the infrahyoid **(a)** and suprahyoid **(b)** neck and the superficial (blue lines), middle (green lines), and deep (red lines) layers of the deep cervical fascia. The contribution of the middle layer of the deep cervical fascia to the carotid sheath is inconsistent and incomplete in the suprahyoid neck (★ in **b**). *SCM* = sternocleidomastoid muscle. **(c)** Illustration shows the extent of the retropharyngeal and danger spaces.

alar fascia attaches to the buccopharyngeal fascia somewhere between the C6 and T4 vertebral levels (Fig 1c). The retropharyngeal space contains fat and deep cervical lymph nodes, including the lateral and medial retropharyngeal lymph nodes. A retropharyngeal abscess can form from an infected lymph node or direct spread (eg, penetrating trauma violating the buccopharyngeal fascia) (8).

The danger space contains a small amount of loose fatty connective tissue and extends from the skull base to the mediastinum. The alar fascia is a well-defined structure in the midline at the level of C1, but it does not reach the skull base laterally and provides a potential point of entry into the danger space (9). If infection spreads to this space (eg, by direct spread or through the gap between C1 and the skull base), it can extend inferiorly to the chest and cause mediastinitis (Fig 1c) (8).

The prevertebral space contains muscles, vertebral arteries, and parts of the brachial plexus. The prevertebral fascia and space extend from the skull base along the entire spine. If infection involves this space, it has the potential to extend all the way to the coccyx (8).

Oral Cavity and Pharynx

Odontogenic Infection

Odontogenic infection is the most common cause of deep neck infection in adults (5). Poor dental care causes inflammation of the gingiva, or *gingivitis*. Further spread of inflammation between the tooth and the alveolar bone causes periodontal disease, or *periodontitis*, and results in widening of the lucency along the side of the tooth (10). Periodontal disease is less likely to cause a soft-tissue abscess than an apical or periapical abscess (10). An apical or periapical abscess (Fig 2a) is located near the tip of the root of the tooth and often is associated with caries and infection of the pulp (10). A periapical abscess causes more complications than does periodontal disease (Fig 2) (10).

Infection of the mandibular molars can involve the masticator and submandibular spaces, because the roots of these teeth extend below the insertion of the mylohyoid muscle, which

Spaces of the Neck Relative to the Hyoid Bone

- Suprahyoid neck
  - Masticator space
  - Parotid space
  - Sublingual space
  - Submandibular space
  - Parapharyngeal space
    - Prestyloid space
    - Poststyloid space
  - Pharyngeal mucosal space
- Infrahyoid neck
  - Visceral space
  - Carotid sheath
- Suprahyoid and infrahyoid neck
  - Superficial compartment and fascia
  - Retropharyngeal space
  - Danger space
  - Perivertebral space
    - Prevertebral space
    - Paraspinal space
  - Posterior cervical space

separates the submandibular space from the sublingual space. Infection of the other mandibular teeth typically involves the overlying facial soft tissues and sublingual space, because the roots of these teeth extend above the insertion of the mylohyoid muscle (11). Infection of the maxillary molars can involve the masticator space in addition to the maxillary sinuses, palate, and overlying soft tissues, as can infection of the other maxillary teeth. If the masticator space is involved (Fig 2b, Fig E1), the infection has the potential to extend intracranially through the foramen ovale along the course of the mandibular nerve (V3 branch of the trigeminal nerve) (Fig E1).

Ludwig Angina

Ludwig angina is a rapidly progressive and potentially life-threatening cellulitis of the floor of the mouth that involves the sublingual and submandibular spaces (12). Sublingual edema from infection can displace the tongue posteriorly and cause airway compromise. The usual cause is mandibular tooth infection and pericoronitis (13). Pericoronitis is an acute localized soft-tissue infection caused by food particles and microorganisms trapped between the gum flaps and teeth that usually is seen in association with the wisdom teeth in adults or during the eruption of the permanent teeth in children (10). Imaging is performed to assess airway patency and to check for an underlying dental infection or abscess (Fig 3).

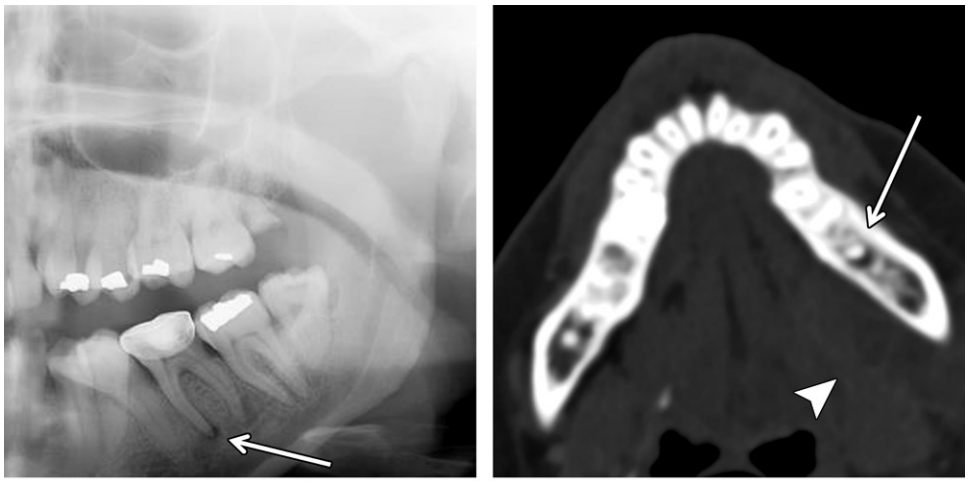
Tonsillitis and Peritonsillar Abscess

Tonsillitis is the most common deep neck infection among children and adolescents (5,14,15). If untreated, the infection can extend to the peritonsillar space (a potential space between the tonsillar capsule and the superior pharyngeal constrictor muscle) and cause a peritonsillar abscess (14,15). A true tonsillar abscess is rare. Patients with tonsillitis, peritonsillar abscess, and true tonsillar abscess may present with a sore throat, odynophagia, and otalgia. Trismus is a common feature of a peritonsillar abscess and can be present in one-third of patients with true tonsillar abscess. Peritonsillar abscess usually is treated with incision and drainage. However, tonsillar abscess usually is treated with antibiotics and supportive measures, and most patients do not require surgical intervention (16). Infection can spread directly to the adjacent parapharyngeal, masticator, or submandibular spaces (Fig 4). Imaging is not always necessary for diagnosis but often is performed, because physical examination is limited and a drainage procedure is under consideration.

