

Head and Neck Infection and Inflammation

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KEYWORDS

• Head and neck • Inflammation • Infection • Abscess

This article discusses the imaging manifestations of infectious and inflammatory conditions of the head and neck. Special attention is paid to the sites, routes of spread, and complications of neck infections. Because the clinical signs and symptoms and the complications of these conditions are often determined by the precise anatomic site involved, anatomic considerations are stressed. Familiarity with the fascial layers and spaces of the neck and the contents of each space is helpful for this discussion.¹ The fascial layers of the neck are important barriers to infection, and once infection is established, the fascial layers play a part in directing its spread.

SUPERFICIAL NECK INFECTIONS

The superficial cervical fascia underlies the skin and is composed of the adipose tissue, superficial neurovascular structures, lymphatics, and platysma muscle (**Fig. 1**). Infection in the superficial space usually manifests as cellulitis, lymphadenitis, or abscess.

As opposed to cervical lymphadenopathy, which simply refers to the enlargement of the lymph nodes in the neck, cervical lymphadenitis is defined as the enlarged, inflamed, and tender lymph nodes of the neck. Cervical lymphadenitis is common in children, and the most common cause is an upper respiratory illness. Because cervical lymphadenitis is typically self-limited, the

exact incidence is difficult to ascertain and imaging is usually not performed. Common sites of cervical lymphadenitis are the submandibular and deep cervical nodes because these nodes filter much of the lymphatic fluid from the head and neck. Acute bilateral cervical lymphadenitis is the most common form and is usually caused by a viral upper respiratory tract infection.² Acute unilateral cervical lymphadenitis is the next most common form and is usually caused by bacterial infection, with the most common causative organisms being *Staphylococcus aureus* and *Streptococcus pyogenes*.³ The incidence of methicillin-resistant *S aureus* has increased in the recent years.⁴ Acute unilateral cervical lymphadenitis in older children or adults with a history of periodontal disease is usually caused by an infection with anaerobic bacteria. Superficial neck abscesses usually begin as lymphadenitis, which then progresses from phlegmon to abscess (**Fig. 2**).

DEEP NECK INFECTIONS

The deep cervical fascia is divided into 3 layers (superficial, middle, and deep) that enclose the contents of the head and neck and give rise to the deep spaces of the neck (see **Fig. 1**).

Intrinsic or deep neck space infections (DNSIs) usually represent the overgrowth of the normal flora of the contiguous mucosal surfaces from

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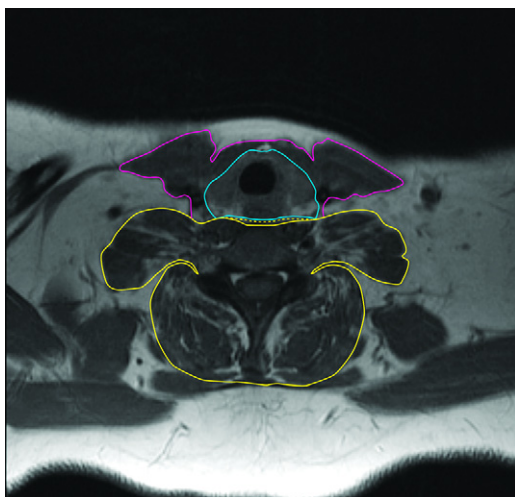


Fig. 1. Layers of deep cervical fascia. The strap muscles and sternocleidomastoid muscle are enclosed by the superficial layer of deep cervical fascia (pink). The visceral space is enveloped by the middle, or visceral, layer of deep cervical fascia (blue). The deep layer of deep cervical fascia (yellow) surrounds the paraspinal and prevertebral components of the perivertebral space. From anterior to posterior, the retropharyngeal and danger spaces are separated by the alar fascia (yellow dashed line), which is part of the deep cervical fascia.



Fig. 2. Suppurative adenitis of a superficial node. Axial contrast-enhanced computed tomography demonstrates an abscess in the left superficial neck resulting from suppurative adenitis (asterisk).

which the infection originated. Most infections are polymicrobial with not only predominantly anaerobic but also facultative oral bacteria.⁵ Even in the antibiotic era, life-threatening complications of DNSIs may occur, including airway compromise, venous thrombosis, and mediastinal extension. Diagnosis of the complications of deep space infections may be delayed because many patients with this condition are started on oral antibiotics as outpatients, which may mask local signs such as edema, fluctuation, and pointing of abscess. The most common clinical signs and symptoms are fever, pain, and swelling, which may be present for a variable period, ranging from 1 day to several weeks. Additional symptoms include odynophagia, dysphagia, and trismus. *Streptococcus viridans*, *Klebsiella pneumoniae*, and *Peptostreptococcus* are the most common pathogens that cause neck infections.⁶ The most common odontogenic organisms are *S. viridans*, *Prevotella*, *Staphylococci*, and *Peptostreptococcus*.⁷

SOURCE AND SITE OF INFECTIONS

Odontogenic infections are the most common source of infections in the head and neck among adults.⁸ Other sources include sialadenitis, trauma, intravenous (IV) drug use, and sinusitis. In a large percentage of cases, no source is found.^{9,10} In children, the most common source of infection is the tonsils.³ Comorbidities frequently found in patients with DNSI are diabetes mellitus, old age, alcohol abuse, systemic disease, and immunosuppressed states, such as in patients with AIDS or in those undergoing chemotherapy. Primary head and neck cancer may occasionally present as a DNSI.¹¹ Recurrent neck infections can also be seen in the setting of developmental lesions such as thyroglossal duct cysts and branchial cleft cysts (Fig. 3).¹²

Odontogenic Spread of Infection

Inflammatory disease of the teeth can be divided into periodontal or endodontal infection.¹³ Endodontal infection occurs at the root apex of the tooth. Typically, the infection originates as dental caries, which destroys enamel and dentine, permitting bacteria to enter the pulp chamber containing the neurovascular bundle. The infection then travels down the root canal and out to the apical foramen, with resultant bone resorption. At this point, an acute abscess or a chronic granuloma may form involving the apex of the tooth (Fig. 4).

Periodontal infection originates as gingivitis and travels along the side of the tooth along the

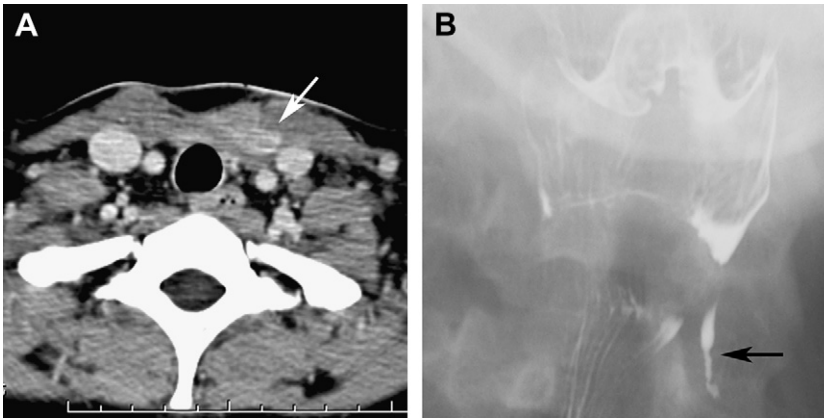


Fig. 3. Fourth branchial cleft cyst causing recurrent perithyroidal inflammation. (A) Axial contrast-enhanced computed tomography demonstrates phlegmonous changes anterior to and inseparable from the left lobe of the thyroid gland (arrow). (B) Barium esophagram demonstrates opacification of a fistulous tract (arrow) from the apex of the left pyriform sinus extending into the thyroid bed.

periodontal ligament, causing bone resorption and formation of a periodontal pocket in which infection propagates, which results in a radiolucency along the side of the tooth (see **Fig. 4**).

