ELSEVIER

Seminars in
ULTRASOUND
CT and MRI

Nontraumatic Vascular Emergencies of the Neck

John A. Coburn, MD, Edwarda Golden, MD, Justin Brucker, MD, and Tabassum A. Kennedy, MD

Nontraumatic vascular emergencies of the head and neck are uncommon, but can occur in patients with central venous catheters, head and neck infections, and in patients with head and neck cancer. Recognizing the imaging findings of vascular complications in these patient populations is critically important to ensure expeditious treatment to avoid significant morbidity and mortality.
Semin Ultrasound CT MRI 40:157-171 Published by Elsevier Inc.

Introduction

Nontraumatic vascular emergencies of the neck may affect the arterial and venous system and can be grouped into two broad categories including vascular occlusive disease and vascular rupture. In the acute setting, venous occlusion within the neck may occur from an indwelling central venous catheter, as propagation from a deep venous thrombosis with the arm or heart, from an infectious source originating from the neck, as direct extension of tumor into the vessel or as a secondary effect from external compression from a mass either within the neck or mediastinum. Nontraumatic arterial occlusion within the neck may result from infectious arteritis or potentially from acute thromboembolism, which may be accelerated in patients treated for head and neck cancer (HNC). Nontraumatic vascular rupture or carotid blow out is a life-threatening complication of head neck radiation and although rare, is critical to recognize. This review focuses primarily on cervical vascular emergencies in patients with catheter-related thrombosis, neck infections, and in those patients treated for HNC.

Anatomy

Arteries and veins within the neck transport blood toward and away from the head, face, and neck respectively with intervening capillary beds. Arteries have a thicker muscular wall, with a smaller rounded lumen than their venous counterparts, which is necessary in maintaining blood pressure and circulation. Venous structures on the other hand have much thinner walls with less vascular resistance and as a result tend to be more prone to compression and luminal compromise from external mass effect.

Arterial Anatomy

The common carotid is a large elastic artery that ascends anteromedial to the internal jugular vein within the carotid sheath, before bifurcating at the C3 or C4 vertebral level and giving rise to the muscular arterial divisions of the external carotid artery (ECA) and the more vertically-oriented internal carotid artery (ICA) (Fig. 1). While the internal carotid artery is chiefly responsible for supplying the majority of the anterior intracranial blood supply, it possesses numerous anastomotic connections with the external carotid and vertebrobasilar territories — thus allowing for potential collateralization between these arterial systems in the event of occlusive disease, or unintended embolization.[1] Despite its relative lack of arborization in the neck, the configuration of the distal cervical segment of the internal carotid artery is prone to mild complexity. Alteration in embryologic development may result in length discrepancy between the internal carotid arteries and neck, allowing for the development of loops or kinks.[2]

The external carotid artery provides blood supply to large areas of the face, scalp, jaw, and deep cervical spaces of the upper neck and skull base (Fig. 1). Perfusion to these areas is hemodynamically balanced across the ECA's various distal

Department of Radiology, University of Wisconsin School of Medicine and Public Health, Madison, WI.
Address reprint requests to Tabassum A. Kennedy, MD, Department of Radiology, Division of Neuroradiology, University of Wisconsin School of Medicine and Public Health, 600 Highland Avenue, Madison, WI 53792-3252. E-mail: tkennedy@uwhealth.org

https://doi.org/10.1053/j.sult.2018.10.008
0887-2171/Published by Elsevier Inc.

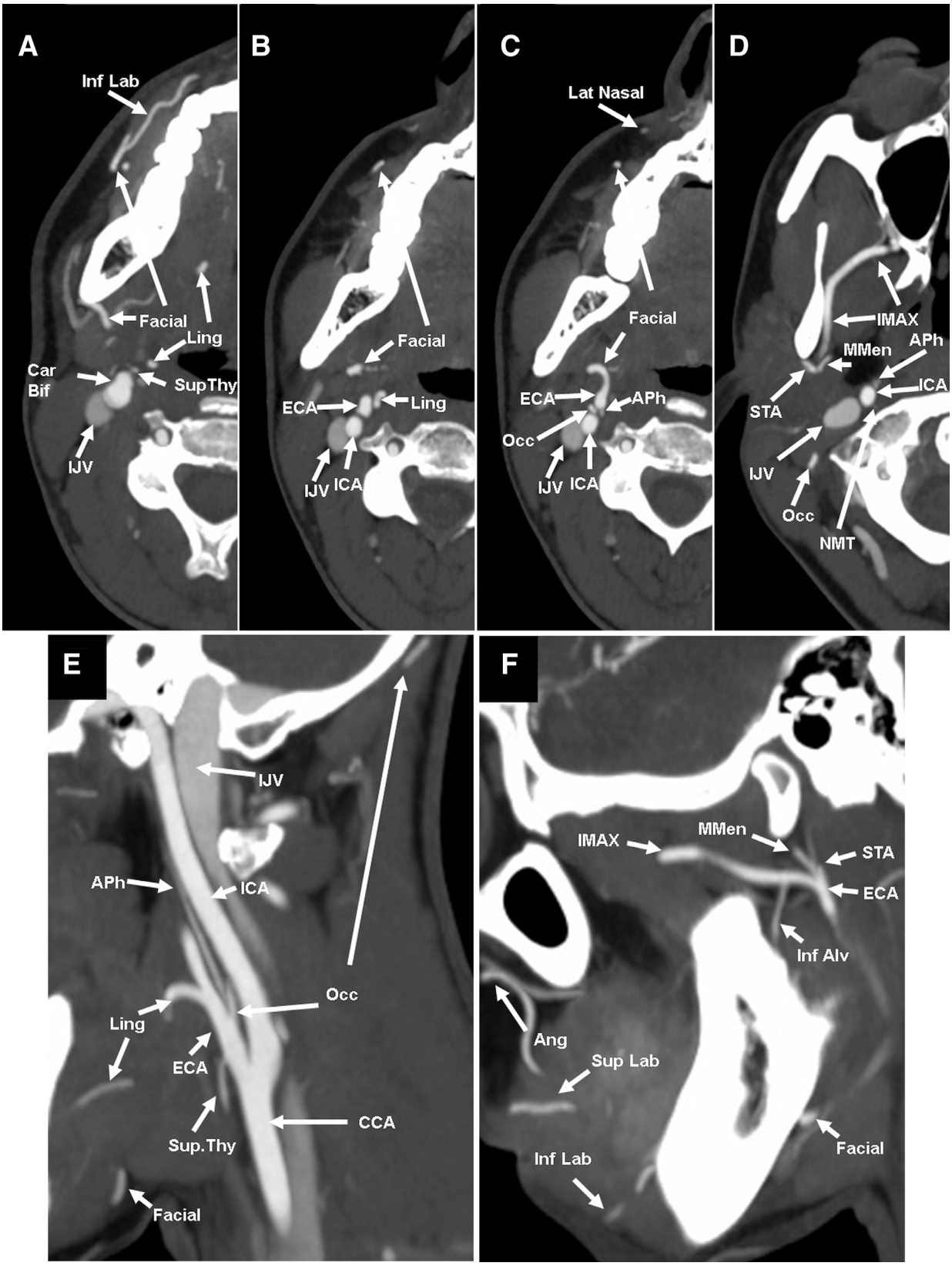

Figure 1 Axial (A-D) and sagittal (E-F) maximum intensity projection images (MIPs) from CT angiogram through the lower face and jaw. Ang, angular artery; APh, ascending pharyngeal artery; Car Bif, carotid bifurcation; CC, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery; IJV, internal jugular vein; Inf Lab, inferior labial artery; IMAX, inferior maxillary artery, Lat Nasal, lateral nasal artey; Ling, lingual artery; MMen, middle meningeal artery; NMT, neuromeningeal trunk; Occ, occipital artery; STA, superficial temporal artery; Sup Thy, superior thyroidal artery.

territories, which are interconnected via multiple bridging arcades — allowing not only for widespread variations in ECA branch anatomy, but also for robust collateral redistribution of blood flow in the case of segmental occlusion. By exception, the distal ranine branches of the lingual artery may be considered true terminal vessels of the ECA territory, as there is a lack of collateral flow to the tip of the tongue.

Although a complete discussion on all possible variations in ECA branch anatomy are beyond the practical limits of this section, it is useful to remember that the branches are usually named according to the anatomical regions they perfuse; therefore, tracing a vessel back from its target to its parent vessel may help delineate variations in vascular origin. The classic expected order of branches arising along the length of the external carotid artery are as follows: superior thyroidal, ascending pharyngeal, lingual, facial, occipital, posterior auricular, superficial temporal, and internal maxillary (Fig. 1).

Venous Anatomy

The internal jugular (IJ) is the primary venous structure that drains blood from the head, face, and neck (Fig. 2). It originates at the skull base within the jugular bulb from the sigmoid and inferior petrosal sinuses and continues inferiorly within the carotid sheath posterolateral to the carotid and vagus nerve. Although cervical venous anatomy is relatively inconsistent, the primary tributaries of the IJ vein are the retromandibular, facial, and lingual veins (Fig. 2).[3] The majority of venous drainage for the superficial and deep structures of the face and anterior scalp converges within the retromandibular vein before entering the internal jugular vein. Superficial venous drainage of the neck is primarily via the external jugular and anterior jugular veins, which descend within the superficial neck in a craniocaudal direction, running relatively parallel to the IJ (Fig. 2). The anterior jugular vein originates at the level of the hyoid bone from the submandibular and superficial facial veins descending medial to the sternocleidomastoid muscles and anterior to the strap musculature.[3] At the level of the mandible, the external jugular vein forms from the posterior auricular and retromandibular veins then courses obliquely along the neck superficial to the sternocleidomastoid muscle with additional distal contribution from the transverse cervical veins.[3] The internal jugular, anterior jugular, and external jugular all eventually drain into the subclavian vein.