Angioedema

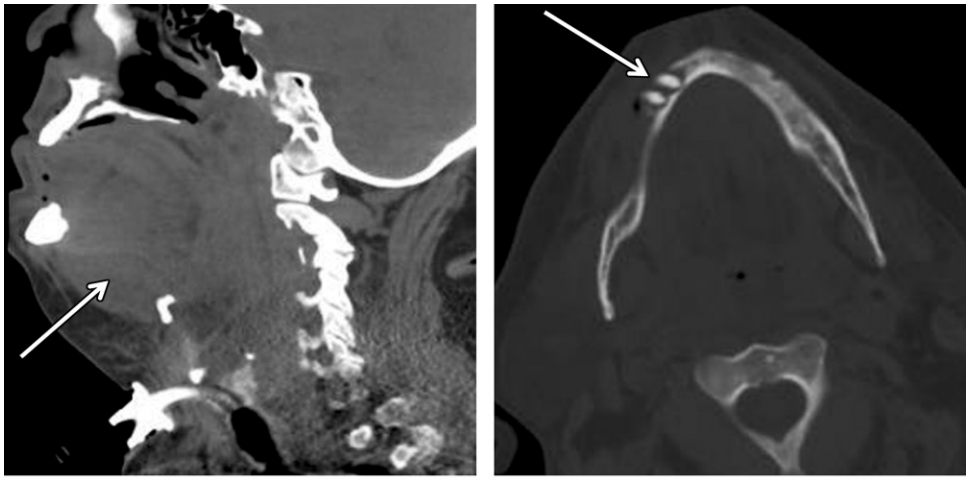
Angioedema manifests with transient subcutaneous or submucosal swelling. It can involve any portion of the body with loose connective tissue but primarily affects the face, tongue, lips, and larynx. It is a potentially life-threatening process because it can quickly cause airway compromise. The causes of angioedema include the use of angiotensin-converting enzyme (ACE) inhibitors or receptor blockers, allergic reaction, and in rare cases, hereditary disorders such as C1 esterase–inhibitor deficiency. In addition, thrombolysis with intravenous tissue plasminogen activator, which is commonly used in treatment of acute ischemic stroke, is associated with increased risk of angioedema, especially with concurrent use of an ACE inhibitor in approximately 2%–5% of patients (17,18). ACE inhibitor–mediated angioedema is not an anaphylactic reaction, and it has a subacute course, usually developing over 24–36 hours and resolving in 2–4 days. The treatment of anaphylactic angioedema is administration of epinephrine, fluids, and oxygen. The treatment of acute allergic angioedema (less severe than anaphylaxis) is administration of antihistamines and steroids. The treatment of ACE inhibitor–mediated angioedema is airway monitoring and discontinuation of the medication (19–21).

The typical imaging findings are diffuse infiltrative transspatial edema with circumferential mucosal thickening and varying degrees of airway narrowing (Fig 5). However, unilateral, focal, and masslike areas of involvement sometimes are seen (Fig E2).



a. b.

Figure 2. Periapical abscess of a mandibular molar in a 46-year-old woman who presented with left temporomandibular joint pain, difficulty opening her mouth, and altered mental status. **(a)** Radiograph shows a periapical lucency involving the mesial root of the left first mandibular molar (arrow). **(b)** Axial CT image (bone window) shows the periapical lucency (arrow), with soft-tissue stranding medial to the mandibular alveolar bone (arrowhead).



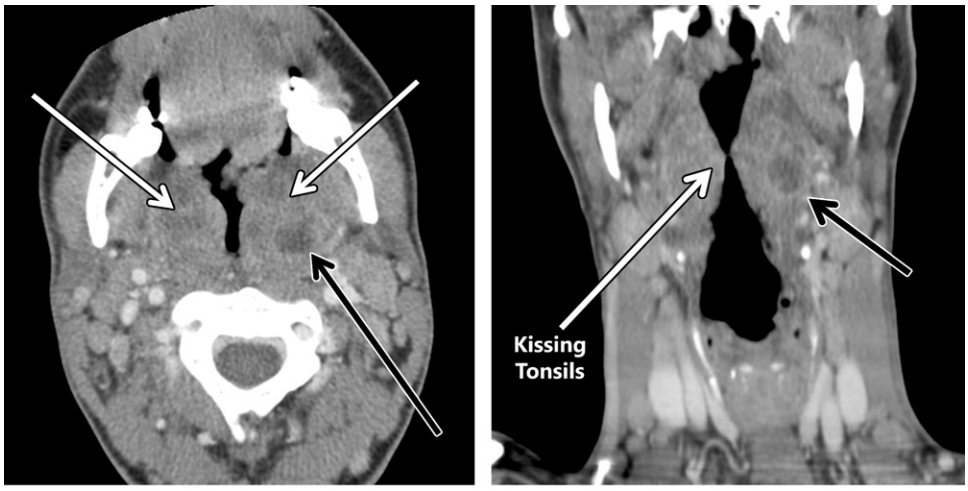
a. b.

Figure 3. Ludwig angina in a 58-year-old man who presented with a sore throat, fever, chills, muffled voice, difficulty breathing, and drooling. **(a)** Sagittal CT image shows extensive soft-tissue swelling and stranding involving the floor of the mouth and tongue (arrow). **(b)** Axial CT image (bone window) shows an anterior mandibular tooth infection (arrow) as the underlying cause. The extensive soft-tissue swelling caused posterior displacement of the tongue and complete obstruction of the pharyngeal airway, resulting in the placement of an emergency tracheostomy tube, which can be seen in **a**.

Neck

Acute Sialadenitis

Acute sialadenitis usually manifests with pain and swelling of the affected gland that worsens with eating or anticipation of eating, which is also known as *salivary colic*. The most common cause of acute sialadenitis is a bacterial infection with *Staphylococcus aureus*. The infection is most commonly caused by salivary duct obstruction by stones (sialoliths). Other common causes are salivary stasis due to dehydration or viral infection (most commonly, mumps parotiditis in children). In addition, bilateral parotid swelling is seen in patients with HIV-associated sialadenitis or immune reconstitution inflammatory syndrome. In rare cases, salivary duct carcinoma is the underlying cause of ductal obstruction and sialadenitis. The submandibular gland is involved most commonly (Fig 6); 85% of stones are seen in the submandibular duct because of the higher viscosity of the submandibular saliva and the upward course of the submandibular duct (22). The treatment usually is endoscopic or surgical stone removal. Less commonly, the duct and gland may be removed when stone removal is unsuccessful or symptoms do not resolve (23).



a. b.

Figure 4. Tonsillitis with a peritonsillar abscess in a 21-year-old man who presented with a sore throat for 1 week, a fever, and worsening throat pain. Axial **(a)** and coronal **(b)** contrast-enhanced CT images show bilateral tonsillar enlargement (white arrows in **a**). The near-midline opposition is often called *kissing tonsils* (white arrow in **b**). A rim-enhancing cystic fluid collection posterolateral to the left tonsil represents a peritonsillar abscess (black arrow).

Acute Epiglottitis

Acute epiglottitis (also known as *supraglottitis*) is inflammation of the epiglottis and adjacent supraglottic structures (Fig 7). In children, it is rapidly progressive and life-threatening, with high risk of airway obstruction. The adult form is more indolent and has a lower risk of airway compromise. Adults better tolerate supraglottic edema because of the larger size of their airway (24,25).

Since the introduction of the *Haemophilus influenzae* type B vaccine, epiglottitis has become less prevalent overall and is more common in adults than in children. An increasing number of cases are caused by other bacteria such as *Streptococcus*, but *H influenzae* type B is still the most common cause due to incomplete community immunity and vaccine failure (26). Epiglottitis can be caused by direct extension of infection from other regions in the head and neck, such as skin cellulitis (Fig E3), dental infection, tonsillitis, and laryngopyocele (1,2,7,27–31).