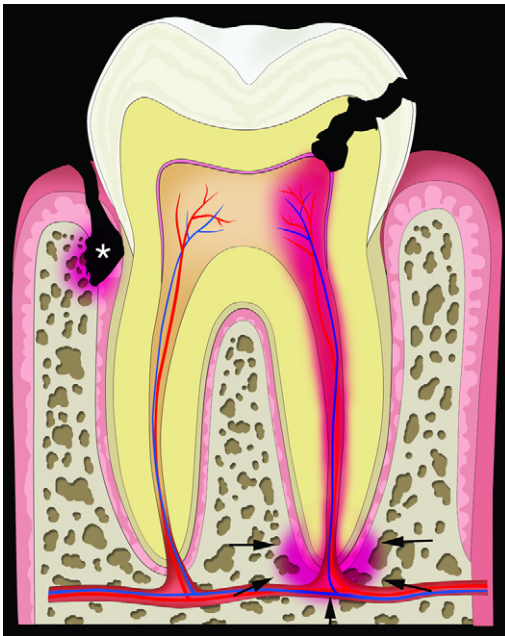


Fig. 4. Endodontal and periodontal spread of infection. On the right side of the image, dental caries allow bacteria to enter the pulp chamber and travel down the root canal to the apical foramen where a periapical lucency corresponds to bone loss (arrows). Pressure from the pulpitis compromises the blood supply to the tooth rendering it nonvital. On the left side of the image, gingivitis alongside of tooth (asterisk) travels along the periodontal ligament to cause bone resorption and formation of a periodontal pocket in which infection propagates.

The most common paths of spread of maxillary odontogenic infection are to the buccal, masticator, and parapharyngeal spaces.⁷ Specifically, infection originating in the maxillary incisors, canines, premolars, and first molars tends to spread to the buccal space, whereas infection originating in the second maxillary molar tends to spread to the masticator space (**Fig. 5**). Infection of the third maxillary molar tends to spread to the parapharyngeal space.¹⁴

In the mandibular teeth, odontogenic infection originating in the incisors, canines, or first premolars is most likely to spread into the sublingual space. Because the second and third mandibular molar tooth roots extend below the mylohyoid muscle insertion, odontogenic infection originating from these teeth is most likely to spread into the submandibular space (**Fig. 6**).¹⁵ Oroantral fistula can occur as a complication of dental infection with resorption of the tooth root. Because the sinus floor is oriented parallel to the transverse plane, it can be difficult to appreciate this complication on axial imaging; thus coronal or panoramic reformations are key in demonstrating the communication.¹⁶

Odontogenic sinusitis refers to the secondary infection of the paranasal sinuses originating from dental infection. Although most paranasal sinus infections result from an upper respiratory tract infection, direct spread of the infection from the teeth and associated oral tissues can result in secondary sinusitis, sometimes with a discrete perforation in the wall of the sinus.¹⁵ The maxillary sinus is the main sinus involved because of the proximity of the posterior maxillary teeth. Infection may originate from a periapical abscess or retained tooth fragment after extraction. Symptoms



Fig. 5. Masticator space infection. Coronal contrast-enhanced computed tomography demonstrates right masticator space abscess (asterisk) originating from an odontogenic infection of the maxillary molar (arrow).

include headache, foul discharge, fever, weakness, and drainage. Findings on computed tomography (CT) include thickening of sinus walls, sclerosis, and perforation. Surgery to increase the size of the ostia in the lateral walls of the nasal cavity may help to improve drainage.

Osteomyelitis may complicate odontogenic infection and can demonstrate 4 radiographic patterns¹⁷: lytic, sclerotic, and mixed forms and a bony sequestrum.

Rarely, odontogenic infections may spread to the orbit and are often clinically unsuspected

(**Fig. 7**). The route of spread may involve the premolar soft tissues, maxillary sinuses, or infra-temporal fossa.¹⁸ These cases require dental surgery for the resolution of infection.

Invasive Fungal Sinusitis

Sinusitis may be suspected to be of fungal origin based on atypical clinical presentations, including nonresponse to antibiotics, or on imaging features. Fungal sinonasal disease is typically divided into 4 broad classes based on pathophysiology: acute invasive (fulminant) sinusitis, chronic invasive sinusitis, noninvasive fungal colonization (or mycetoma), and allergic mycotic sinusitis. Fungal hyphal forms are the most common causes of acute invasive sinusitis, with the most frequent being that of *Aspergillus* species. Mucormycosis is the next most common, with especially high prevalence in persons with diabetes, and is caused by the genus *Mucor*, belonging to the class of Zygomycetes. Members of this class thrive in high-sugar environments and are thus also found in decaying fruit. Although less common than hyphal forms, yeast forms occur in immunocompromised hosts and include *Candida*, *Histoplasma*, *Cryptococcus*, and *Coccidioides*.

Common CT features of invasive fungal sinusitis include soft tissue changes in the sinus with high-density opacification. In chronic infection, there is often thickened reactive bone, whereas acute invasive sinusitis often demonstrates focal bony erosion.¹⁹ Mycetoma has a characteristic hypointensity on T2-weighted magnetic resonance (MR) images. Mucormycosis has a predisposition for the orbits, cavernous sinuses, and skull base (**Figs. 8 and 9**) and also perineural invasion.

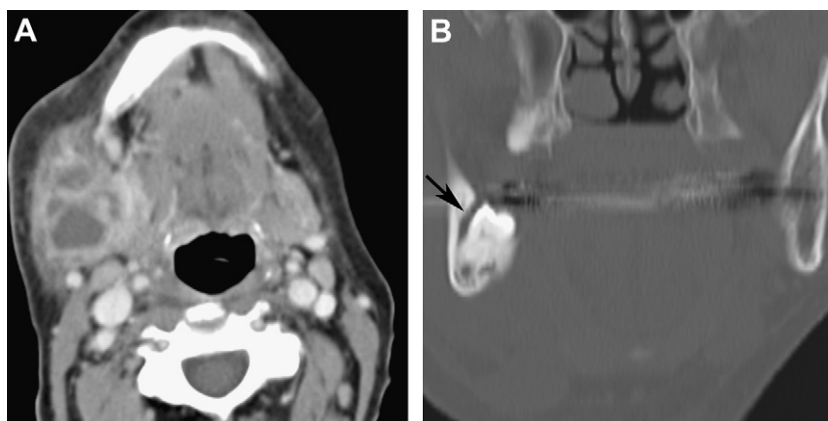


Fig. 6. Odontogenic submandibular space abscess. (A) Axial contrast-enhanced computed tomography demonstrates multiple low-density collections in the right submandibular space with circumferential rim enhancement as well as enlargement of the submandibular gland. (B) Coronal reformation with the bone window demonstrates the underlying periodontal disease (arrow).

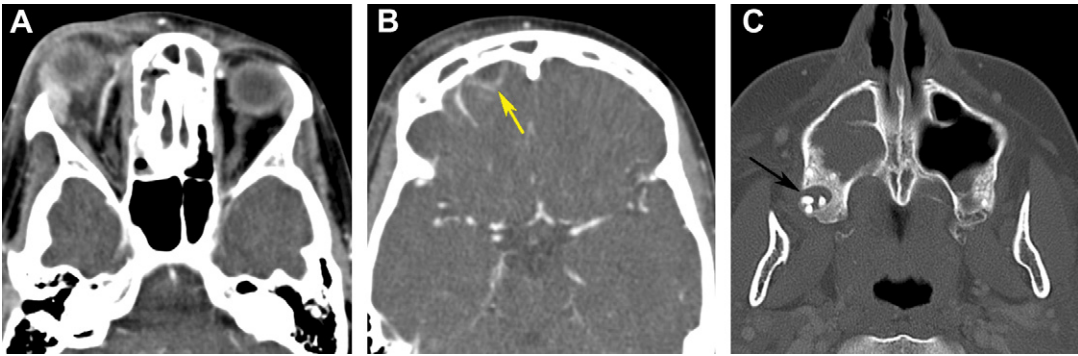


Fig. 7. Odontogenic orbital infection and epidural abscess. (A) Axial contrast-enhanced CT (CECT) through the right orbit demonstrates orbital cellulitis and phlegmon. (B) Axial CECT demonstrates an epidural abscess of the right frontal lobe (*arrow*). (C) Axial CECT demonstrates periapical lucency in a right maxillary molar, which was the source of infection in this patient (*arrow*). This patient also had odontogenic sinusitis of the maxillary sinus.