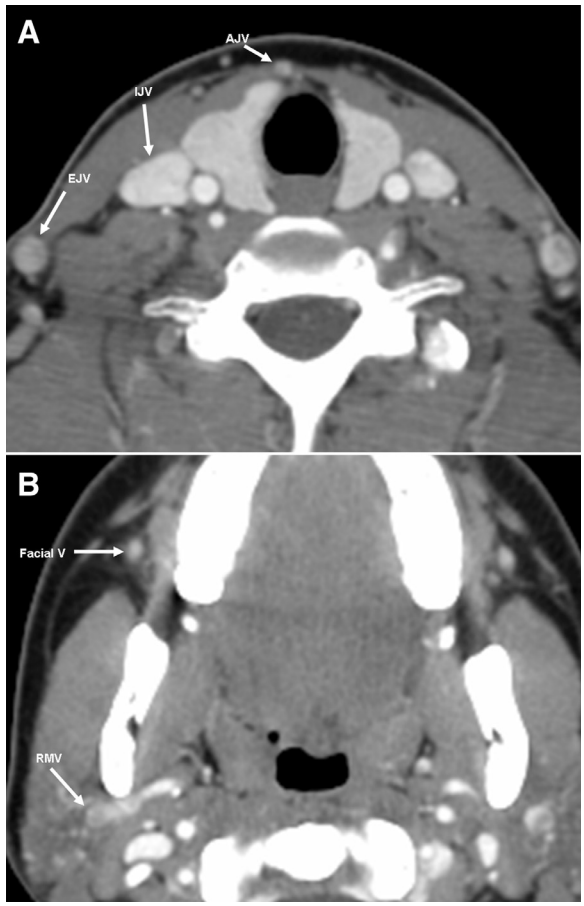

Figure 2 Axial images (A-B) from a contrast-enhanced CT through the neck and face. AJV, anterior jugular vein; EJV, external jugular vein; Facial V, facial vein; IJV, internal jugular vein; RMV, retromandibular vein.

Catheter-Related Thrombosis

Introduction and Epidemiology

Central venous catheters (CVC) are ubiquitous. They are necessary for the delivery of life-saving medications in intensive care unit patients and in patients receiving chemotherapy, long-term antibiotics, dialysis, blood transfusions, apheresis, and parental nutrition. Catheters can range by size, entry position, number of lumens, and valves. Although these catheters are medically necessary, catheter-related thrombosis (CRT) is a known common complication occurring in up to 28% of symptomatic patients and up to 66% of asymptomatic patients with central venous catheters.[4-6] Risk factors that may increase the incidence of CRT in patients with a CVC include those related to the catheter type, the insertion method, catheter position as well as patient factors related to immobility and hypercoagulable state.[4,6] CRT and should be suspected in the patients with systemic infection, which is an independent risk factor for the development of thrombosis.[4,6]

Pathophysiology

The development of thrombosis from an indwelling catheter is thought to arise from the combination of endothelial damage from the trauma of catheter insertion, venous stasis and hypercoagulable state — so called "Virchow's triad".[4,7] The tip of a CVC should ideally be placed at the junction between the superior vena cava and the right atrium. A mal-positioned catheter either too proximal or too distal may result in endothelial damage at the catheter tip. A CRT results if thrombus extends from the catheter proper into the venous system.[6]

Clinical Presentation

The majority of CRT are found incidentally in asymptomatic patients.[4,6,8] When symptomatic, patients will likely present with signs of obstruction resulting in arm and neck edema, arm or neck pain, or catheter malfunction.[4,8] The most serious complication of CRT is a pulmonary embolism occurring in 10%-15% of patients.[9] Other serious complications including right atrial clot formation, infection, delay in medication delivery, and post-thrombotic syndrome may also occur. Post-thrombotic syndrome is an unusual constellation of symptoms including limb pain, edema, limb heaviness, and in some cases ulceration.[4,6] A high index of suspicion is therefore required to assess for CVT in the cross-sectional imaging evaluation of all patients with indwelling central venous catheters.

Imaging Findings

If CRT is suspected, contrast venography is considered the gold standard for diagnosis.[8] However, due its relative invasive nature, requirement of contrast and radiation, noninvasive tests such as ultrasound, MR or CT may be performed as the initial examination for evaluation. Doppler ultrasonography is the least invasive examination and performs well in the evaluation for catheter related deep venous thrombosis with sensitivities and specificities ranging between 84% and 97% and is performed most often.[8] Veins within the neck and arm are relatively accessible and can be easily interrogated by ultrasound. Ultrasound may show a hyperechoic filing defect within the vein and may not be compressible due to clot filling the vessel. For thrombosis within the subclavian veins behind the clavicle or within the superior vena cava, ultrasound is less effective.[4,6]

Contrast-enhanced CT may be an effective alternative at demonstrating a catheter related thrombosis, particularly at demonstrating thrombosis that extends into the thoracic cavity. An obstructing or partially obstructing intraluminal filling defect outlined by contrast upstream from the CVC is the characteristic imaging finding (Fig. 3). If fat infiltration or abscess formation is identified within the soft tissues adjacent to the thrombosed vein, secondary infection may be

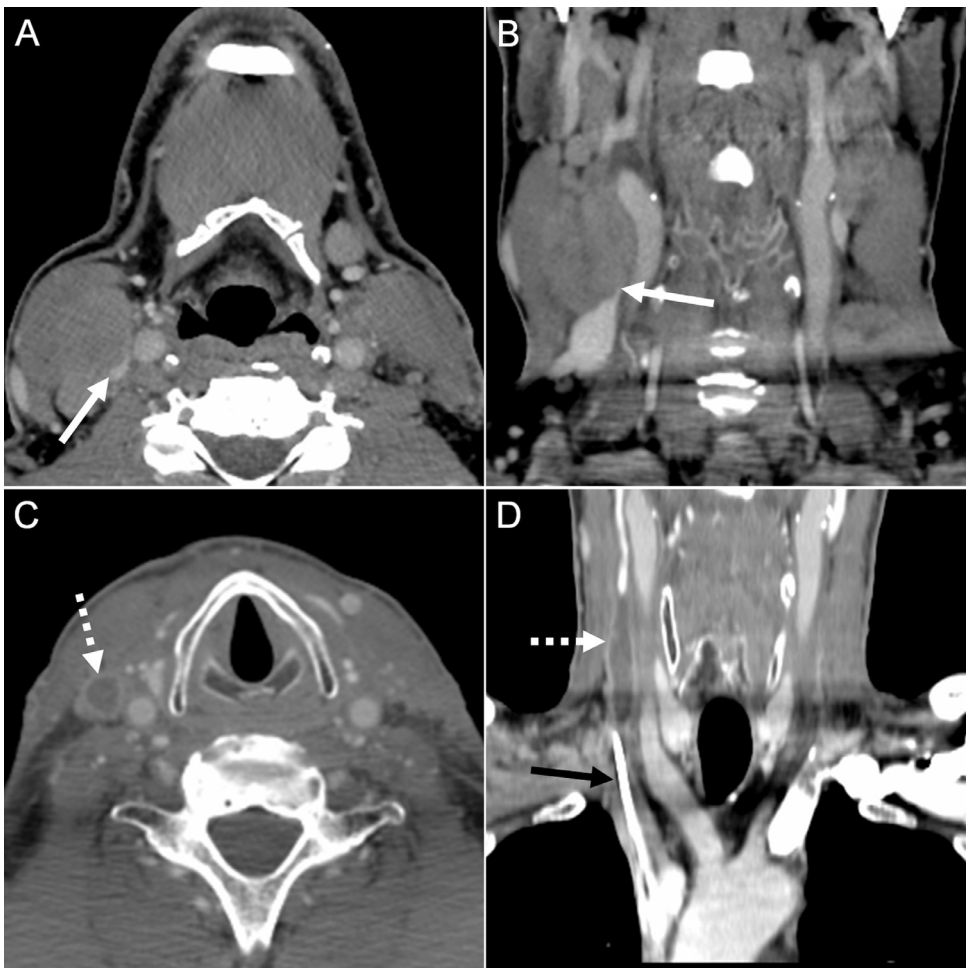

Figure 3 A 54-year-old male with a history of squamous cell cancer of the base of tongue. Initial staging CT in the axial (A) and coronal (B) planes demonstrates a large right level II/III nodal conglomerate with mass effect on the right internal jugular vein (solid white arrows). He had tumor recurrence and subsequently had a right internal jugular venous port-a-catheter placed for chemotherapy. At routine follow up, axial (C) and coronal (D) CT images demonstrate a tubular luminal filling defect (dashed white arrows) upstream from the catheter (black arrows) consistent with catheter-related thrombosis.

suspected, which is more apparent on CT than with other imaging modalities (Fig. 4).