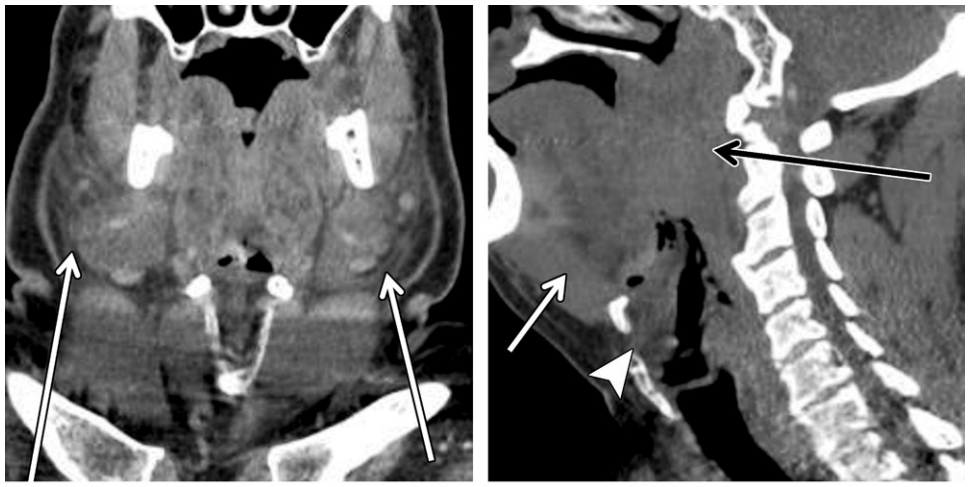
Epiglottitis also can be caused by noninfectious inflammatory processes (Fig E4) such as sarcoidosis. Up to 8% of patients with sarcoidosis have laryngeal involvement, and it usually is limited to the supraglottic larynx (32). Because the true vocal cords have a sparse lymphatic system, they are usually spared (32). Patients may be asymptomatic or may present with hoarseness, dysphonia, dyspnea, and stridor, with reported cases requiring tracheostomy (33). The differential diagnosis for laryngeal supraglottic sarcoidosis includes types of infectious epiglottitis such as tuberculosis, primary laryngeal cancer (especially if it manifests as a focal polypoid mass), lymphoma, amyloidosis, granulomatosis with polyangiitis (formerly called Wegener granulomatosis), and laryngeal pseudotumor (34). Biopsy is required even in patients with a known diagnosis of sarcoidosis because cases of concurrent sarcoidosis and laryngeal carcinoma have been reported.

Superinfected Laryngocele (Pyolaryngocele)

Laryngoceles are air-filled or fluid-filled dilatations of the laryngeal saccule. They are classified as internal or combined laryngoceles. Internal laryngoceles are contained medial to the thyrohyoid membrane in the paraglottic space (35). Combined laryngoceles extend through the thyrohyoid membrane into the superficial neck. The average age at presentation is 50 years, with male predominance (36). Laryngoceles are usually seen in wind instrumentalists, glassblowers, and chronic coughers or are due to mechanical obstruction from an underlying neoplasm (15%–20% of cases). In 8% of cases, laryngoceles become severely infected (Fig 8) (37).

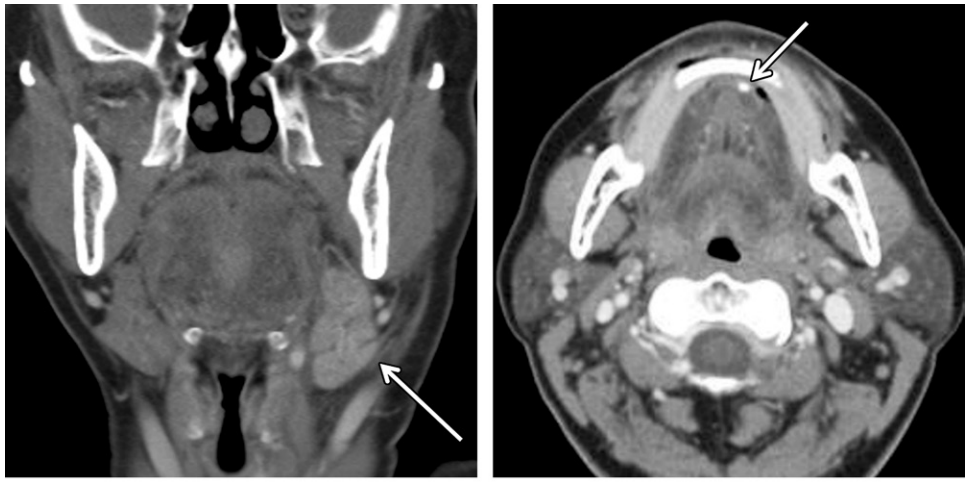
Infected Branchial Cleft Cysts

Branchial cleft cysts are due to persistence of the branchial clefts or pouches. Patients are usually young (10–40 years of age) and often present because of a superimposed infection (Fig E5). Branchial cleft cysts are located along the anterior border of the sternocleidomastoid muscle from the tragus to the clavicle (38). First branchial cleft cysts are seen above the level of the mandible angle near the external auditory canal within or close to the parotid gland. Second branchial cleft cysts are located between the level of the mandible angle and the carotid bifurcation deeper than



a. b.

Figure 5. ACE inhibitor–mediated angioedema in a 59-year-old man with hypertension who presented with 4 days of hoarseness, oral edema, difficulty swallowing, and drooling. Coronal **(a)** and sagittal **(b)** CT images show diffuse transspatial edema involving the bilateral submandibular spaces (white arrows in **a**), tongue, floor of the mouth (white arrow in **b**), pre-epiglottic space (arrowhead in **b**), and pharynx (black arrow in **b**), causing marked pharyngeal airway narrowing.



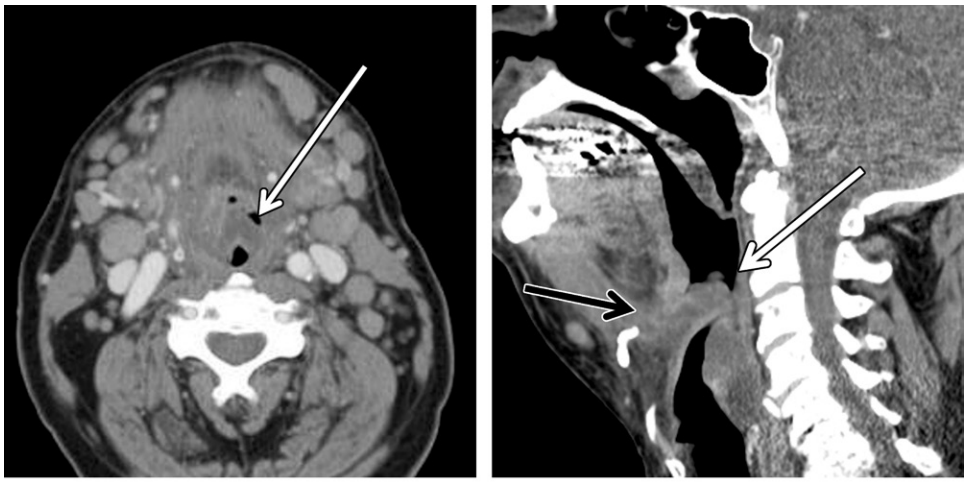
a. b.