Sialolithiasis/Sialadenitis

Sialolithiasis refers to salivary gland stones or calculi and is the most common benign condition affecting the salivary glands. The submandibular gland is by far most commonly affected because its secretions are more mucous, alkaline, and viscous than that of the other glands.²⁰ Most stones are radiopaque, but CT is more sensitive than radiography to demonstrate sialolithiasis and its complications, including sialadenitis and abscesses.

Sialadenitis, or glandular inflammation of the salivary glands, is most commonly caused by sialolithiasis. Other inflammatory conditions that cause sialadenitis include mumps, AIDS, influenza, and infection with Coxsackie viruses. Bacterial infections, granulomatous disease, and idiopathic parotitis are less common. On CT, the gland demonstrates enlargement, hypodensity

on precontrast scan, and avid postcontrast enhancement, with adjacent inflammatory stranding and fascial thickening. The salivary duct may be dilated and thick-walled, and an obstructing sialolith is usually seen (**Fig. 10**). On MR imaging, gland enlargement and hyperintensity on T2-weighted images are because of inflammation and edema. Although the sialolith is not as well appreciated, its presence may be inferred if a punctate signal void is seen on all sequences.

Orbital Infection

Orbital subperiosteal abscess is a form of post-septal orbital cellulitis in which there is inflammatory tissue and edema formation beneath the orbital periosteum. This form of orbital cellulitis should be suspected in patients presenting to the emergency department with rhinosinusitis and ocular symptoms. Sinusitis of the ethmoid

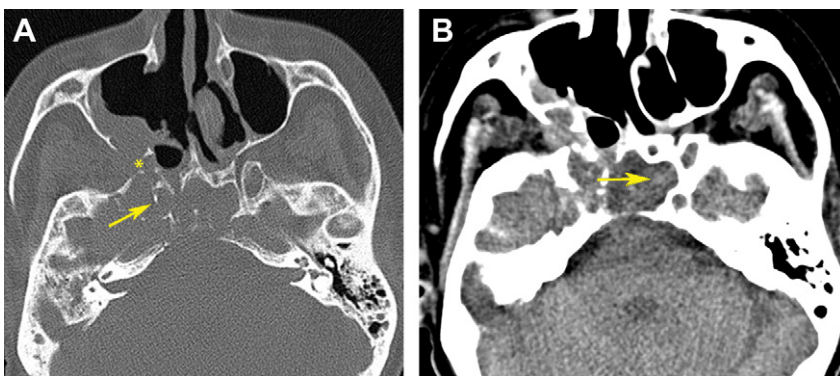


Fig. 8. Fungal sinusitis. (A) Axial contrast-enhanced CT in the bone window demonstrates soft tissue opacification of the right maxillary and sphenoid sinus with extensive osseous destruction of the central skull base (*arrow*). There is erosion of the posterior wall of the right maxillary sinus, the pterygopalatine fossa (*asterisk*), the sphenoid bone, the sella, and the right carotid canal. (B) Soft tissue window demonstrates hyperdense contents of the sphenoid sinus (*arrow*).

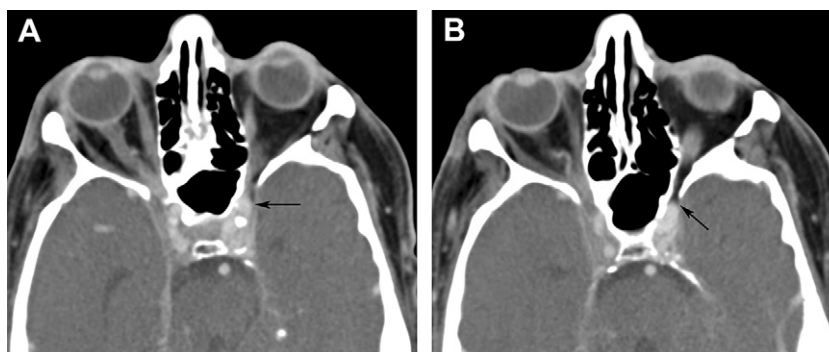


Fig. 9. Fungal sinusitis. (A) Axial contrast-enhanced CT (CECT) demonstrates enlargement and enhancement of the left cavernous sinus (arrow). (B) Axial CECT also demonstrates involvement of the left orbital apex (arrow).

sinus is a common predisposing condition, with the lateral aspect of the ethmoid sinus being a commonly involved site.²¹ An odontogenic source of infection is a less common cause.²² CT findings include a soft tissue mass extending from the bony wall of the orbit causing the displacement of the extraocular muscles. There may be a thin strip of preserved extraconal fat separating the abscess from the medial rectus muscle (**Fig. 11**). Clinically, there may be orbital swelling, chemosis, proptosis, and progressive impairment of extraocular motility. If these conditions are allowed to progress, ophthalmic vein thrombosis and cavernous sinus thrombosis are associated complications.



Fig. 10. Sialadenitis. Axial contrast-enhanced CT demonstrates enlargement and hyperenhancement of the right parotid gland (arrow), dilation of Wharton duct with ductal wall thickening (white arrowheads), and an obstructing sialolith (black arrowhead). Also note the adjacent soft tissue inflammatory change and fascial thickening.

Coalescent Mastoiditis

Mastoiditis most commonly occurs as a result of infection of the middle ear, with the route of spread involving the aditus ad antrum, the mastoid antrum, and eventually the mastoid air cells. Mastoiditis can less frequently be the result of the complication of a cholesteatoma. Destruction of the septa of the mastoid air cells signals the transformation of the incipient mastoiditis to the coalescent form, which portends a worse prognosis. In addition, coalescent mastoiditis has several associated complications, including epidural abscess, sigmoid sinus thrombosis, petrous apicitis, subdural empyema, carotid artery spasm or arteritis, and Bezold abscess.²³ On CT, coalescent mastoiditis demonstrates destruction of the mastoid air cell septa. If the mastoid tip suffers osteolysis, phlegmon and eventually abscess may organize with extent into the cervical soft tissues inferiorly; the resultant abscess is termed Bezold abscess (**Fig. 12**).

Malignant Otitis Externa

Necrotizing, or malignant, otitis externa is an entity distinct from the other forms of ear infection. This form affects the external auditory canal (EAC), begins at the cartilaginous/bony junction, and results in purulent drainage. Most cases occur in elderly persons with diabetes, and the causative agent is *Pseudomonas* species. HIV-positive patients are also at an increased risk. It is important to recognize and treat the infection early because it can spread to the nasopharynx, infratemporal fossa, parapharyngeal space, temporomandibular joint, middle and inner ears, and skull base, manifesting as skull base osteomyelitis.