Treatment

Treatment of CRT for CVC is somewhat controversial as reliable data is limited in patients with upper extremity deep venous thrombosis (DVT). Treatment strategies and algorithms are often extrapolated from lower extremity DVT data.[4] Anticoagulation for a minimum of 3 months is typically indicated.[6] If the central venous catheter is well positioned, remains functioning, without signs or symptoms of infection, catheter removal is often not indicated.[4,6] However, if any of these criteria are not met, catheter removal is typically performed.[4,6]

Infection

Venous Thrombophlebitis

Introduction and Epidemiology

Thrombophlebitis of the venous structures of the neck is a rare complication of head and neck infection, most commonly resulting from spread of infection from the oropharynx related to pharyngitis or tonsillitis.[10] In addition, thrombophlebitis may result as a complication from an indwelling central venous catheter.[4] Thrombophlebitis It is primarily a disease of adolescence and young adulthood (age 16–30 years),[11] although the disease has been reported in patients ranging from age 2 months to the elderly.[10] Although strict criteria have varied in the literature since Lemierre's landmark case series in 1936,[12-15] in general, when features of head and neck infection are seen in combination with local venous thrombophlebitis and signs of systemic infection (including the presence of metastatic septic emboli), the constellation of findings is referred to as Lemierre's syndrome.[11,13,16] Epidemiologic data regarding Lemierre's syndrome is sparse due to the rarity of the disease, but a study from Denmark within the last 10 years estimated the incidence of Lemierre's syndrome to be approximately 3.6 cases per million persons per year.[17]

Pathophysiology

The most common causative organism of Lemierre's syndrome reported in the literature is the Fusobacterium necrophorum species, a gram-negative nonspore-forming anaerobic bacterium which can be found as part of the normal human oral flora. Other implicated species include Bacteroides, Streptococcus, Staphylococcus, Lactobacillus, and other Fusobacterium species.[15,16] The pathomechanism by which *F. necrophorum*, a typically noninvasive species, crosses mucosal surfaces leading to septic thrombophlebitis is incompletely understood, although some authors have posed that either changes in the integrity of the mucosa related to primary viral throat infection or activation of proteolytic enzymes, possibly enhanced by nicotine use, may play a role.[11,15,18] Regardless, infectious involvement of the veins of the neck and/or face (most commonly the internal jugular vein) with subsequent thrombophlebitis may occur either by direct extension, or via lymphatic or hematogenous spread via the peritonsillar vessels.[15]

Clinical Presentation

Patients with infectious thrombophlebitis of the neck most often present with symptoms of pharyngitis, with sore throat being the most common presenting symptom. Neck mass and/or neck pain are also common complaints which, taken together with sore throat, account for up to 76% of presenting symptoms.[10] Sore throat typically precedes symptoms related to thrombophlebitis by several days. There are also clinical manifestations related to distant septic emboli,

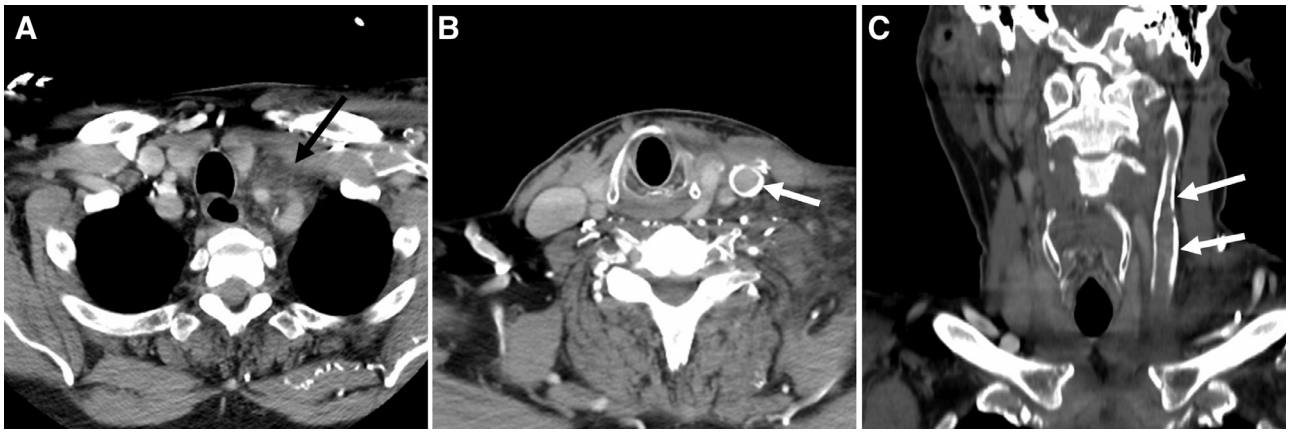

Figure 4 A 59-year-old female with a history of myasthenia gravis with a left internal jugular tunneled pheresis catheter who recently presented with fevers and chills. Infection was suspected and the catheter was removed. Axial (A,B) and coronal (C) reconstructions from a contrast-enhanced CT scan were performed to evaluate for infection which demonstrates inflammatory fat stranding within the left supraclavicular fossa (black arrow) suggestive of inflammation. There is a luminal filling defect within the left internal jugular vein outlined by contrast consistent with venous thrombosis (white arrows). These findings are consistent with catheter related septic thrombophlebitis.

including respiratory, musculoskeletal, hepatic or neurologic findings, among others.[10,11]

The clinical work up of patients with suspected Lemierre's syndrome includes laboratory and imaging evaluation. Laboratory evaluation typically reveals a moderate leukocytosis and elevated inflammatory markers. Blood cultures are positive in the large majority of cases.

Imaging

The most common initial imaging test in previous review series has been chest radiography,[10] likely due to its low cost, widespread accessibility, and frequent concomitant respiratory symptoms in patients with Lemierre's syndrome. Chest radiographs may reveal areas of consolidation, cavitary lesions, and pleural fluid collections, although a normal chest radiograph does not exclude the diagnosis.[15] The objective confirmation of internal jugular vein thrombosis by imaging is often the first concrete evidence suggesting Lemierre's syndrome.[11,13] The initial imaging study of choice is somewhat debated, and may include ultrasound of the neck, contrast-enhanced CT, or MRI. Other modalities used historically to diagnose jugular vein thrombosis, including conventional contrast venography or nuclear medicine scintigraphic studies, are rarely used in the modern era.[10,18]

Ultrasound has been advocated as a sensitive, rapid, and inexpensive initial evaluation for jugular venous thrombosis with the additional benefits of no ionizing radiation and no need for intravenous contrast.[15] The hallmark imaging findings of acute venous thrombosis on gray scale ultrasound include visualization of low-level echoes representing thrombus within the vein lumen, venous distention with incomplete or noncompressibility of the vein with dynamic compression by the sonographer, and diminished or absent venous pulsation.[19] Color and pulsed Doppler imaging can also be of great value by confirming absence of normal intraluminal color flow in the vein and absence or aberration of the conventional venous waveform on pulsed Doppler imaging, including loss of the typical pulsed Doppler changes seen with respiration and augmentation.[20] However, ultrasound is suboptimal for evaluation of venous structures deep to the mandible or below the clavicle due to poor ultrasound beam penetration, and may miss acute low attenuation clot (which can be markedly hypoechoic), particularly in areas where it is difficult to perform venous compression.[18]

Contrast-enhanced CT is likely the best overall initial imaging test in the acute setting due to rapid acquisition time, superior spatial resolution, and ability to easily localize and characterize primary and secondary sites of infection and evaluate for drainable fluid collections.[15,18] Contrast-enhanced CT is also excellent at assessing for signs of phlebitis, which may manifest as venous wall thickening and hyperenhancement as well as low-attenuation intraluminal filling defects consistent with thrombus in the internal jugular vein[18,21] (Figs. 4 and 5). Thrombophlebitis of the facial veins, anterior jugular veins, and external jugular veins, in isolation or in combination with internal jugular vein involvement, is an additional reported manifestation of Lemierre's syndrome (Figs. 6 and 7).[22] Other advantages of CT include demonstration of the extent of venous thrombosis and assessment of the carotid arteries for rare manifestations of neck infection such as mycotic pseudoaneurysm or arterial thrombosis.

Treatment

The mainstay of treatment for Lemierre's syndrome is prolonged antibiotic therapy.[11,15,16] Anticoagulation is also commonly implemented, although its added benefit/necessity remains controversial.[23,24] Abscesses that are sizable and accessible are typically drained, and very rarely, surgical ligation of the thrombosed vein may be performed to prevent further thromboembolic events, typically in patients who are extremely ill.[16,18,25] Although mortality has been reported to occur in roughly 0%-18% of cases in the postantibiotic era,[25]

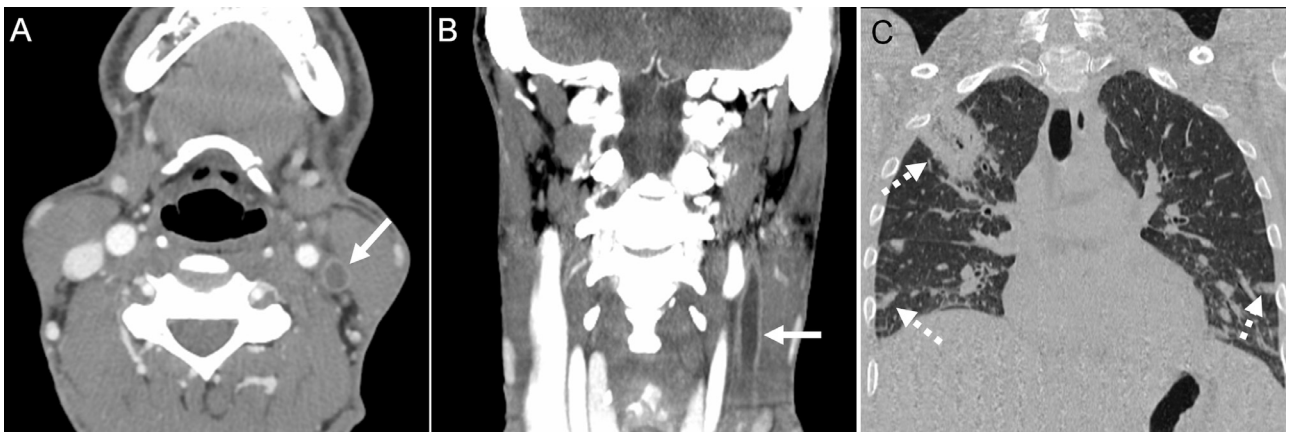

Figure 5 A 45-year-old with multifocal pneumonia status post recent course of pharyngitis. Contrast-enhanced CT images of the neck in the axial (A) and coronal (B) planes show a filling defect within the left internal jugular vein (A, B white arrow) consistent with thrombosis. A coronal CT reconstruction of the lungs (C) shows multifocal air space opacities throughout the lungs (dashed arrows) consistent with multifocal pneumonia. The constellation of findings is characteristic of Lemierre's syndrome.