Figure 6. Sialadenitis in a 50-year-old woman with left neck swelling and severe pain after eating lunch. **(a)** Coronal CT image shows enlargement and enhancement of the left submandibular gland, with mild surrounding edema (arrow). **(b)** Axial CT image shows a stone at the submandibular duct orifice (arrow) in the floor of the mouth.

the platysma and the superficial layer of the deep cervical fascia (38). Third and fourth branchial cleft cysts are seen in the infrahyoid neck (38). Fourth branchial cleft cysts are usually adjacent to the thyroid gland (38). However, differentiation between third and fourth branchial cleft cysts is often difficult. The second branchial cleft cyst is the most common type (90%–95% of patients with brachial cleft cysts) (38). First branchial cleft cysts are seen in less than 10% of patients (39), and third and fourth branchial cleft cysts are rare. The differential diagnosis for these lesions includes a broad range of conditions such as paramedian thyroglossal duct cysts, thyroid nodules and cysts, necrotic lymph node metastases (usually from squamous cell and papillary thyroid carcinomas), infectious adenitis (eg, tuberculosis), vascular lesions on nonenhanced CT images (eg, mycotic aneurysms of the neck), lymphatic malformations, neurogenic tumors with cystic degeneration (eg, schwannomas), and cervical dermoid cysts.

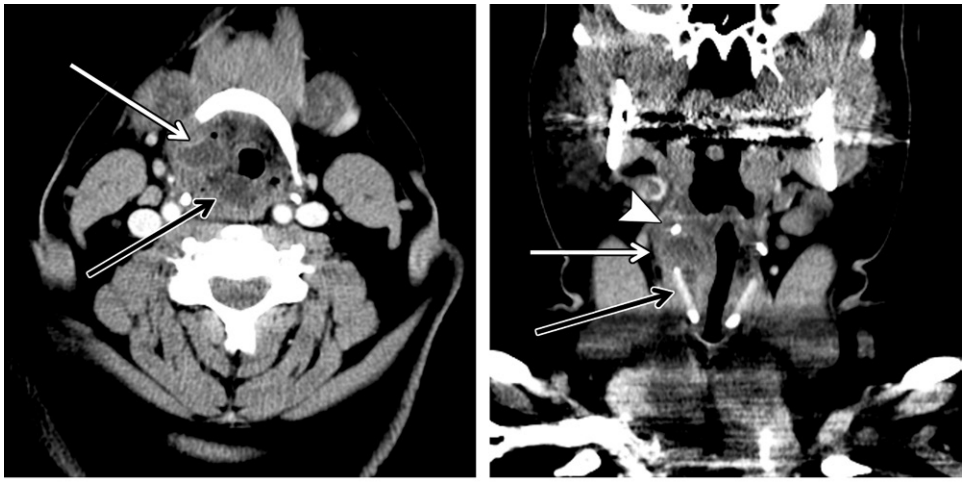
Neck Hemorrhage

Neck hemorrhage is rare, and it usually results from carotid blowout syndrome or extraglandular bleeding due to an underlying thyroid or parathyroid lesion. The mass effect from a soft-tissue hematoma may lead to dysphasia and airway



a. b.

Figure 7. Acute epiglottitis in a 70-year-old man with a sore throat for 3 days and progressive odynophagia. Axial **(a)** and sagittal **(b)** CT images show an enlarged and edematous epiglottis (white arrow) with mucosal enhancement and some stranding of the adjacent supraglottic larynx and tongue base (black arrow in **b**). Substantial airway narrowing is present.



a. b.

Figure 8. Pyolaryngocele in a 47-year-old woman with a severe sore throat, muffled voice, odynophagia, and stridor. Axial **(a)** and coronal **(b)** CT images show a rim-enhancing collection of fluid centered in the right paraglottic space (white arrow) in the expected region of the laryngeal saccule near the thyrohyoid membrane between the hyoid bone (arrowhead in **b**) and the thyroid cartilage (black arrow in **b**), with adjacent soft-tissue stranding and inflammation extending to the right aryepiglottic fold and arytenoids (black arrow in **a**).

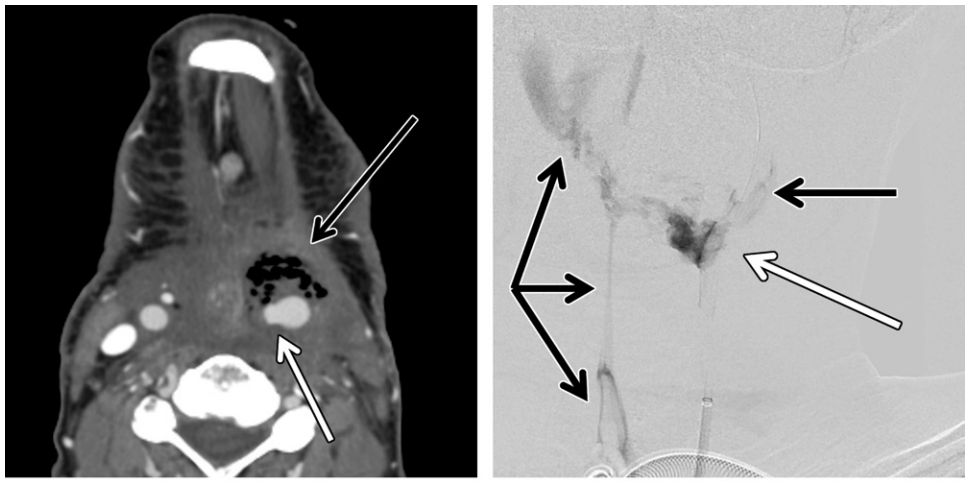
compromise. Severe hemorrhage can happen, especially with carotid blowout syndrome (40,41).

Carotid blowout syndrome refers to the rupture of the carotid artery and its branches. The typical cause is pseudoaneurysm rupture as a complication of radiation treatment for head and neck cancer. The other causes of pseudoaneurysm rupture include infection (mycotic pseudoaneurysm), inflammatory diseases, or direct neoplastic invasion (Fig 9) (41).

Extraglandular thyroid hemorrhage can be seen with thyroid cancers, cysts, nodular goiter, or subacute thyroiditis, and extraglandular parathyroid hemorrhage can be seen with gland enlargement due to hyperplasia, adenoma, or cancer (42–44). Patients may present with swelling and ecchymosis centered in the lateral neck, dysphagia, and dyspnea. A primary parathyroid lesion should be suspected in a patient with hypercalcemia and high parathyroid hormone levels (44) (Fig E6).

Lemierre Syndrome

Lemierre syndrome is a potentially life-threatening condition characterized by suppurative thrombophlebitis in the neck, usually involving the jugular veins. Most commonly, it is a complication of acute pharyngitis or tonsillitis in otherwise healthy young adults (mean age, 20 years) (45). Other



a. b.

Figure 9. Carotid artery blowout syndrome in a 51-year-old woman with recurrent laryngeal cancer who was treated with total laryngectomy and radiation and who presented with intermittent hematemesis. **(a)** Axial contrast-enhanced CT image shows a peripherally enhancing necrotic mass with internal foci of air that is suggestive of ulceration and connection to the pharynx (black arrow). Involvement of the distal common carotid artery with formation of a pseudoaneurysm (white arrow) raises concern for an impending rupture with intermittent hematemesis. **(b)** Oblique angiogram from left common carotid artery injection shows the pseudoaneurysm in the distal left common carotid artery (white arrow), with extravasation of arterial contrast material into the pharynx (black arrows).

head and neck infections (eg, dental, ear, paranasal sinus, and orbit infections, and superficial cellulites from lip piercing), gastrointestinal infections, and trauma may precede Lemierre syndrome (45–47). Patients may develop disseminated abscesses and septic pulmonary emboli (Fig 10; Fig E7). Lemierre syndrome most often is caused by anaerobic gram-negative organisms and most frequently the *Fusobacterium* necrophorum, which is a part of normal oropharyngeal flora. Treatment is with anticoagulation therapy (in the absence of contraindications), intravenous antibiotics, and surgical drainage of nonresolving abscesses (48).