CT is the imaging modality typically performed first for diagnosis.²⁴ On CT, malignant otitis externa manifests as soft tissue in the EAC, middle ear cavity, and mastoids. There is bony erosion of the

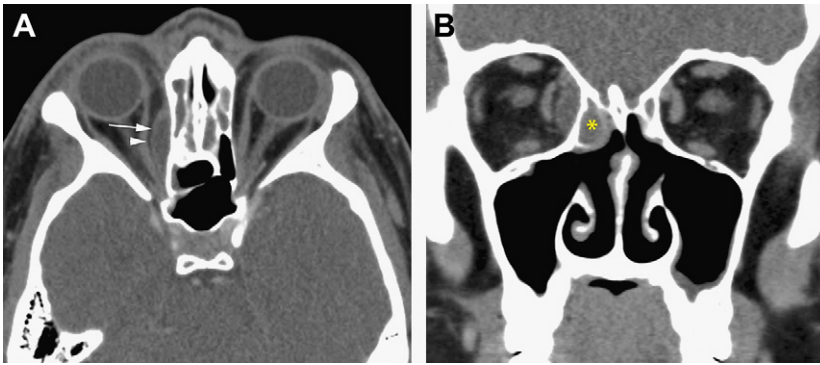


Fig. 11. Orbital subperiosteal abscess. (A) Axial contrast-enhanced CT demonstrates opacification of the ethmoid sinuses and a low-density collection along the medial aspect of the right orbit with a thin peripheral enhancement (*arrow*). Note the sparing of a thin stripe of extraconal fat (*arrowhead*) and lateral deviation of the right medial rectus muscle. (B) Coronal reformation demonstrates the extent of subperiosteal abscess and also a mucocele of the right ethmoid sinus (*asterisk*).

EAC, which, when extensive, may extend to the skull base (**Fig. 13**). On MR imaging, there is extensive edema and fluid in the EAC, middle ear, and mastoid air cells; soft tissue infiltration of the skull base and jugular foramen; and, occasionally, formation of an epidural or prevertebral abscess.

Ludwig Angina

Ludwig angina is a submandibular space infection originally described by von Ludwig²⁵ in 1836 as a "...gangrenous induration of the connective tissues of the neck which advances to involve the tissues that cover the small muscles between the larynx and the floor of the mouth."

The clinical presentations are usually cellulitis, sublingual/submandibular space fasciitis, and involvement of muscle and connective tissue, but without actual submandibular sialadenitis. There is a tender, firm anterior neck edema without

fluctuance. Other signs and symptoms include drooling, tachypnea, dyspnea, and stridor. The cause is odontogenic in 80% to 90% of patients.²⁶ Although Ludwig angina is a cellulitis with diffuse infiltration, subsequent abscess formation is not uncommon. The mode of spread is typically direct, rather than lymphatic.

Ludwig angina may result in a life-threatening airway compromise. In a review of 210 patients with deep neck abscess, overall 20% of patients required tracheostomy as compared with 75% of patients with Ludwig angina.²⁷ CT may demonstrate either a diffusely infiltrative process or a more discrete low-attenuation area, reflecting abscess (**Fig. 14**).²⁸

Peritonsillar Abscess

Peritonsillar abscess, sometimes referred to colloquially as "quinsy," is the most common deep

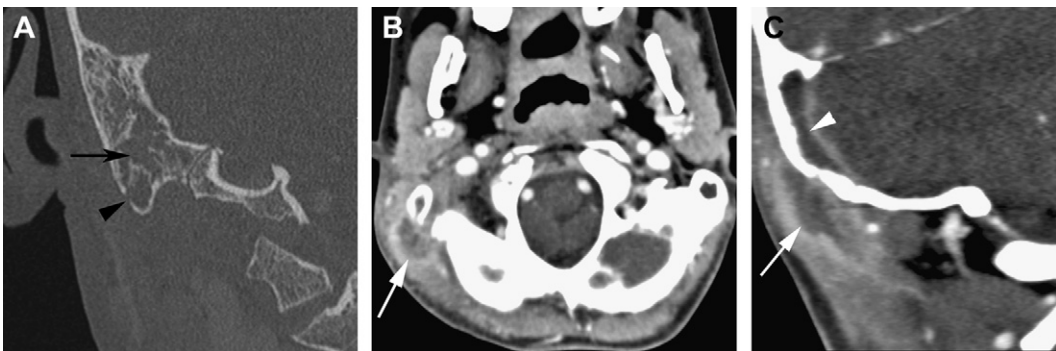


Fig. 12. Coalescent mastoiditis. (A) Coronal bone reformation CT image demonstrates extensive opacification and coalescence of septa within the right mastoid air cells (*arrow*). Note the cortical disruption of the mastoid tip inferiorly (*arrowhead*). (B, C) Axial and coronal contrast-enhanced CT with soft tissue windows demonstrate a low-density fluid collection inferior to the mastoid tip with a rim of enhancement indicating Bezold abscess (*arrows*). Also note right sigmoid sinus thrombosis as a complication of mastoiditis (*arrowhead*).

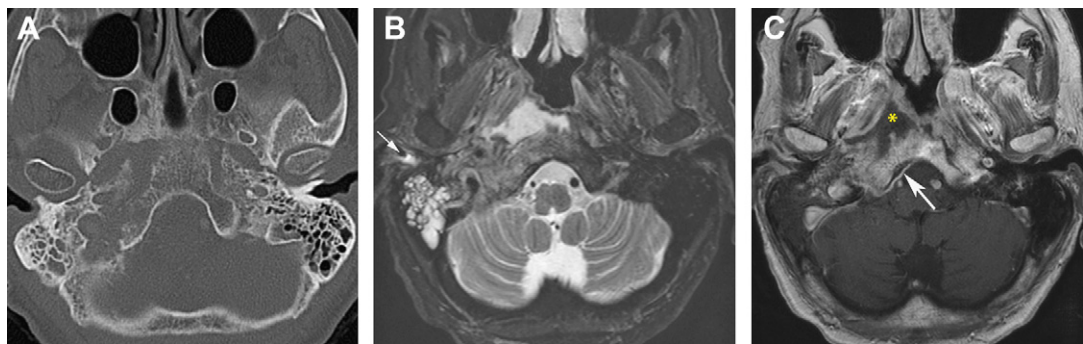


Fig. 13. Malignant otitis externa. (A) Axial contrast-enhanced CT with bone windows demonstrates osteomyelitis involving the right skull base. The clivus, right petrous apex, and jugular foramen demonstrate lytic change. (B) Axial T2-weighted MR image demonstrates edema involving the right temporal bone, including the EAC (arrow), skull base, jugular foramen, and prevertebral space. (C) Axial T1 postcontrast MR image demonstrates enhancement of the same regions. There is an abscess in the prevertebral space (asterisk). Also note an epidural abscess along the clivus (arrow). Inflammatory changes surround the internal carotid artery on the right.

neck infection.² Peritonsillar abscess is often polymicrobial. The most common aerobic bacteria are group A *Streptococcus*, *S aureus*, and *Haemophilus influenzae*, whereas the most common anaerobic bacteria are *Fusobacterium*, *Peptostreptococcus*, and pigmented *Prevotella*.²⁹ The presentation often includes fever, malaise, dysphagia, odynophagia, trismus, and “hot-potato” voice. On physical examination, bulging of the superior tonsillar pole and soft palate and deviation of the uvula are often seen. Inflammation of the tonsils is referred to as tonsillitis, which causes a typically striated appearance with the enhancement of the mucosa and submucosal edema (Fig. 15). Anatomically, peritonsillar abscess represents an infection between the tonsillar capsule and superior constrictor muscle (Fig. 16) and is usually a complication of

acute tonsillitis. Recently, inflammation of the Weber glands,²⁹ which are approximately 2 dozen mucous salivary glands in the soft palate just superior to the tonsil, has also been implicated in the pathogenesis of peritonsillar abscess. Thus, peritonsillar abscess is also possible in patients who have had tonsillectomy. If infection spreads outside the constrictor ring, it represents a parapharyngeal abscess (Fig. 17).