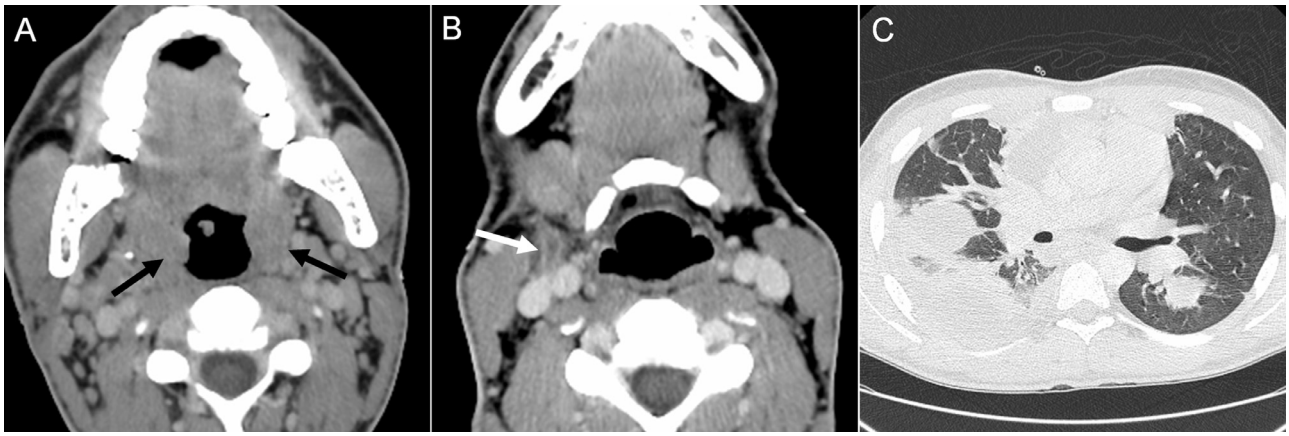

Figure 6 A 16-year-old with pharyngitis. Contrast-enhanced CT images of the neck in the axial planes (A,B) demonstrate evidence of enlarged bilateral tonsils (A, black arrows). The internal jugular veins are patent bilaterally; however, on the right, there is a filling defect within a confluent trunk that drains the retromandibular vein and tonsillar vein (B, white arrow). An axial CT scan of the lungs shows multifocal air space opacities consistent with multifocal pneumonia as well as a large right pleural effusion. The constellation of findings represents another case of Lemierre's syndrome. In this case though, a smaller tributary vein is thrombosed rather than the internal jugular vein.

recent systematic reviews estimate the mortality rate as closer to between 2% and 5%, and the overall prognosis in the modern era is good with prompt diagnosis and timely and appropriate treatment.[10,16]

Infectious Arteritis of the Neck

Introduction and Epidemiology

Infectious involvement of the extracranial carotid and vertebral arteries is extremely rare. Manifestations include mycotic pseudoaneurysm formation with the associated rare complication of pseudoaneurysm rupture, or arterial thrombosis/occlusion.[26-44] Some studies additionally suggest a possible link between recent upper respiratory tract infection and spontaneous cervical arterial dissection[45,46] although cervical artery dissection will not be addressed in this section.

Specific epidemiological data regarding infectious arteritis are sparse owing to the rarity of the disease process, but it appears at least that the incidence of infectious carotid pseudoaneurysm has been relatively stable in recent decades, with approximately 20 cases per year reported in the literature over a 30 years period leading up to 2013.[27] Infectious pseudoaneurysm formation has been documented in the cervical carotid arteries and even more rarely in the cervical vertebral arteries,[27,30-32,34,35,37,39] and has been seen in patients of all ages, from infants to the elderly.[27] Cervical arterial occlusion or rupture secondary to infection is an even rarer event.[33,35,36,42-44]

Pathophysiology

In the modern era, implicated pathogens of extracranial infectious arteritis of the neck are most commonly bacteria

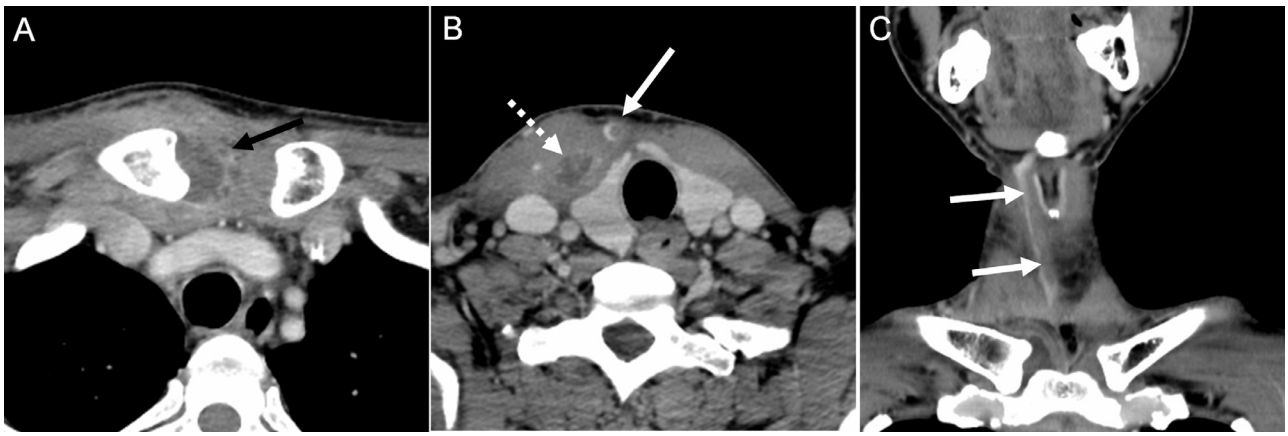

Figure 7 A 56-year-old who presents with right neck swelling. A contrast-enhanced CT examination was performed with reconstructions in the axial (A,B) and coronal (C) planes which demonstrates a rim enhancing fluid collection consistent with an abscess associated with the right sternoclavicular joint consistent with septic arthritis (A, arrow). There is abscess tracking superiorly along the right sternocleidomastoid muscle (B, dashed arrow). In addition there is a tubular filling defect within the right anterior jugular vein (B,C white arrows). This represents another case of septic thrombophlebitis but of the anterior rather than internal jugular venous system.

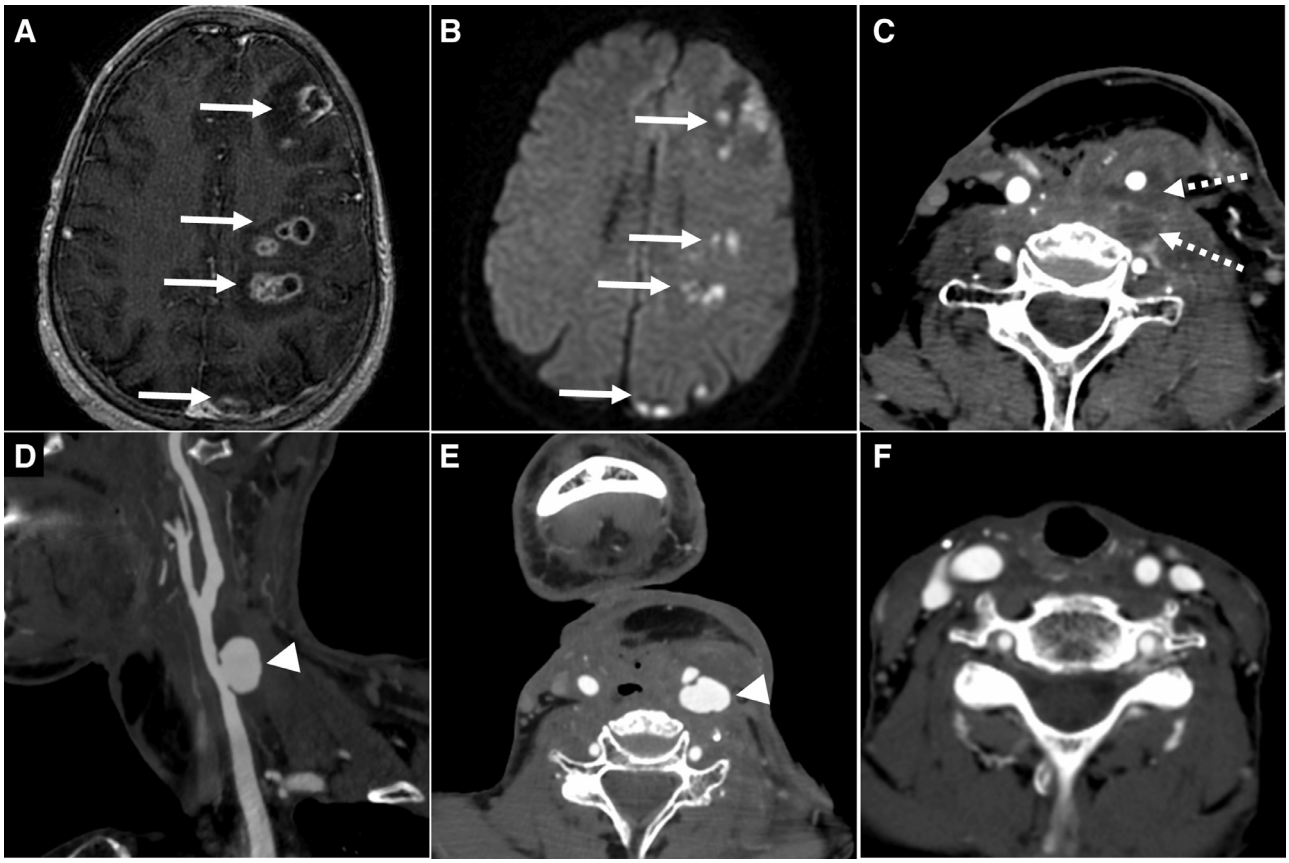

Figure 8 A 58-year-old with a history of advanced laryngeal cancer treated with chemoradiation and subsequent salvage laryngectomy, now presents with right body weakness. A brain MRI was performed including axial postcontrast (A) and diffusion weighted (B) sequences demonstrating multiple ring-enhancing lesions within the left hemisphere (A, arrows) with corresponding central diffusion restriction (B, arrows) concerning for septic emboli. A proximal vascular abnormality was suspected and a CTA of the head and neck was performed (C-E) which demonstrates a rim enhancing collection surrounding the left common carotid artery consistent with infection (C, dashed arrows). A focal vascular outpouching arising from the left common carotid artery (D-E, arrowheads) was noted centered within this region of infection consistent with an infected carotid pseudoaneurysm representing the proximal source for the intracranial thromboembolic disease. These findings were all new compared to the patient's baseline exam performed several years prior (F).