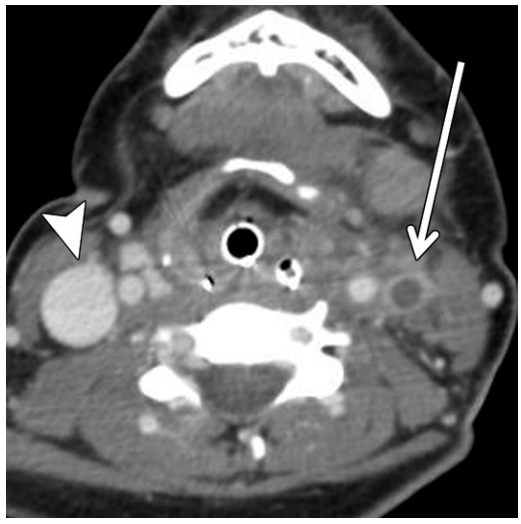


Figure 10. Lemierre syndrome in a 20-year-old woman with left-sided neck pain after a few days of sore throat and flulike symptoms. Axial contrast-enhanced CT image shows intraluminal thrombosis and enlargement of the left internal jugular vein, with rimlike enhancement of the venous wall and adjacent inflammation (arrow) and a normal patent right internal jugular vein (arrowhead).

Carotidynia

Carotidynia, also known as transient perivascular inflammation of the carotid artery, is an inflammatory pseudotumor-like disease (49). Patients present with pain and tenderness in the region of the carotid bifurcation. The symptoms are treated with antiinflammatory medications and resolve on their own in days or weeks. Imaging shows circumferential thickening of the distal common carotid, bifurcation, and proximal internal carotid artery walls (49) (Fig E8). Carotidynia is a diagnosis of exclusion, and imaging should be repeated after treatment to confirm resolution and exclude other abnormalities. The differential diagnosis for the imaging findings includes vasculitis and dissection.

Retropharyngeal Abscess

Retropharyngeal abscesses are caused by the spread of infection from a site with drainage to the retropharyngeal lymph nodes such as tonsillitis, pharyngitis, otitis, and oral cavity infections; direct spread from adjacent discitis-osteomyelitis; and inoculation from penetrating trauma. Patients usually present with a fever, sore throat, neck pain, and a limited range of motion. CT allows the radiologist to localize the infection accurately and to distinguish a true abscess from retropharyngeal edema (Fig 11; Fig E9). Accurate diagnosis is important to avoid unnecessary and potentially

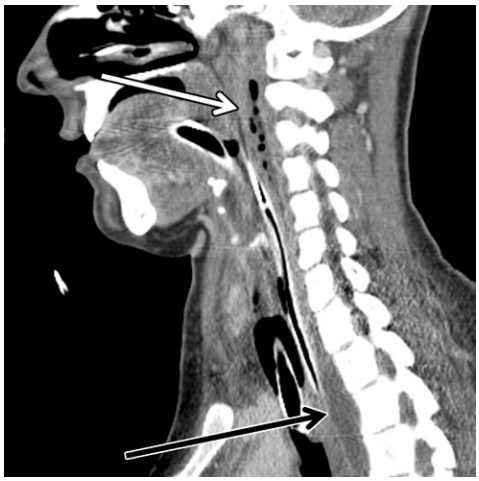


Figure 11. Retropharyngeal abscess complicated by descending mediastinitis in a 22-year-old woman with worsening dyspnea and neck pain after 5 days of pharyngitis. Sagittal contrast-enhanced CT image shows fluid and air (white arrow) in the retropharyngeal space, which is consistent with a retropharyngeal abscess. A more posterior fluid collection (black arrow) extends inferiorly into the mediastinum, indicating extension of the infection into the danger space.

harmful surgery for drainage of a reactive noninfected effusion. A true abscess appears as a rim-enhancing hypoattenuating collection that expands the retropharyngeal space and may contain foci of air (24,50). Imaging is also useful for assessment of the potential complications, such as extension of the abscess into the mediastinum through the danger space; airway compromise; direct extension to the spine and epidural space; and involvement of the major cervical vessels, which can cause internal jugular vein thrombosis, carotid pseudoaneurysm, or carotid artery stenosis. Descending mediastinitis is a rare but serious complication of oral cavity and neck infections and has a high mortality rate because its nonspecific subtle symptoms and clinical findings often lead to delayed diagnosis. The abscess most commonly spreads through the danger space to the mediastinum (24,50), but it also may spread through the carotid sheaths and visceral space (Fig E9).

Longus Colli Calcific Tendinitis

Longus colli calcific tendinitis is a self-limited disease that is caused by deposition of hydroxyapatite crystals and responds to nonsteroidal anti-inflammatory drugs. The clinical manifestation is similar to that of retropharyngeal abscess, and CT is performed to differentiate between these conditions (Fig 12). Patients usually present with acute onset of neck pain and stiffness. Patients may have a fever or odynophagia, and a blood test result may show mild leukocytosis and an elevated C-reactive protein level similar to that of retropharyngeal abscess (51). Accurate diagnosis is important to avoid unnecessary and potentially harmful surgery. The CT findings include effusion in the prevertebral space. The absence of substantial rim enhancement and detection of a small amount of calcification or ossification at the insertion of the longus colli muscles to the anterior arch of C1 are characteristic findings (51). The longus colli muscles also may appear hypoattenuating because of inflammation and edema.

Discitis-Osteomyelitis

The most common cause of discitis-osteomyelitis is hematogenous seeding from a distant focus, and it is more common in the thoracic and lumbar spine than in the cervical spine (52). Other less common causes are recent spinal procedures and spreading from adjacent soft tissue infections (52). CT findings are initially subtle and include loss of disk height and endplate irregularities, vertebral body collapse, and perivertebral soft-tissue inflammation and abscess (24). MRI is more sensitive than CT for early detection of the disease and epidural or paravertebral abscesses (Fig 13) (53). Risk factors for discitis-osteomyelitis include intravenous drug use, infective endocarditis, prior spinal surgery, diabetes, steroid therapy or other immunocompromised states, and degenerative disease (53).