Retropharyngeal and Danger Space Abscess

The retropharyngeal space extends from the skull base to approximately T3-T4 and is bounded anteriorly by the slip of the middle layer of the deep cervical fascia known as the visceral layer. Posteriorly, the retropharyngeal space is bound by a slip of the deep layer of the deep cervical fascia known

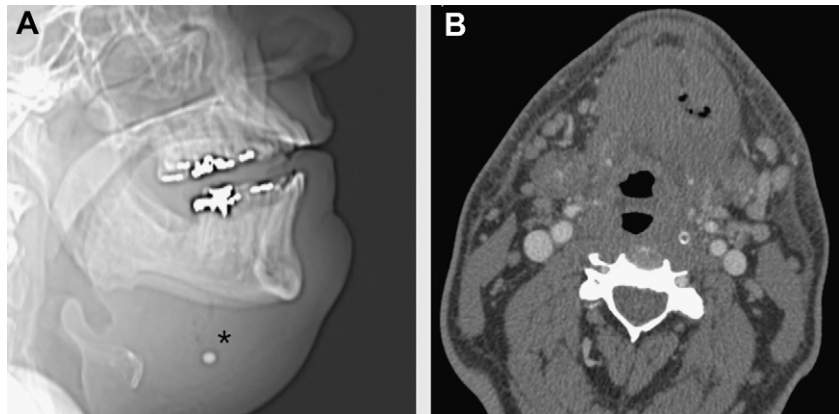


Fig. 14. Ludwig angina. (A) Scout CT view demonstrates submandibular space soft tissue swelling and small locules of gas (asterisk). (B) Axial contrast-enhanced CT demonstrates diffuse infiltration of the sublingual and submandibular spaces, small locules of gas, and thickening of platysma and fascial planes. The airway is mildly effaced. No discrete abscess is seen.

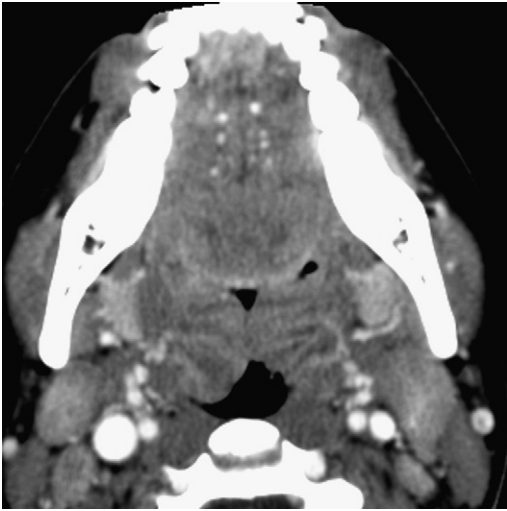


Fig. 15. Tonsillitis. Axial contrast-enhanced CT demonstrates enlargement of the palatine tonsils, with a striated appearance representing mucosal enhancement. Low-density areas between the striations represent parenchymal edema.

as the alar fascia. Immediately posterior to the alar fascia is the danger space (see **Fig. 1**), which extends inferiorly into the posterior mediastinum. An infectious process in these compartments can be seen in a spectrum ranging from phlegmon to suppurative adenitis/frank abscess (**Fig. 18**). It is not possible to differentiate the retropharyngeal space from the danger space on imaging (**Fig. 19**), and danger space infection can be inferred only if



Fig. 16. Peritonsillar abscess. Axial contrast-enhanced CT demonstrates a low-density fluid collection with rim enhancement along the lateral tonsillar wall on the right (arrow).

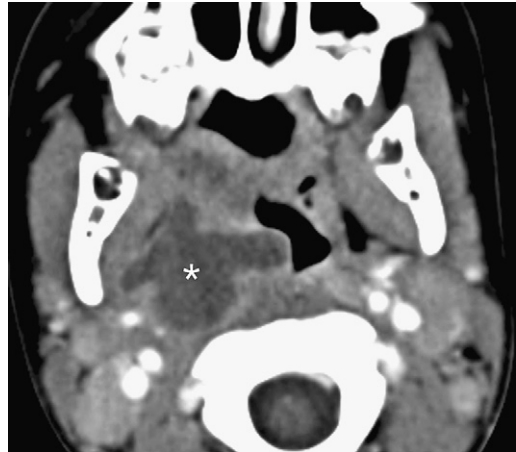


Fig. 17. Parapharyngeal abscess. Axial contrast-enhanced CT demonstrates a low-density collection of the right parapharyngeal space (asterisk) caused by peritonsillar abscess, which has extended beyond the constrictor ring.

the infection spreads below the T4 level. Retropharyngeal abscess may extend posteriorly to involve the danger and prevertebral spaces as well as cause osteomyelitis of the spine.

Unlike peritonsillar abscess, which is medial to the constrictor ring, parapharyngeal and retropharyngeal abscesses are lateral. The most common sources for retropharyngeal abscess in children is suppurative adenitis.³⁰ Cervical spine surgery is another potential cause of infection of



Fig. 18. Suppurative adenitis of a retropharyngeal node. Axial contrast-enhanced CT demonstrates a low-density lymph node (arrow) in the left retropharyngeal space indicating suppurative adenitis causing abscess.

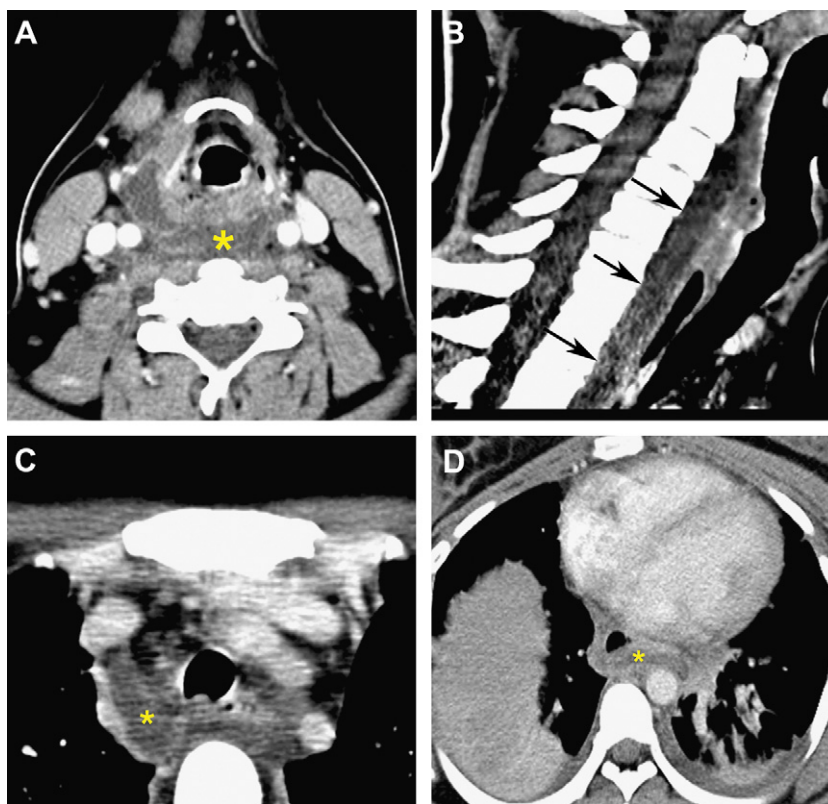


Fig. 19. Danger space abscess. (A) Axial contrast-enhanced CT demonstrates fluid in the retropharyngeal and danger spaces at the level of the hyoid bone (asterisk). (B) Sagittal reformation through the midline subglottic neck demonstrates the cervical danger space abscess (arrows). (C, D) Axial CT images at the level of the aortic arch and diaphragm demonstrate the most inferior extent of the abscess, with both right paratracheal and posterior mediastinal components (asterisks).

the prevertebral/retropharyngeal spaces. It is difficult to differentiate suppurative adenitis of the retropharyngeal nodes from frank abscess (see the section Abscess vs Phlegmon).