(typically *Staphylococcus, Streptococcus,* or *Salmonella* species), with rarer instances of fungal or mycobacterial infectious causes.[27] The source of pathogen inoculation may be related to contiguous spread from a primary site of head and neck infection, related to septic embolic phenomenon (eg, endocarditis or sepsis), or related to vascular manipulation, either from surgical/endovascular procedures, or trauma.

Infectious arteritis has been proposed to occur when there is inoculation of a pathogen into the arterial wall, leading to acute vessel wall inflammation and subsequent weakening. Inoculation may occur through compromise of the intimal layer in the case of an intravascular source of infection (eg, septic emboli), or via compromise of the adventitia in the case of contiguous spread of infection from an adjacent primary site. Compromise of the intima has been proposed to occur via mechanical disruption related to a septic embolic event itself, via breech in the intima related to trauma or iatrogenic causes, or due to pre-existing primary vascular disease such as arteriosclerosis/atherosclerosis.[28] Others have suggested that inoculation could occur via the vasa vasorum in large arteries, such as the aorta.[28] Regardless, pathologic changes have been documented within the arterial wall on histologic examination, including degeneration of the muscular media and elastic lamina, predisposing to pseudoaneurysm formation and rupture.[28] Damage to the intima associated with local inflammation may also predispose to thrombosis/occlusion.

Clinical Presentation

Clinical findings of infectious arteritis depend on the manifestation of arterial involvement. Most patients with infectious pseudoaneurysm present with progressive neck swelling/growing pulsatile neck mass with or without associated fever and neck pain.[26,27,39] Lower cranial nerve palsy or Horner's syndrome may also be a presenting feature.[27,40,41] Rupture of a mycotic pseudoaneurysm may manifest as recurrent or unexplained epistaxis or oropharyngeal bleeding, slow or incomplete resolution of an appropriately treated neck infection, anemia greater than would be expected clinically, or oropharyngeal mucosal ecchymosis/discoloration.[42,43]

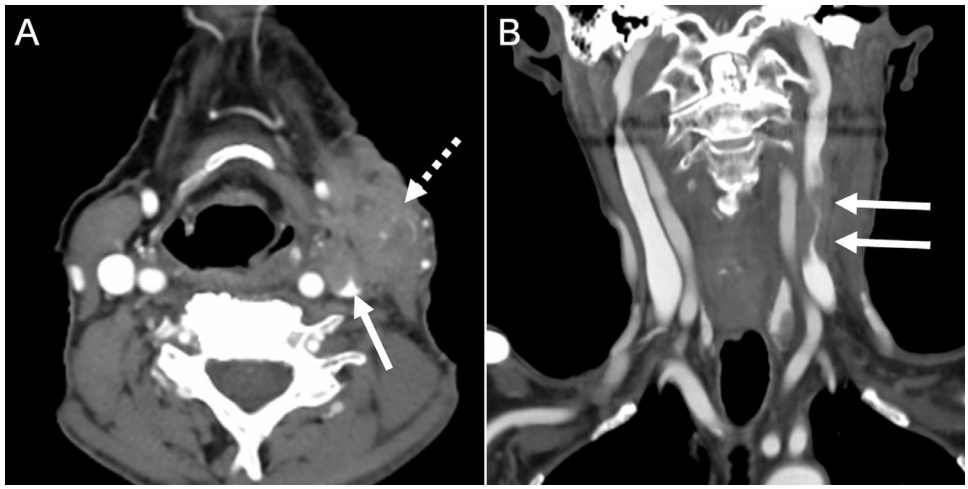

Figure 9 A 79-year-old with follicular lymphoma presents with shortness of breath. A postcontrast CT study of the neck (A,B) shows a large nodal mass within the left level II and III nodal stations (A, dashed arrow) with resultant tumor thrombus that extends into the left internal jugular vein (A-B, solid arrows).

Of the few rare cases of infection-associated cervical arterial thrombosis reported in the literature, some showed no signs of associated cerebral infarction (most likely related to chronicity of the occlusion with collateral arterial flow),[35,36] while one case presented with new onset dense hemiparesis related to a massive middle cerebral artery territory infarct.[44] Cases of infectious intracranial carotid stenosis,[47] as well as infectious arterial occlusion leading to cerebral infarcts, have also been reported.[48]

Imaging

The diagnosis of infectious arteritis is most commonly made with ultrasound or contrast-enhanced CT. On ultrasound a pseudoaneurysm appears as a saccular or fusiform outpouching or fusiform dilatation of the artery, often with eccentric low-level internal echoes representing clot along the wall of the aneurysm. At times the aneurysm may be mistaken for a fluid collection, but careful inspection will demonstrate that the apparent fluid collection emanates from the artery and that there is turbulent flow noted within the aneurysm sac. Classically, a pseudoaneurysm demonstrates the classic Yin-Yang Color Doppler sonographic pattern within the aneurysm sac and a so-called "to-and-fro" flow-pulsed Doppler waveform pattern across the neck of the aneurysm, owing to antegrade and retrograde flow across the aneurysm neck.

On CT, a pseudoaneurysm manifests as an outpouching of the parent artery which may be saccular, fusiform or irregular in configuration and typically demonstrates filling of the aneurysm sac with hyperdense contrast material (Fig. 8). There is often some irregular eccentric hypoattenuating clot along the wall of the aneurysm sac. The additional advantage of CT is superior characterization of the perivascular soft tissues as compared with ultrasound.[27] In the case of an infectious pseudoaneurysm, there will typically be fat-stranding

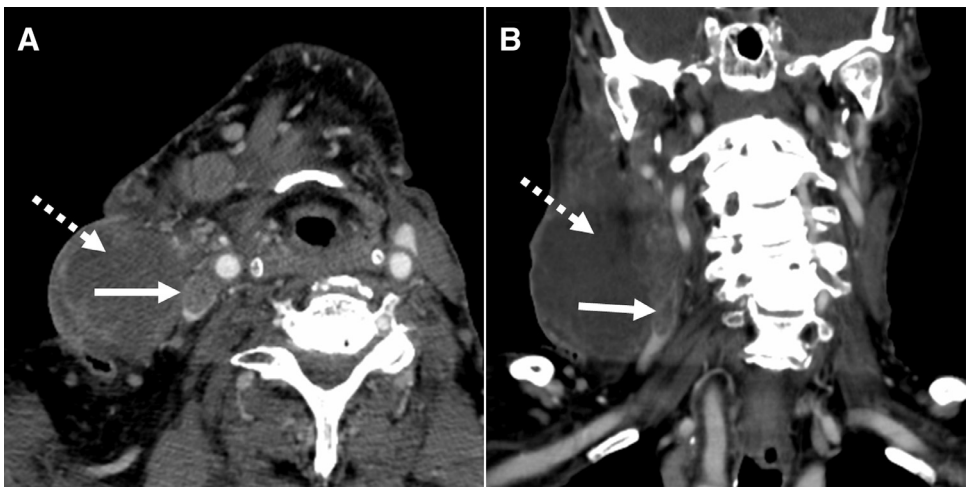

Figure 10 A 94-year-old male with known soft tissue sarcoma presents with neck swelling and fever. A contrast-enhanced CT examination in the axial (A) and coronal planes (B) demonstrates a large soft tissue mass that is inseparable from the right sternocleidomastoid muscle (A-B, dashed arrows). There is directed extension of tumor from the neck into the adjacent right internal jugular vein (A-B, solid arrows) consistent with tumor thrombus.

in the soft tissues surrounding the aneurysm, and additional findings of infection may be present, such as infected fluid collection/abscess, soft tissue swelling, and subcutaneous edema, and/or soft tissue gas in the case of necrotizing infection. In addition, evidence of distal thromboemboli within the brain may be evident on CT or MRI (Fig. 8).

Treatment

The cornerstone of treatment of infectious arteritis is long-term antibiotics. Surgical/procedural interventions are employed for the treatment of infectious pseudoaneurysms. Surgical treatment most commonly consists of excision of the pseudoaneurysm with restoration of arterial continuity where possible, and debridement of surrounding infected tissues.[27] Revascularization after aneurysm excision may be established using primary repair, patch-plasty of the parent artery, or arterial bypass. Bypass may be performed using autologous arteries, veins, cryopreserved arterial allografts, or synthetic prostheses. Proximal surgical ligation of the parent artery without revascularization is reserved for cases where revascularization is not technically possible. Endovascular treatment of infected pseudoaneurysms of the extracranial head and neck with covered stenting in combination with long-term antibiotics has also been described but is less well established.[27,49,50]

Vascular Emergencies in Head and Neck Cancer Patients

Introduction and Epidemiology

Vascular emergencies affecting the venous and arterial system may occur in HNC patients related to the underlying neoplastic disease process or as a consequence of treatment. The internal jugular vein is particularly susceptible to narrowing from external mass effect primarily due its relatively thin wall, but also due its proximity with level II, III, and IV nodal disease (Fig. 3). Rarely, tumor thrombus may propagate in a retrograde fashion from the primary cancer or nodal disease (Figs. 9 and 10) and has been described in patients with thyroid and squamous cell cancer.[51,52] Patients are often able to compensate for narrowing or occlusion of the internal jugular venous system due to the rich vascular collateralization within the neck.