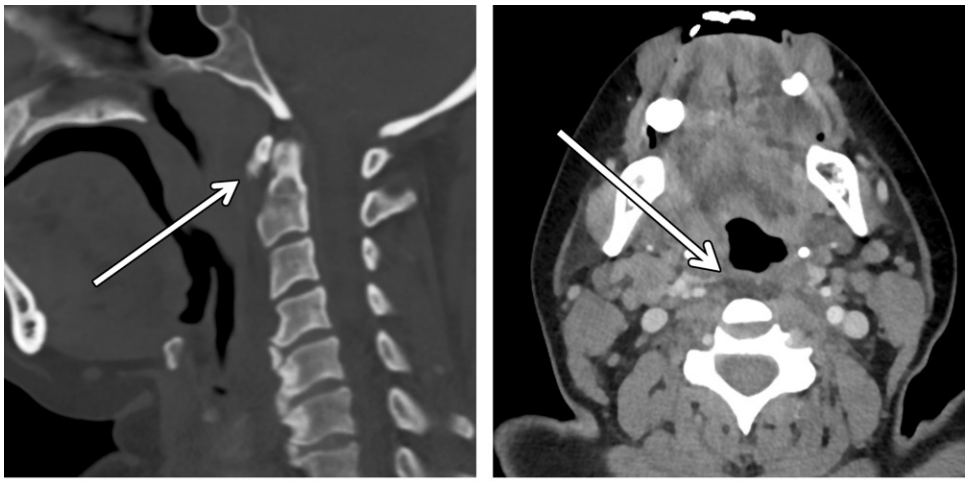
Septic Facet Arthritis

Septic facet arthritis is an uncommon but serious cause of acute neck pain that typically involves a single level of the spine (54). CT findings are usually subtle, especially early in the course of the disease, and include mixed lytic and sclerotic changes in the facet and edema in the adjacent soft tissues. MRI is more sensitive than CT for early diagnosis and detection of the potential complications. Typical MRI findings are bone marrow edema and enhancement, expansion of the facet joint capsule with effusion and rim-like enhancement, and edema in the adjacent prevertebral and paraspinal spaces (Fig 14). The infection can extend into the adjacent spinal canal, causing an epidural abscess, with potential to cause spinal cord injury or meningitis (55). The causes of spinal cord injury are mass effect and direct compression by the abscess, thrombophlebitis of the adjacent veins, occlusion of the arterial supply, and direct extension of the infection and inflammation to the spinal cord.

Sinonasal Cavities

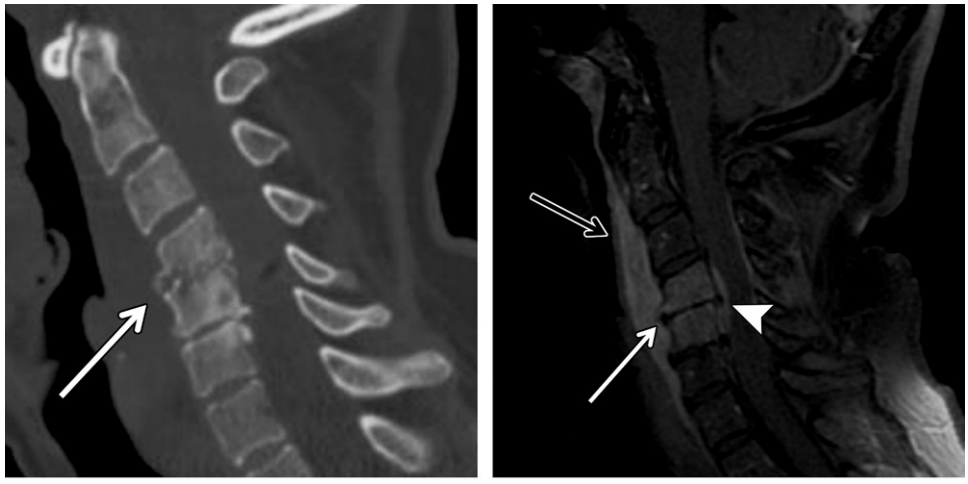
Acute Rhinosinusitis

The term *rhinosinusitis* is preferred over the term *sinusitis* because the nasal cavity is involved in almost all cases. Acute rhinosinusitis is a clinical



a. b.

Figure 12. Longus colli calcific tendinitis in a 47-year-old man with transient pain and tenderness over the right middle neck. **(a)** Sagittal CT image (bone window) shows amorphous calcification (arrow) at the insertion of the longus colli tendon near the anterior arch of the C1 vertebra. **(b)** Axial contrast-enhanced CT image shows hypoattenuating effusion (arrow) in the prevertebral space without surrounding enhancement.



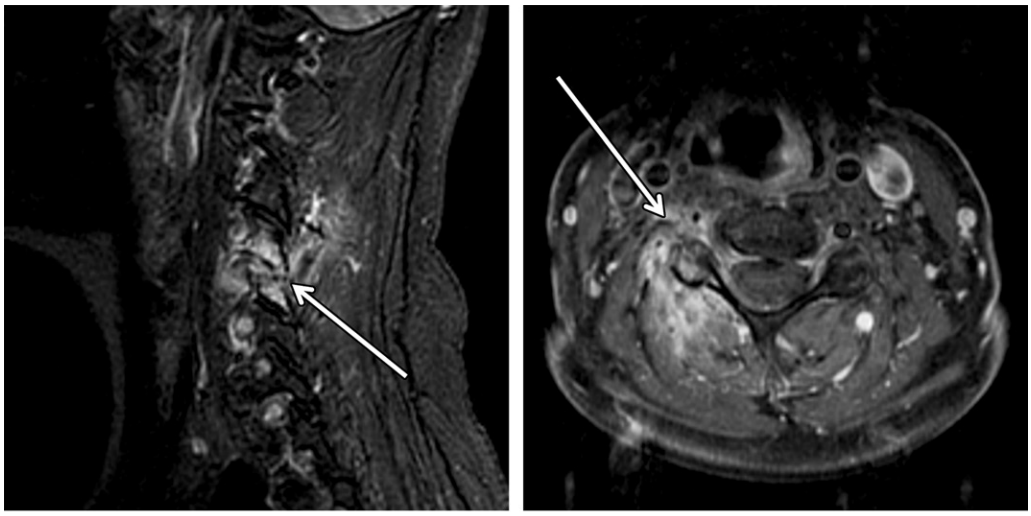
a. b.

Figure 13. Discitis-osteomyelitis in a 55-year-old woman with a history of splenectomy who presented with 3 weeks of neck pain and tenderness in the right middle neck. Sagittal CT **(a)** and sagittal contrast-enhanced fat-suppressed T1-weighted MR **(b)** images show loss of disk height and endplate irregularities that involve two adjacent vertebral bodies (white arrow) with prevertebral (black arrow in **b**) and epidural (arrowhead in **b**) soft-tissue inflammation and abscess.

diagnosis defined by symptom duration of less than 4 weeks. Imaging findings are nonspecific and can be seen in up to 40% of asymptomatic adults and more that 80% of patients with minor upper respiratory tract infections. Therefore, the imaging findings should be interpreted with attention to clinical and endoscopic findings. The imaging findings are mucosal thickening, air-fluid level in the paranasal sinuses, aerated fluid or secretions, and obstruction of the osteomeatal complexes. CT can be performed if complications such as orbital cellulites, venous sinus thrombosis, or intracranial extension are suspected. The maxillary teeth also should be assessed carefully because up to 20% of maxillary sinus infections are odontogenic (56).

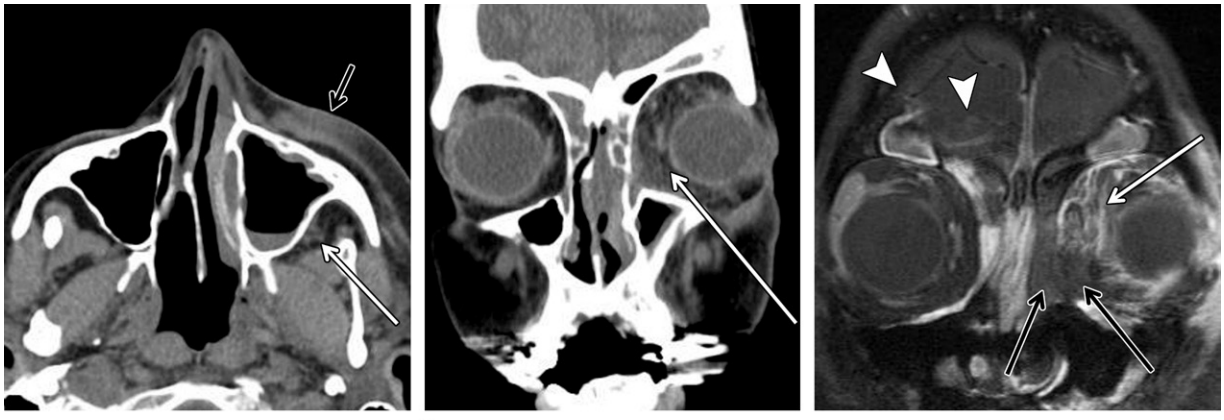
Invasive Fungal Sinusitis

Invasive fungal sinusitis is seen almost exclusively in immunocompromised patients with neutropenia or neutrophil dysfunction, usually in those with hematologic malignancies or poorly controlled diabetes and those undergoing immunosuppressive therapy for organ transplant or chemotherapy. It is most commonly caused by *Aspergillus* or *Mucor* fungi. Patients usually present with fever, facial pain, nasal congestion, and sensation loss in the malar areas. Patients



a. b.