Calcific tendonitis affecting the longus coli muscle was first described by Hartley³¹ in 1964. The clinical presentation for this condition is varied, but there is generally no history of head/neck infection and no cervical lymphadenopathy. There may be a mild leukocytosis and/or an elevated erythrocyte sedimentation rate.³² The classic radiographic description is an amorphous calcification anterior to the C1-C4 vertebral bodies with prevertebral soft tissue swelling. However, radiography is unreliable for the diagnosis, and CT has long been considered the preferred modality.³³ On CT, an amorphous calcification is seen more clearly, is localized to the prevertebral space, and is reliably differentiated from an avulsed fracture (Fig. 20). A diffuse edema or discrete effusion can be differentiated with MR imaging,³⁴ although this distinction is usually not necessary. However, differentiating this entity

from a retropharyngeal abscess is necessary to avoid drainage or surgery. After treatment with nonsteroidal antiinflammatory drugs, this condition is self-limited in about 1 to 2 weeks.³⁵

Necrotizing Fasciitis

Necrotizing fasciitis is an aggressive infection of both the superficial and deep soft tissues of the neck. Both aerobic and anaerobic bacteria can act as so-called flesh-eating bacteria. Skin, mucosal, dental, or tonsillar sites of origin may be identified, and overall necrotizing fasciitis occurs more frequently in immunocompromised patients. Complications include sepsis, making prompt surgical exploration necessary. At times, it may be difficult to differentiate necrotizing fasciitis from a less-aggressive infection, but the presence of gas-forming organisms is a clue.

Diffuse thickening of the cutis and subcutis and reticulation of fat indicate cellulitis. Enlargement, thickening, and enhancement of fasciae indicate fasciitis. Enlarged edematous muscle indicates

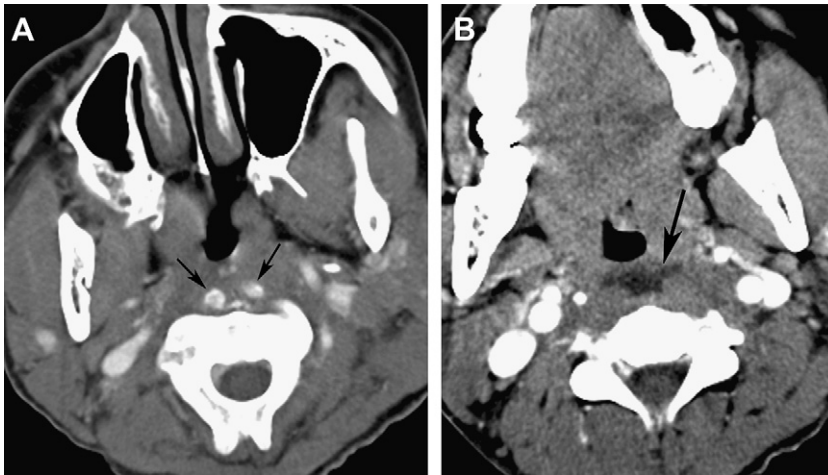


Fig. 20. Calcific tendonitis of the longus colli tendon. (A) Axial contrast-enhanced CT image demonstrates amorphous calcification of the longus colli tendon (arrows). (B) There is an associated effusion in the retropharyngeal space, without enhancement (arrow).

myositis. Fluid collections in multiple spaces indicate soft tissue necrosis (**Fig. 21**).³⁶ Airway compromise can occur with severe infection.

The visceral layer of the deep cervical fascia surrounds the thyroid, trachea, anterior wall of the esophagus, and thyroid cartilage to the superior mediastinum, defining the anterior visceral space. Most infections involving this space result from the perforation of the esophagus, with risk factors being instrumentation, foreign body, or trauma. Infection of the visceral space portends an increased risk of mediastinitis and airway compromise.

Although the most common cause of acute mediastinitis is esophageal rupture, descending

necrotizing fasciitis from DNSI traveling along the retropharyngeal space, danger space, carotid space, prevertebral space, or anterior visceral space can also spread into the mediastinum (see **Fig. 19B**). The most common source of descending cervical mediastinitis is odontogenic infection.³⁷ Comorbid conditions such as diabetes may make certain patients highly susceptible to spreading cellulitis. Mediastinitis may also result from direct extension from an adjacent source of infection, including osteomyelitis of the sternoclavicular joint, pulmonary infections, and spread of granulomatous disease from mediastinal lymph nodes. The treatment is IV antibiotics, and because mediastinitis is a surgical emergency, surgical debridement, drainage, and airway management are typically necessary.³⁷

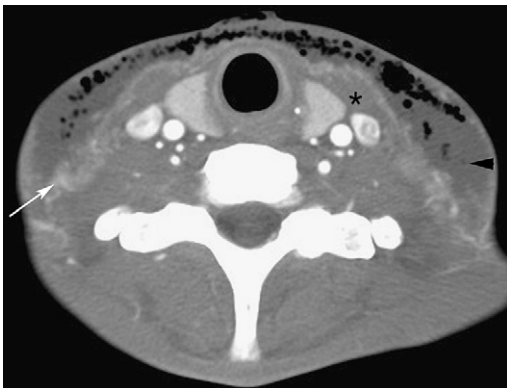


Fig. 21. Necrotizing fasciitis. Axial contrast-enhanced CT at the level of the thyroid gland demonstrates subcutaneous gas in the anterior neck with low-density appearance of the sternocleidomastoid muscle indicating myositis (asterisk) as well as enhancement of the fascia (arrow) and fluid collections (arrowhead).

Epiglottitis

Acute epiglottitis in adults is a cellulitis of the epiglottis, aryepiglottic folds, and adjacent tissues (**Fig. 22**). If not treated promptly, this condition can progress to a life-threatening airway obstruction. Epiglottitis results from bacteremia or direct invasion of the epithelial layer by the pathogenic organism, which typically travels from the posterior nasopharynx. Although the incidence of epiglottitis in children has decreased because of the *H influenzae* type b (Hib) vaccination, this bacterium is still the most common causative organism.³⁸ The incidence of epiglottitis in adults appears to be increasing.^{39,40} Adult epiglottitis may represent a distinct form of epiglottitis because the supraglottic tissues are involved in most cases, and culture yields are uniformly lower

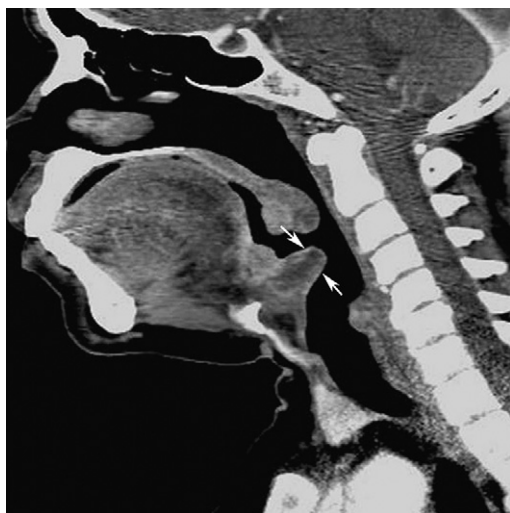


Fig. 22. Epiglottitis. Sagittal contrast-enhanced CT demonstrates edema and swelling of the epiglottis compatible with epiglottitis (arrows). (Courtesy of Dr Robert Morales University of Maryland Medical Center, Baltimore, MD.)

in cases of this condition than in traditional pediatric cases of acute epiglottitis. Other causative organisms are *Streptococcus* and *Staphylococcus* in immunocompetent hosts and *Pseudomonas* and *Candida* in immunocompromised hosts.

Laryngopyocele

Laryngopyocele represents infection of a dilated laryngeal ventricle. This entity is thought to arise in a preexisting laryngocele with stasis, resulting in secondary infection. The incidence is low and has been reported as 1 in 2.5 million in the United Kingdom.⁴¹ Although a rare diagnosis, laryngopyocele can present as an acute airway emergency,⁴² and thus it is important to recognize this condition early. Clinical presentation may include odynophagia, aspiration, and respiratory compromise. Intubation or tracheostomy, use of broad-spectrum antibiotics, and needle or surgical decompression are considered necessary in the treatment of laryngopyocele. CT demonstrates a fluid or mixed fluid-air density collection emanating from the laryngeal ventricle, with extension superolaterally into the paraglottic fat. Thick rim enhancement of the walls indicates inflammation (**Fig. 23**).