It is more likely that true vascular emergencies of the neck occur in the post-treatment setting which will be the focus of this section. HNC patients often receive chemotherapy from a tunneled central venous catheter. As previously discussed, the venous system should be interrogated in all patients receiving chemotherapy for cancer treatment as line related thrombosis is a known common complication.[4]

Injury to the major arterial system after radiation therapy is a well-described phenomenon in HNC patients. Radiation injury to the vessel wall can lead to stenosis of the vessel or weakening of the wall integrity resulting in pseudoaneurysm formation and carotid blowout syndrome (CBS). The major medical emergencies that result from postradiation injury can thus be categorized into two categories: neurologic ischemia and acute hemorrhage. Neurologic ischemia results from occlusion from carotid stenosis, plaque rupture from accelerated atherosclerosis, or embolism from a thrombosed pseudoaneurysm (Fig. 7). Acute hemorrhage occurs due to a vessel rupture at a site where radiation injury has resulted in a pseudoaneurysm or other vessel wall disruption.

Radiation therapy is a significant risk factor for TIA and CVA, particularly in younger patients.[53] One study found that the risk of stroke after any radiation therapy to be about 6%.[54] The risk is significantly higher in patients younger than 60, around 12%, with a RR of 10 compared to the age and sex matched controls in the general population.[53] This increased risk of stroke is due to radiation-induced accelerated atherosclerosis and radiation-induced carotid stenosis.[55] A recent review of nine studies estimates the incidence of radiation-induced "significant" carotid stenosis (ie, greater than 50% stenosis) to range from 18%-38% of patients who undergo radiation therapy, compared to 2%-8% in the

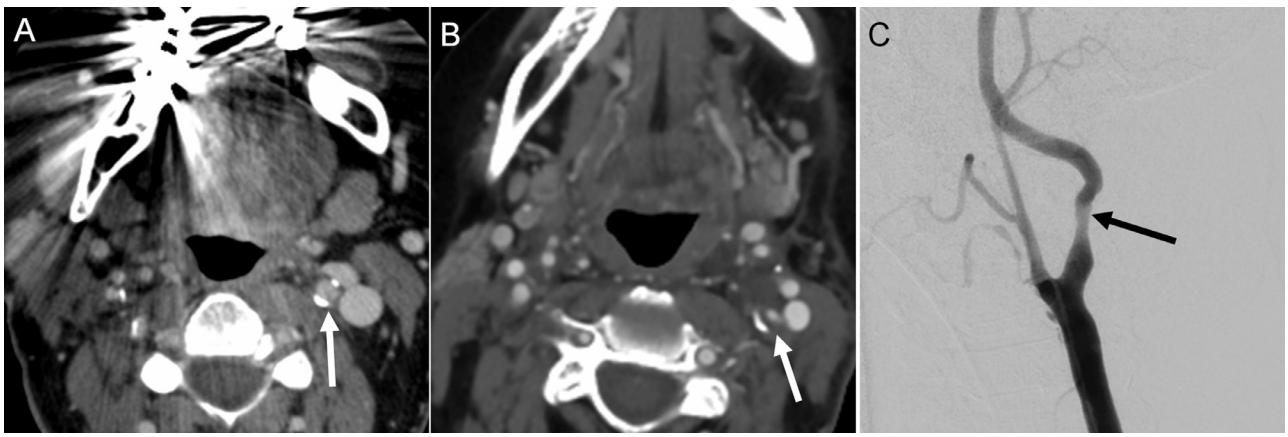

Figure 11 A 56-year-old initially diagnosed with a T2N0 squamous cell cancer of the oral cavity treated with surgery and chemoradiation presents for follow up. An axial CT scan of the neck at diagnosis (A) shows mild atherosclerosis of the left internal carotid artery (A, arrow). At a subsequent follow up 2 years following treatment (B), there is focal narrowing of the left internal carotid artery related to accelerated atherosclerosis (B, arrow). This finding was confirmed at digital subtraction angiography (C, arrow).

general population.[55] Compared to the general population, there is a greater incidence of involvement of the external carotid artery (45% vs 2%), and greater incidence of bilateral involvement in the RT group (11%-69% vs 3%-33%).[55]

Carotid pseudoaneurysms are uncommon and can result from a myriad of causes including surgery, infection, chemotherapy, trauma, and radiation. The true incidence of pseudoaneurysm secondary to radiation therapy is unknown but is usually more common in patients that have required multiple rounds of radiation therapy.[56] Though the focus here is on extracranial carotid pseudoaneurysms, postradiation pseudoaneurysms have been found to arise both in the extracranial and intracranial portions of the carotid artery.[57-59] Although uncommon, postradiation pseudoaneurysms are an important clinically sequela of radiation therapy, as complications can have significant morbidity and mortality. These complications range from thromboembolic stroke from a thrombosed pseudoaneurysm to CBS.

CBS is a rare but clinically significant complication of treatment for head and neck cancers, particularly after surgical resection and reirraditaion.[60] It can be seen in patients with pseudoaneurysms, though it can present without them.[61] The incidence of CBS has been reported to range from 2.9% to 4.3% in patients with a history of surgical resection and 2.6%-10% in patients with a history of reirradition.[60]

Pathophysiology

Although distinct disease entities, it is generally understood that the etiology of radiation-induced vasculopathies are likely secondary to the same mechanisms of radiation-induced vessel injury. The exact mechanism by which radiation therapy damages the vessels of the head and neck has not yet been elucidated, though it is generally thought to be multifactorial; a combination of accelerated atherosclerosis, direct injury to the vessel wall from the radiotherapy, and

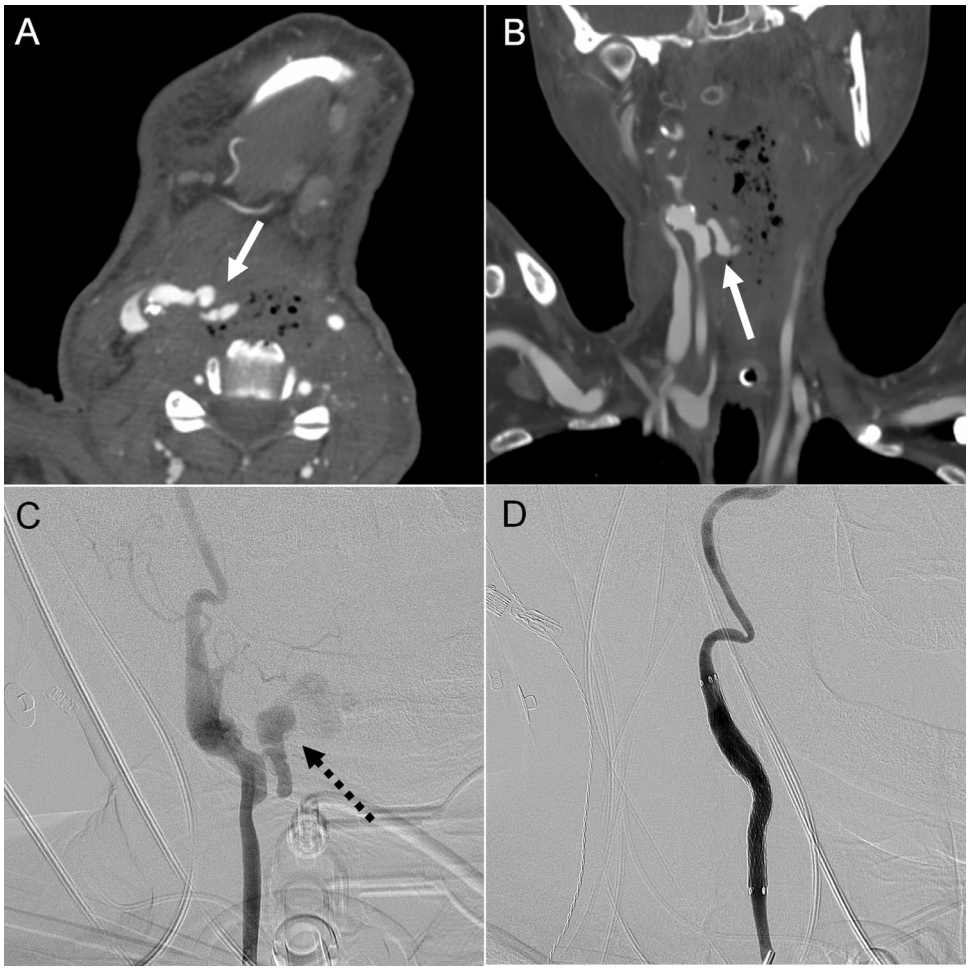

Figure 12 A 57-year-old with a history of a remote laryngeal cancer, total laryngectomy, and radiation who presents with brisk hemoptysis. A CT angiogram in the axial (A) and coronal planes (B) show a multilobulated focal outpouching consistent with a carotid pseudoaneurysm arising from the medial edge of the right common carotid artery and communicates with the airway (A-B white arrows) consistent with a carotid blow out. The patient was taken for emergent catheter based angiography (C) which confirms the multiobulated pseudoaneurysm (C, dashed arrow) suspected on CTA. The patient was successfully treated with an endovascular stent placed over the pseudoaneurysm (D).