Figure 14. Septic facet arthritis in a 58-year-old woman with fever, neck pain, and stiffness. Sagittal fat-suppressed T2-weighted **(a)** and contrast-enhanced T1-weighted **(b)** MR images show edema at one of the cervical facet joints, with adjacent soft-tissue inflammation (arrow) that extends into the prevertebral and paraspinal spaces and the adjacent neural foramen.



a. b. c.

Figure 15. Invasive fungal sinusitis in a 62-year-old man with neutropenia after bone marrow transplant who presented with sinus disease, eye pain, and periorbital swelling and ecchymosis. **(a, b)** Initial axial **(a)** and coronal **(b)** CT images show a small air-fluid level in the left maxillary sinus and opacification of the ethmoid air cells and frontal sinuses with mild premaxillary (black arrow in **a**), retromaxillary (white arrow in **a**), and orbital (arrow in **b**) fat stranding. **(c)** Contrast-enhanced fat-suppressed T1-weighted MR image from follow-up examination performed 24 hours after initial CT shows marked worsening of the orbital soft-tissue inflammation (white arrow), intracranial extension with meningeal enhancement (arrowheads), and extensive necrosis (black arrows), indicated by lack of contrast enhancement along the medial orbit and lateral wall of the left nasal cavity.

also may have a change in vision or mental status. The nasal and oral cavities should be examined carefully for necrotic ulceration. The infection rapidly spreads from the sinuses by means of vascular invasion, causing bony erosions, with extension to the orbit, brain, cavernous sinus, or carotid artery. Immediate treatment with systemic antifungal medications and aggressive surgical débridement is required. However, despite early treatment, the mortality rate is very high at approximately 50%–60% (57,58). CT findings include sinonasal mucosal thickening and opacification; stranding in the periantral fat, pterygopalatine fossa, nasolacrimal duct, lacrimal sac, and orbit; bone erosion and dehiscence; and nasal septal ulceration. The imaging findings may be mild, especially early in the course of disease. The degree of sinonasal mucosal thickening and opacification may be minimal and is not sensitive or specific for diagnosis of invasive fungal sinusitis in at-risk patients. Subtle stranding of the periantral fat can be the first imaging finding of vascular invasion and spread of infection beyond the sinuses (Fig 15). A retrospective matched case-control analysis (59) of 42 patients with histopathologically proven invasive fungal sinusitis showed that the presence of only one of the CT findings has sensitivity of 95% and specificity of

86% for diagnosis of invasive fungal sinusitis in at-risk patients. The same study showed sensitivity of 88% and specificity of 100%, with two or more CT findings in at-risk patients. Leptomeningeal enhancement, cavernous sinus thrombosis, carotid pseudoaneurysm, cerebritis, and brain abscess can be seen with intracranial invasion (60).

Juvenile Nasopharyngeal Angiofibroma

Juvenile nasopharyngeal angiofibroma is a tumor that arises from the sphenopalatine foramen, posterior nasal cavity, or nasopharynx (61) (Fig 16). It is seen almost exclusively in young male patients (aged 8–25 years). It usually manifests with epistaxis, which can be life-threatening, or chronic otomastoiditis from obstruction of the eustachian tube. Imaging is crucial for diagnosis because the tumor is highly vascular, and biopsy should be avoided because of the risk of severe hemorrhage. CT findings include a lobulated soft-tissue mass with prominent enhancement near the sphenopalatine foramen, with extension to the nasopharynx and pterygopalatine fossa (62). The tumor typically causes remodeling of the adjacent bones and widening of the sphenopalatine foramen. The lesion has intermediate signal intensity at T1-weighted MRI. At T2-weighted MRI, the lesion is heterogeneous and usually contains flow void (62). Similar to its appearance at CT, the lesion shows prominent enhancement at MRI (62). The treatment is endovascular embolization to avoid life-threatening hemorrhage, followed by resection.

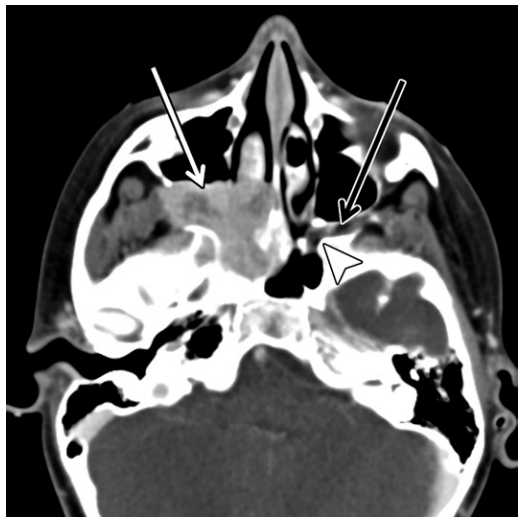


Figure 16. Juvenile nasopharyngeal angiofibroma in an 11-year-old boy who presented with severe epistaxis. Axial CT image shows a lobulated and intensely enhancing mass involving the right sphenopalatine foramen, posterior nasal cavity, right sphenoid sinus, and adjacent nasopharynx (white arrow), causing remodeling and widening of the sphenopalatine foramen and pterygopalatine fossa, and the normal contralateral sphenopalatine foramen (arrowhead) and pterygopalatine fossa (black arrow).

Orbits

Preseptal Cellulitis

Preseptal or periorbital cellulitis is an infection limited to the skin and soft tissues anterior to the orbital septum. The orbital septum is a thin sheet of fibrous tissue that extends from the orbital rim periosteum to the tarsal plates in the eyelids and separates superficial soft tissues from the orbit. Preseptal cellulitis often is caused by contiguous spread of an infection from the teeth, sinuses, or ocular adnexa; insect bites; or trauma (63) (Fig E10). It is treated with oral antibiotic therapy.