VASCULAR COMPLICATIONS OF HEAD AND NECK INFECTIONS

Head and neck infections can also result in numerous vascular complications, especially when the infection involves the parapharyngeal



Fig. 23. Laryngopyocele. Coronal contrast-enhanced CT demonstrates a mixed internal and external laryngopyocele (asterisk) with rim enhancement and edema of the glottis and supraglottis.

space. Major vascular complications include Lemierre syndrome, cavernous sinus thrombosis, and arterial complications, such as vasospasm, arteritis, pseudoaneurysm, and carotid blowout.

Lemierre Syndrome

In 1936, Andre Lemierre reported a syndrome of anaerobic septicemia that resulted from infection in 1 of 6 possible sites and caused septic thrombophlebitis and metastatic abscesses. The first of these sites was the "...nasopharynx, particularly tonsillar and peritonsillar abscesses."⁴³ Although other sites were as remote as the endometrium in postpartum women, the term Lemierre syndrome has since been used to describe a narrower clinical syndrome resulting from head and neck infections in which venous thrombophlebitis and septicemia occur with or without metastatic abscesses. Most cases that Lemierre described in Paris in the 1930s were caused by *Bacillus funduliformis* (now *Fusobacterium necrophorum*), but he also described "...several species of anaerobic organisms which are specifically distinct from one another." Lemierre's original description of the progression of infection was "...thrombophlebitis of the tonsillar and peritonsillar veins which can spread to the internal jugular vein or even to the facial vein."



Fig. 24. Lemierre syndrome. Axial contrast-enhanced CT demonstrates septic thrombophlebitis of the right internal jugular vein with thickening and enhancement of the walls of the thrombosed vein and surrounding tissues (arrow).

Because Lemierre syndrome was thought to be uncommon in the postantibiotic era, it was termed in 1984 as the “forgotten disease”.⁴⁴ However, recent reviews of the literature suggest an increasing incidence.^{45,46}

Often, those affected are previously healthy immunocompetent adolescents and young adults, with a male predominance. The offending organism is most commonly *Fusobacterium* anaerobic gram-negative bacilli, which are part of the normal flora of the mouth. Predisposing factors include trauma, edema, anoxia, or tissue

destruction after dental disease. A typical lag time is 4 to 8 days between the primary event and septicemia.⁴³ Imaging demonstrates enlargement and thrombosis of the internal jugular vein, with enhancement of the walls of the vein and perivenular soft tissues (Fig. 24). Metastatic abscesses may also occur with superficial thrombophlebitis without frank internal jugular vein thrombophlebitis (Fig. 25).

Cavernous Sinus Thrombosis

The cavernous sinus is a venous space, which is part of the anteroinferior group of dural venous sinuses. The cavernous sinus drains the insular cortex and parenchyma around the sylvian fissure via the sphenoparietal sinus. Each cavernous sinus communicates with the contralateral side and the pterygoid plexus as well as the superior ophthalmic vein. Because these sinuses anastomose with the facial veins, which are valveless, paranasal, odontogenic, and facial infections (eg, a furuncle that was manipulated) can spread into the cavernous sinus (Fig. 26). Patients with cavernous venous sinus thrombosis often have a history of midface infection for 5 to 10 days. Presenting signs and symptoms include headache, fevers, and cranial nerve signs. The patient may have orbital pain, visual disturbances, and fullness with periorbital edema or discrete abscess. Other symptoms include confusion, drowsiness, and coma as a result of central nervous system involvement and/or sepsis. Although there are only small series that compare CT with MR imaging for diagnosing cavernous sinus thrombosis,⁴⁷ CT venography seems to be superior to

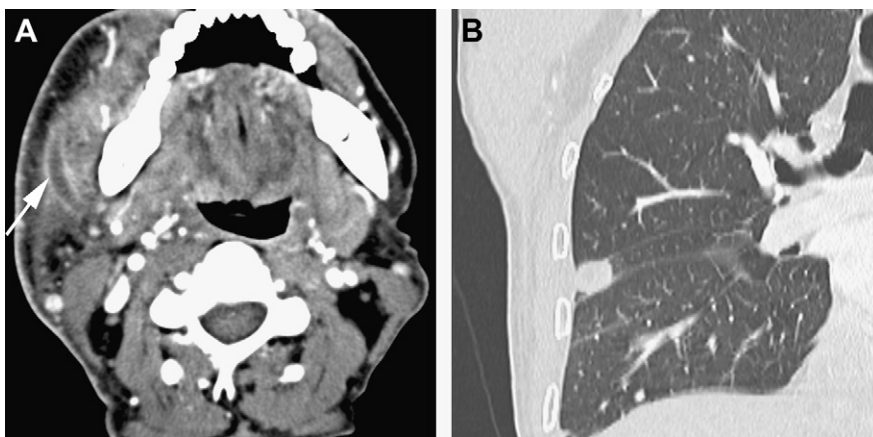


Fig. 25. Odontogenic septic thrombophlebitis without internal jugular vein (IJV) thrombosis. (A) Axial contrast-enhanced CT demonstrates odontogenic masticator and parotid space infection with septic thrombophlebitis of the retromandibular vein (arrow). (B) Coronal reformation with lung windows demonstrates resulting septic pulmonary embolus. Note that these complications may occur without demonstrated IJV thrombosis as in this case.



Fig. 26. Cavernous sinus thrombosis. (A, B) Axial postcontrast CT and T1-weighted MR images demonstrate enlargement and filling defects (arrows) in the cavernous sinuses bilaterally, indicating cavernous sinus thrombosis. There is enlargement of the right superior ophthalmic vein, indicating ophthalmic vein thrombosis with edema and proptosis of the right orbit.

MR venography in the identification of the cerebral venous sinuses and at least equivalent in the diagnosis of cerebral venous sinus thrombosis.⁴⁸ Matched mask bone elimination may help further automate CT venography and improve cavernous sinus evaluation.⁴⁹ In the authors' experience, CT venography or routine CT of the skull base with a contrast is a reliable tool for the evaluation of cavernous venous sinus thrombosis. Findings include the enlargement of the cavernous sinus and presence of filling defects representing thrombosis (see Fig. 26). The superior ophthalmic vein may also be enlarged and thrombosed.

Arterial Complications

Vasospasm is a common finding in which there is narrowing of the ipsilateral internal carotid artery related to the retropharyngeal lymphadenitis and abscess (Fig. 27). However, this condition has not been associated with neurologic deficits and is generally regarded as benign.⁵⁰

True arterial complications of head and neck infection were reported more frequently before the 1930s, with a dramatic decrease of these complications in the more recent literature.⁵¹ Arteritis represents an inflammation of the internal carotid artery but is not associated with narrowing. The only histologically reported confirmation of arteritis due to deep neck infection does not include a description of the layers involved.⁵²

If pseudoaneurysm complicates arteritis, the patient may develop a pulsatile mass, pain, and cranial neuropathies. *Salmonella* and *Klebsiella* produce elastase and may facilitate the formation of pseudoaneurysms (Fig. 28).⁵³

Carotid blowout represents a life-threatening hemorrhage of the internal carotid artery as

a complication of deep neck infection.⁵⁴ The traditional therapies are carotid ligation or balloon occlusion. More recently, stent-graft therapy has been attempted and may be a temporizing measure, but the long-term safety and patency of this therapy are not established. Also, many of the published data are in patients with a history of head and neck cancer who have undergone radiation therapy.⁵⁵

ABSCESS VERSUS PHLEGMON

One limitation of CT examination is in distinguishing phlegmon versus abscess. An abscess is defined as a drainable cavity of infected debris,

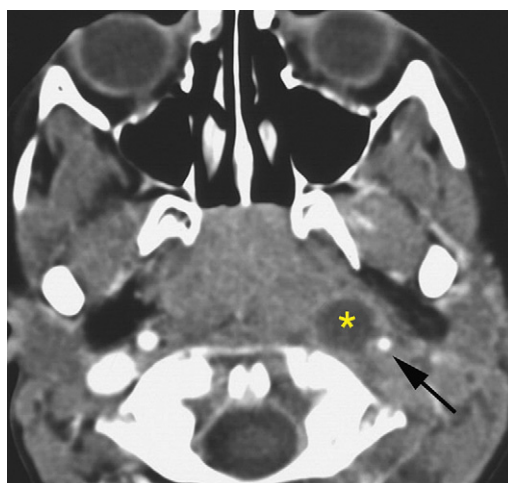


Fig. 27. Retropharyngeal abscess with internal carotid artery (ICA) spasm. Axial contrast-enhanced CT demonstrates retropharyngeal abscess (asterisk) on the left with associated narrowing of the left ICA (arrow).