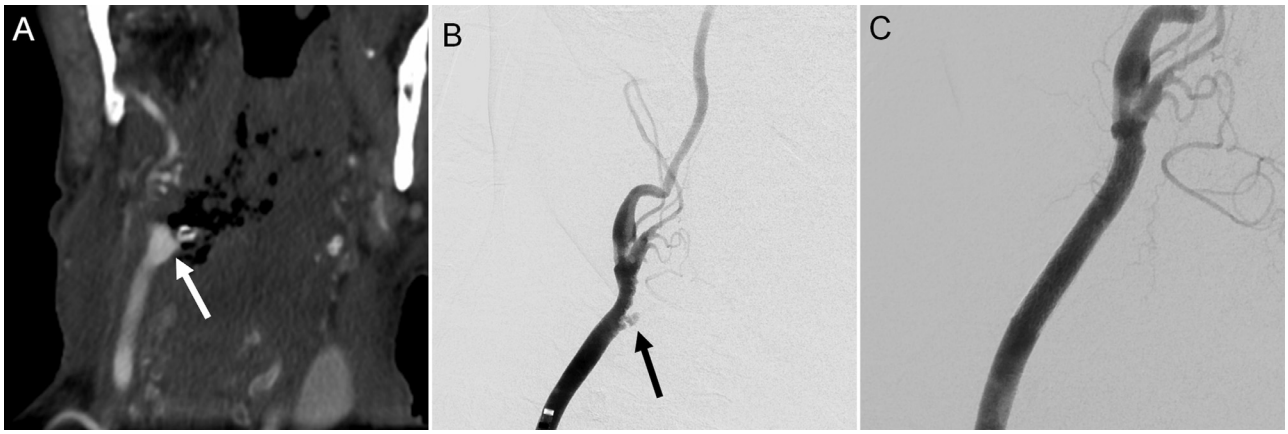

Figure 13 A 75-year-old with a history of laryngeal cancer presents with bleeding from tracheostomy following completion of chemotherapy and radiation five months prior to current presentation. Coronal CTA (A) shows a contour abnormality with a subtle focal bulge along the medial aspect of the right common carotid artery (A, white arrow). Catheter based angiography (B) confirms the focal outpouching arising from the medial aspect right common carotid artery consistent with a pseudoaneurysm (B, black arrow), which was successfully treated with an endovascular stent (C).

indirect injury to the vessel due to injury to the vaso vasorum.[62,63] Radiation is theorized to cause direct injury to the vessel wall in several ways. There is direct radiation injury to the endothelial cells characterized by nuclear damage, a proliferation of reactive oxygen species created by ionizing radiation which in turn causes DNA, lipid, and protein damage, impaired nitric oxide-dependent relaxation, and inflammatory-mediated damage and fibrosis to the site of injury.[63-65] This disruption of the endothelial barrier allows for the lipid deposition and the subsequent lysosomal activation, and endothelial cell proliferation that characterizes the formation of atheromatous plaques.[63] Injury to the vaso vasorum, likely by the aforementioned mechanisms, results in occlusive disease and downstream ischemic necrosis of the vessel walls they supply. Over time the necrotic tissue is remodeled and replaced with fibrosis.[65]

There is evidence that there is also accelerated atherosclerosis in patients who have undergone radiation therapy. One study investigated carotid intima media thickness (CIMT), a known surrogate marker for presence of atherosclerosis, in patients who had undergone radiotherapy for nasopharyngeal carcinoma and found that CIMT was significantly increased in those patients compared to controls (Fig. 11).[64] CIMT is also linearly correlated with stroke risk[64] and an absolute difference of 0.1 mm increases stroke risk by 13%-18%.[63] In some cases, this fibrosis, endothelial dysfunction, and accelerated atherosclerosis result in extended plaques and stenosis of the vessels.

In the case of pseudoaneurysm formation, a vessel wall weakened by ischemia and fibrosis is then subjected sheer stress and to the water hammer effects from the patient's blood pressure.[58] The water hammer effects are also likely enhanced in these patients given the likelihood of stenosis elsewhere in the vessel. A vessel wall weakened by radiation injury, ischemia, and fibrosis can also lead to carotid blow out syndrome, even in the absence of pseudoaneurysm formation.[60] The weakened vessel wall is not able to withstand a patient's blood pressure and this can lead to rupture.[60] The rates of carotid blow out are highest in patients with a history of both surgery and radiation compared to a history of either alone.[60] Radical neck dissections that expose the carotid artery are thought to contribute further to the radiation induced injury by damaging the adventitia which supplies 80% of the carotid artery's blood supply.[66] These radical neck dissections are commonplace in HNC treatment and may partially explain a higher incidence of carotid blowout in HNC patients even in the absence of radiation.[66] Poor postoperative wound healing in these patients, especially those with a history of radiation, and the risk of infection, which may be subclinical, also contribute to CBS.[66]

Clinical Presentation

Generally, patients with critical carotid stenosis or acute plaque rupture leading to thromboembolic occlusion of the vessel will present with CVAs or TIAs consistent with the affected vascular bed. The clinical presentation of carotid pseudoaneurysms vary. Many carotid pseudoaneurysms are asymptomatic. In one study examining extracranial carotid pseudoaneurysms of all causes, nearly half (49%) were asymptomatic. Of the remaining symptomatic patients, a plurality (38%) presented with a painless mass.[67] For the remaining symptomatic patients, the clinical presentation varied with the most common presentation being either TIAs or CVAs secondary to embolization of a thrombus formed in the pseudoaneurysm lumen leading to occlusion downstream.[67] Pseudoaneurysms can also present with CBS.[61] CBS is a syndrome which refers to carotid rupture or imminent carotid rupture. It is categorized into three types. Type I is *threatened* CBS and presents as asymptomatic carotid artery wall exposure detected on imaging.[60,68] Type II is known as *impending* CBS and presents with a herald bleed which may or may not immediately precede acute rupture.[66,68] Type III is acute rupture and can present with acute airway compression, pharyngeal hemorrhage, or massive epistaxis.[56,60,66,68]

Imaging

Doppler ultrasonography is the main imaging modality used to detect carotid stenosis, particularly for screening purposes. It is noninvasive and also allows the level of stenosis to be quantified.[63] B-mode ultrasonography can also measure CIMT specifically.[63] CIMT is linearly correlated with stroke risk and is thus predictive of future cerebrovascular events.[64] Additionally an increase in CIMT is one of the earliest postradiation changes that we are able to detect with imaging.[63] Digital subtraction angiography is considered the gold standard for the diagnosis of carotid stenosis.[63] However, the expense and its invasive nature preclude its widespread use for diagnostic purposes, noninvasive imaging such as CT angiography (CTA) and MR angiography (MRA) are usually favored to further characterize lesions and for treatment planning.[63] CTA and MRA typically demonstrate progressive luminal narrowing of the carotid artery by noncalcified plaque (Fig. 11).

Much like carotid stenosis, ultrasonography is the mainstay for detecting extracranial carotid pseudoaneurysms. However, most patients will go on to also have CTA or MRA which can illustrate focal vascular outpouching indicative of a pseudoaneurysm, its location relative to other structures, and whether there is associated thrombosis (Figs. 8, 12, and 13). CT is superior to ultrasound in that it offers the additional benefit of outlining the regional post treatment anatomy, which is often very complicated in this patient population. In addition, the relationship of the pseudoaneurysm and associated hemorrhage within the surrounding soft tissues including the airway is also better assessed with CT than with other imaging modalities.

Treatment

Carotid endarterectomy (CEA) is the gold standard for patients with nonradiation-induced carotid stenosis.[63] However, in the setting of patients with a history of head and neck cancers, many of whom have undergone both surgical resection and radiation, CEA has significant limitations. CEA is more technically difficult in radiation patients. The stenosis in patients with prior radiation is usually more diffuse and extensive than those without prior radiation.[69] Additionally, adhesions from radiation therapy pose further challenges for CEA.[63] Many HNC patients also have a history of surgical resection. A history of surgical resections independently increases the risk for wound complications and cranial nerve injury with CEA.[63] Due to the high risks associated with CEA in this patient population, endovascular procedures, namely carotid artery angioplasty and stenting is the preferred treatment (Figs. 12 and 13) CAS is not without its disadvantages however. Although the early stroke rate with CAS is the same as that with CEA, with fewer of the risks of CEA, the stroke rate later on is higher.[70] Therefore, for the patient who is a good candidate for surgery, it is better to recommend CEA.[70]

Patients with asymptomatic, extracranial carotid pseudoaneurysms, patients are usually observed with serial imaging. A retrospective review of 141 patients with carotid aneurysms of all causes, found that for unruptured aneurysms, the decision to intervene was usually dependent on size, if the aneurysm was expanding, or if infection was present.[67] It is not well described in the literature if conservative management is acceptable in patients with a history of radiation given that radiation induced pseudoaneurysms can present with CBS. All symptomatic extracranial carotid pseudoaneurysms are repaired. In patients with a history of radiation, an endovascular approach is preferred to open approaches.[57]

Intervention is not always necessary for patients with Type I CBS and a normal angiogram, and may introduce greater risk of bleeding and stroke to the patient. If the patient proceeds with an intervention, then the therapy is similar to that of Type II and III.[68] Endovascular therapy with carotid artery embolization has replaced surgical ligation as the treatment of choice for CBS as it associated with dramatically lower morbidity and mortality rates.[68] Although lower than surgical ligation, embolization still carries a 15%-20% risk of neurologic impairment from cerebral ischemia. Therefore, prior to embolization, a balloon occlusion test is done to assess for collateral circulation. If a patient fails balloon occlusion, then the patient is treated with stent placement. There is a high rate of recurrence of CBS in these patients and there is concern about the prudence of placing stents in an irradiated field that has tissue breakdown and microbial colonization.[68] In these patients, stenting may be viewed as a temporizing measure, with future bypass grafting and embolization later.[68] If a patient is unstable and interventional radiology is not available, then the treatment is surgical ligation.[68]

Conclusion

Nontraumatic vascular emergencies in the neck are relatively rare, but critically important to recognize. Patients with central venous catheters, head and neck infection and those treated for HNC are particularly susceptible to the development of venous and arterial injury. These patients should be evaluated with a high index of suspicion for vascular compromise as failure to recognize such injury may lead to delay in diagnosis and serious catastrophic complications.

Conflict of Interest

The authors have no conflicts of interest, financial or otherwise, related to this work.