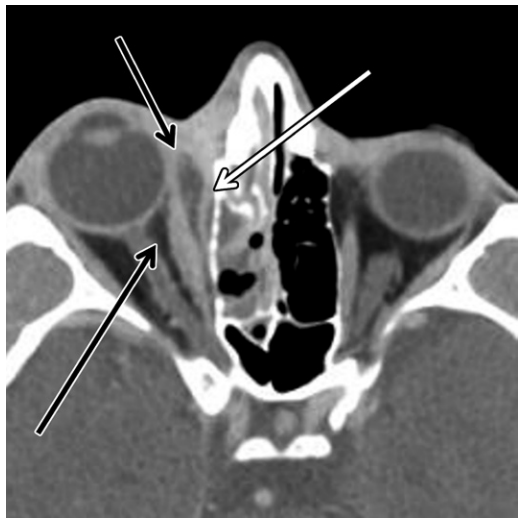
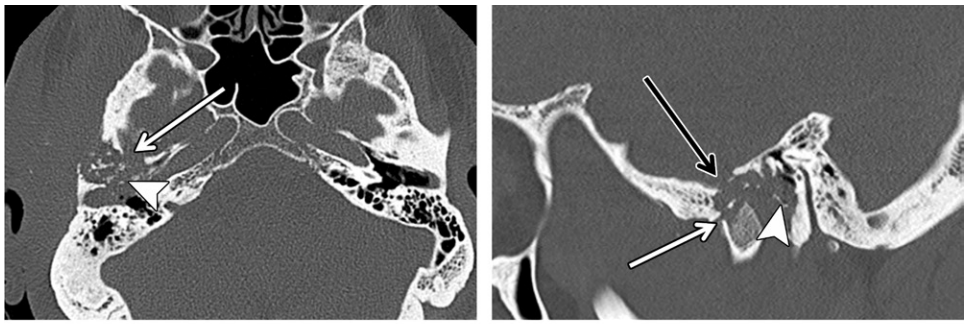


Figure 17. Orbital cellulitis in a 19-year-old man with right eye swelling and pain after sinusitis, which worsened with eye movement. Axial contrast-enhanced CT image shows opacification of the right ethmoidal air cells, with a small adjacent subperiosteal abscess (white arrow) and orbital fat stranding (black arrows).

Orbital Cellulitis

Orbital cellulitis is an infection of the orbit posterior to the orbital septum, typically due to rhinosinusitis (64) (Fig 17). Orbital cellulitis is an emergency, and complications include loss of vision, superior ophthalmic vein thrombosis, and intracranial extension that leads to cavernous sinus thrombosis, meningitis, and intracranial abscess. Treatment is administration of intravenous antibiotics; however, surgical drainage may be necessary if there is a large subperiosteal abscess, poor response to antibiotics, or if vision is threatened (65). Orbital cellulitis must be differentiated from an orbital pseudotumor, or nonspecific orbital inflammation, which usually responds quickly to treatment with steroids. The muscles, lacrimal gland, or other orbital soft tissues may be involved. Immunoglobulin G4–related disease accounts for 25%–50% of cases of



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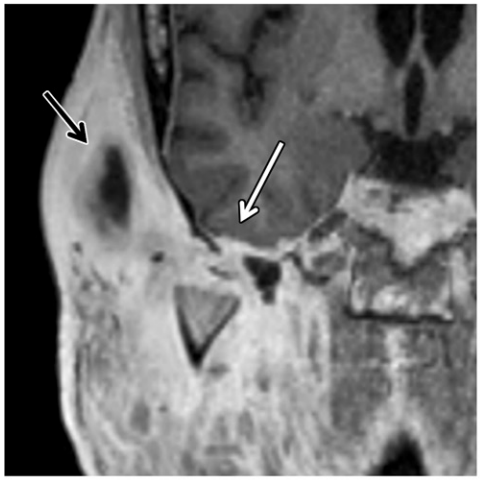

c.

Figure 18. Malignant otitis externa with skull base osteomyelitis in a 55-year-old man with diabetes and recently treated otitis externa who presented with increased drainage from his external auditory canal, periauricular swelling, and pain worsened by mastication. **(a, b)** Axial **(a)** and coronal **(b)** CT images show destruction of the right temporomandibular joint (white arrow), destruction of the anterior wall of the fluid-filled right external auditory canal (arrowhead), and destruction of the mandibular fossa of the right temporal bone (black arrow in **b**). **(c)** Coronal contrast-enhanced fat-suppressed T1-weighted MR image shows extensive soft-tissue enhancement with a right periauricular abscess (black arrow) and intracranial extension of the infection through the eroded mandibular fossa as demonstrated by enhancement of the dura in the right middle cranial fossa (white arrow).

orbital pseudotumor (66). Acute progression of thyroid eye disease (also known as *thyroid orbitopathy*) also should be considered in the differential diagnosis of orbital cellulitis and orbital pseudotumor. The structures chiefly affected during the acute inflammatory phase are the orbital extraocular muscles and orbital adipose tissue. Unlike orbital pseudotumors, thyroid eye disease typically is characterized at imaging by bilateral and symmetrical enlargement of the extraocular muscle bellies, with sparing of the tendinous insertions(67).

Dacryocystitis

Dacryocystitis is an acute infection of the nasolacrimal system. The predisposing factor is stasis of lacrimal fluid due to stenosis or obstruction of the lacrimal duct, which can be congenital or acquired in patients who have experienced trauma, prior surgery, or systemic diseases such as sarcoidosis and Wegener granulomatosis (68). Patients usually present with acute onset of pain, erythema, warmth, and tenderness over the lacrimal sac, with purulent discharge. Complications are preseptal or orbital cellulitis, conjunctivitis, sepsis, and meningitis (68). The diagnosis typically is made clinically, but imaging may be performed for assessment of possible complications. CT findings include a cystic fluid collection with rim enhancement along the inner canthus, with adjacent inflammation and soft-tissue thickening (Fig E11). Treatment is administration of either oral or intravenous antibiotics, depending on the severity of the infection and its complications.

Ears

Malignant External Otitis

Malignant external otitis, or necrotizing external otitis, is an invasive infection of the external auditory canal, skull base, and adjacent soft tissues that usually is seen in elderly patients with diabetes (Fig 18) or immunocompromised patients who are infected with HIV (69). The cause is almost always infection with *Pseudomonas aeruginosa*. The imaging findings include soft-tissue attenuation or cortical bone erosion of the external auditory canal and extension into the periauricular soft tissues and deep neck spaces such as the masticator space, nasopharynx, and parapharyngeal space, opacification of the mastoid air cells and the middle ear from direct extension of the infection, and skull base bone erosion with intracranial extension. The imaging appearance can be misinterpreted as a malignancy if one relies on imaging findings only without attention to the patient's clinical symptoms at presentation (70,71).

Complications include temporomandibular joint involvement, cranial nerve palsy due to skull base involvement, meningitis, brain abscess, and dural venous sinus thrombophlebitis.

Acute Middle Ear and Mastoid Infection (Otomastoiditis)

These common ear infections may be complicated by extension into the contiguous soft tissues or intracranial extension resulting in epidural abscess, sigmoid sinus thrombosis, meningitis, and intracranial abscess, particularly in children. Imaging is not necessary unless complications are suspected. The CT findings include fluid and soft-tissue attenuation in the middle ear cavity and mastoid, thickening and outward bulging of the tympanic membrane, or tympanic membrane perforation. When the mastoid trabeculae are destroyed, it is called coalescent mastoiditis, which may cause further erosion of the outer and inner cortex of the mastoid, resulting in periauricular cellulitis with or without an abscess and intracranial extension, respectively (72).

Conclusion

CT is an accurate tool for diagnosis of most of the nontraumatic head and neck emergencies and their complications, but MRI provides higher accuracy in diagnosis of abnormalities such as discitis-osteomyelitis, septic facet arthritis, and intracranial complications. Familiarity with the anatomic landmarks and the imaging appearance of head and neck diseases and their complications makes head and neck imaging a less intimidating subject for radiologists.

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