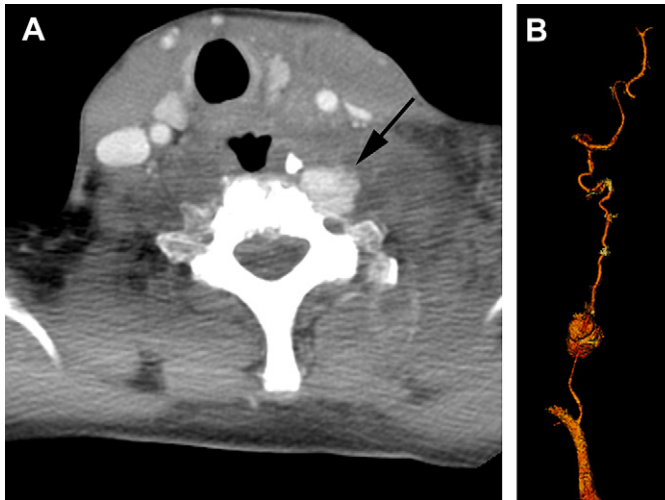


Fig. 28. Vertebral artery pseudoaneurysm. (A) Axial CT angiography source image demonstrates a left vertebral artery pseudoaneurysm (*arrow*) resulting from a neck abscess in a patient with perforated Zenker diverticulum. CT performed 1 week prior had shown a normal-caliber vertebral artery. (B) Volume-rendered image helps delineate the extent of pseudoaneurysm.

whereas a phlegmon is an infected tissue without necrosis and therefore without a drainable fluid collection. A fluid-attenuation collection with peripheral rim enhancement has been considered as the classic imaging finding for abscess on CT examination, whereas phlegmon has been described as a low-density edematous tissue without discrete peripheral enhancement. However, the use of these criteria is expected to

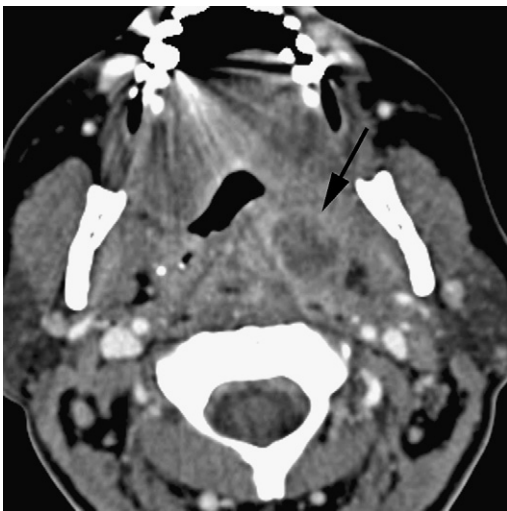


Fig. 29. Parapharyngeal space phlegmon. Axial contrast-enhanced CT demonstrates a low-density fluid collection with partial rim enhancement in the left parapharyngeal space (*arrow*) with a mass effect on the airway. Although the appearance suggested abscess, no pus was aspirated.

result in significant false-positive results (**Fig. 29**). The positive predictive value of these features has been reported as 71% to 83%.^{3,56,57} Therefore, CT offers the most value when used to exclude abscess because negative predictive values are uniformly high if those in whom no pus was obtained or who recovered without surgical management are considered not to have had clinically relevant abscess. Thus, the decision for drainage should be made clinically. However, information useful in guiding drainage procedures can be obtained, and the presence of complications can be assessed by CT. Indications for drainage include continued fevers, lack of improvement or worsening clinically, and continued leukocytosis despite antibiotics. One study analyzed the false-positive results of CT by size and found that they all occurred in collections less than 3.5 cm.⁵⁸ Therefore, a low-density collection of more than 3.5 cm with rim enhancement should confidently suggest the presence of drainable pus at surgery.

SUMMARY

The signs, symptoms, and complications of the infectious and inflammatory disorders of the head and neck are determined largely by the anatomic compartment of origin. The fascial layers serve as important barriers to infection and often determine the path of the spread of infection. Cervical lymphadenitis is common in children and is the usual origin of superficial neck abscesses. Adults are more likely than children

to have a DNSI, with odontogenic infection being the most common source. There are specific risk factors that can raise the suspicion for DNSI. Multiple routes of spread are possible from odontogenic infection, which can commonly result in the involvement of the buccal, masticator, parapharyngeal, submandibular, and sublingual spaces. Odontogenic sinusitis, osteomyelitis, and orbital infections are additional potential complications of odontogenic infection.

Invasive fungal sinusitis may be suspected clinically or based on imaging features. Mucormycosis has a special predisposition for orbital, cavernous sinus, skull base, and perineural involvement. Sialolithiasis is the most common cause of sialadenitis, which by far most commonly involves the submandibular gland, with diagnostic imaging findings. Orbital subperiosteal abscess should be suspected when rhinosinusitis occurs with ocular symptoms, with the medial orbit being a commonly affected site. The coalescent form of mastoiditis must be recognized, which has specific associated complications with characteristic imaging features. Malignant otitis externa is distinct from other forms of ear infection and has specific patterns of spread and several known complications, which can be evaluated on CT or MR imaging.

Ludwig angina is a submandibular/sublingual space infection, which is also usually odontogenic. Peritonsillar abscess is the most common DNSI and can also result in parapharyngeal abscess. Retropharyngeal and danger space infections have similar imaging findings, but danger space infection can be inferred when there is spread of infection below T4. Longus colli calcific tendonitis can mimic retropharyngeal abscess but has specific imaging findings that should allow its differentiation. Necrotizing fasciitis is a serious infection often signaled by the presence of gas-forming organisms and requires early recognition and treatment. Mediastinitis usually results from esophageal rupture but can also be a complication of DNSI and in either case can result in a life-threatening airway compromise. Epiglottitis is a cellulitis of the epiglottis and aryepiglottic folds, which can lead to a life-threatening airway obstruction. Epiglottitis in adults may be a distinct form, and its incidence appears to be increasing despite the decreased pediatric incidence as a result of the Hib vaccination. Laryngopyoceles are a rare but serious infection in a preexisting laryngocele and is differentiated by clinical data and an enhancing rim with inflammatory change.

Lemierre syndrome has a range of presentations and imaging findings, and despite once being referred to as the forgotten disease, the incidence

of this condition is increasing. Cavernous sinus thrombosis is a serious complication of DNSI, for which CT venography or routine CT of the skull base with contrast is appropriate for evaluation. Although less common than in the preantibiotic era, arterial manifestations of DNSI include vasospasm, arteritis, pseudoaneurysm, and carotid blowout. With the exception of vasospasm, these manifestations are potentially serious complications that require early recognition.

Although a useful tool in the evaluation of DNSI, contrast-enhanced CT is expected to yield up to a 25% false-positive rate for the diagnosis of abscess. This rate is lower when larger fluid collections are considered, and essentially all abscesses greater than 3.5 cm can be reliably differentiated from phlegmon.

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