References

1. Morris P: Practical Neuroangiography. Philadelphia: Lippincott Williams & Wilkins, 2007
2. Paulsen F, Tillmann B, Christofides C, et al: Curving and looping of the internal carotid artery in relation to the pharynx: Frequency, embryology and clinical implications. J Anat 197:373-381, 2000
3. Drake RV, A Wayne Mitchell, Adam WM: Gray's Anatomy for Students. 2015
4. Wall C, Moore J, Thachil J: Catheter-related thrombosis: A practical approach. J Intensive Care Soc 17:160-167, 2016

5. Feliu J, Lecumberri R, Jerez A, et al: Incidence of deep venous thrombosis associated with central venous catheters (DVT-CVC) in cancer patients: A multicenter study. J Clin Oncol 24:8627, 2006. -8627
6. Kamphuisen PW, Lee AY: Catheter-related thrombosis: lifeline or a pain in the neck. Hematology Am Soc Hematol Educ Program 2012:638-644, 2012
7. Kumar DR, Hanlin E, Glurich I, et al: Virchow's contribution to the understanding of thrombosis and cellular biology. Clin Med Res 8:168-172, 2010
8. Di Nisio M, Van Sluis GL, Bossuyt PM, et al: Accuracy of diagnostic tests for clinically suspected upper extremity deep vein thrombosis: a systematic review. J Thromb Haemost 8:684-692, 2010
9. Verso M, Agnelli G: Venous thromboembolism associated with long-term use of central venous catheters in cancer patients. J Clin Oncol 21:3665-3675, 2003
10. Karkos PD, Asrani S, Karkos CD, et al: Lemierre's syndrome: A systematic review. Laryngoscope 119:1552-1559, 2009
11. Eilbert W, Singla N: Lemierre's syndrome. Int J Emerg Med 6:40, 2013
12. Lemierre A: On certain septicĥmias due to anaerobic organisms. Lancet 227:701-703, 1936
13. Riordan T: Human infection with Fusobacterium necrophorum (Necrobacillosis), with a focus on Lemierre's syndrome. Clin Microbiol Rev 20:622-659, 2007
14. Osowicki J, Kapur S, Phuong LK, et al: The long shadow of Lemierre's syndrome. J Infect 74(Suppl 1):S47-S53, 2017
15. Venglarcik J: Lemierre's syndrome. Pediatr Infect Dis J 22:921-923, 2003
16. Johannesen KM, Bodtger U: Lemierre's syndrome: Current perspectives on diagnosis and management. Infect Drug Resist 9:221-227, 2016
17. Hagelskjaer Kristensen L, Prag J: Lemierre's syndrome and other disseminated Fusobacterium necrophorum infections in Denmark: A prospective epidemiological and clinical survey. Eur J Clin Microbiol Infect Dis 27:779-789, 2008
18. Golpe R, Marin B, Alonso M: Lemierre's syndrome (Necrobacillosis). Postgrad Med J 75:141-144, 1999
19. Wing V, Scheible W: Sonography of jugular vein thrombosis. AJR Am J Roentgenol 140:333-336, 1983
20. Longley DG, Finlay DE, Letourneau JG: Sonography of the upper extremity and jugular veins. AJR Am J Roentgenol 160:957-962, 1993
21. Capps EF, Kinsella JJ, Gupta M, et al: Emergency imaging assessment of acute, nontraumatic conditions of the head and neck. Radiographics 30:1335-1352, 2010
22. Karnov KK, Lilja-Fischer J, Randrup TS: Isolated facial vein thrombophlebitis: A variant of Lemierre syndrome. Open Forum Infect Dis 1, 2014. ofu053
23. Cupit-Link MC, Nageswara Rao A, Warad DM, et al: Lemierre syndrome: A retrospective study of the role of anticoagulation and thrombosis outcomes. Acta Haematol 137:59-65, 2017
24. Phan T, So TY: Use of anticoagulation therapy for jugular vein thrombus in pediatric patients with Lemierre's syndrome. Int J Clin Pharm 34:818-821, 2012
25. Murray M, Stevens T, Herford A, et al: Lemierre syndrome: Two cases requiring surgical intervention. J Oral Maxillofac Surg 71:310-315, 2013
26. Laupland KB: Vascular and parameningeal infections of the head and neck. Infect Dis Clin North Am 21:577-590, 2007. viii
27. Pirvu A, Bouchet C, Garibotti FM, et al: Mycotic aneurysm of the internal carotid artery. Ann Vasc Surg 27:826-830, 2013
28. Wilson SE, Van Wagenen P, Passaro E Jr.: Arterial infection. Curr Probl Surg 15:1-89, 1978
29. Lisan Q, Tran H, Verillaud B, et al: Infectious arteritis of the internal carotid artery complicating retropharyngeal abscess. Eur Ann Otorhinolaryngol Head Neck Dis 133:55-57, 2016
30. Gupta T, Parikh K, Puri S, et al: The forgotten disease: Bilateral Lemierre's disease with mycotic aneurysm of the vertebral artery. Am J Case Rep 15:230-234, 2014
31. Machens A, Dralle H: Mycotic aneurysm of common carotid artery induced by *Staphylococcus aureus* infection after cervical reoperation. World J Surg 25:1113-1116, 2001
32. Dawson KJ, Stansby G, Novell JR, et al: Mycotic aneurysm of the cervical carotid artery due to *Salmonella enteritidis*. Eur J Vasc Surg 6:327-329, 1992
33. Jamaan T, Raedecke J, Kayser C, et al: Septic bleeding of the common carotid artery following total thyroidectomy: an atypical complication. Case Rep Med 2010:953282, 2010
34. Bolender NF, Bassett MR, Loeser JD, et al: Mycotic aneurysm of the internal carotid artery. A surgical emergency. Ann Otol Rhinol Laryngol 93:273-276, 1984
35. Chamseddin KH, Kirkwood ML: Lemierre's syndrome associated mycotic aneurysm of the external carotid artery with primary internal carotid artery occlusion in a previously healthy 18-year-old female. Ann Vasc Surg 36:291, 2016. e211-291 e214
36. Maalikjy Akkawi N, Borroni B, Magoni M, et al: Lemierre's syndrome complicated by carotid thrombosis. Neurol Sci 22:403-404, 2001
37. Benedetto F, Barilla D, Pipito N, et al: Mycotic pseudoaneurysm of internal carotid artery secondary to Lemierre's syndrome, how to do it. Ann Vasc Surg 44:423, 2017. e413-423.e417
38. Panduranga Kamath M, Shetty AB, Hegde MC, et al: Presentation and management of deep neck space abscess. Indian J Otolaryngol Head Neck Surg 55:270-275, 2003
39. Civgin E, Toprak U, Parlak S, et al: Fissuration of vertebral artery mycotic aneurysm due to Lemierre syndrome. Diagn Interv Imaging 99:43-45, 2018
40. Amano M, Ishikawa E, Kujiraoka Y, et al: Vernet's syndrome caused by large mycotic aneurysm of the extracranial internal carotid artery after acute otitis media—Case report. Neurol Med Chir (Tokyo) 50:45-48, 2010
41. O'Connell JB, Darcy S, Reil T: Extracranial internal carotid artery mycotic aneurysm: Case report and review. Vasc Endovascular Surg 43:410-415, 2009
42. Shah RS, Kulkarni BK, Oak SN, et al: Ruptured pseudo-aneurysm of the cervical internal carotid artery in a child (a case report). J Postgrad Med 37:225-228, 1991. 228A
43. Misra BK, Shaw JF: Haematemesis from distal extracranial carotid aneurysms. Case report and literature review. Childs Nerv Syst 2:329-332, 1986
44. Goyal MK, Kumar G, Burger R: Necrobacillosis resulting in isolated carotid thrombosis and massive stroke: A unique Lemierre variant. J Neurol Sci 287:108-110, 2009
45. Guillon B, Berthet K, Benslamia L, et al: Infection and the risk of spontaneous cervical artery dissection: A case-control study. Stroke 34:e79-e81, 2003
46. Grau AJ, Brandt T, Buggle F, et al: Association of cervical artery dissection with recent infection. Arch Neurol 56:851-856, 1999
47. Jones TH, Bergvall V, Bradshaw JP: Carotid artery stenoses and thrombosis secondary to cavernous sinus thromboses in Fusobacterium necrophorum meningitis. Postgrad Med J 66:747-750, 1990
48. Bentham JR, Pollard AJ, Milford CA, et al: Cerebral infarct and meningitis secondary to Lemierre's syndrome. Pediatr Neurol 30:281-283, 2004
49. Bulsara KR, Aruny JE, Muhs B: Infectious pseudoaneurysm of the internal carotid artery treated with a covered stent. J Neurointerv Surg 1:51-52, 2009
50. Baril DT, Ellozy SH, Carroccio A, et al: Endovascular repair of an infected carotid artery pseudoaneurysm. J Vasc Surg 40:1024-1027, 2004
51. Kawano F, Tomita M, Tanaka H, et al: Thyroid carcinoma with extensive tumor thrombus in the superior vena cava: A case report. Int J Surg Case Rep 29:25-29, 2016
52. Wakasaki T, Kiyohara H, Omori H, et al: Massive internal jugular vein tumor thrombus derived from squamous cell carcinoma of the head and neck: two case reports. Oral Maxillofac Surg 21:69-74, 2017
53. Dorresteijn LD, Kappelle AC, Boogerd W, et al: Increased risk of ischemic stroke after radiotherapy on the neck in patients younger than 60 years. J Clin Oncol 20:282-288, 2002
54. Arthurs E, Hanna TP, Zaza K, et al: Stroke After radiation therapy for head and neck cancer: What is the risk. Int J Radiat Oncol Biol Phys 96:589-596, 2016
55. Fernandez-Alvarez V, Lopez F, Suarez C, et al: Radiation-induced carotid artery lesions. Strahlenther Onkol 194:699-710, 2018
56. Lam JW, Chan JY, Lui WM, et al: Management of pseudoaneurysms of the internal carotid artery in postirradiated nasopharyngeal carcinoma patients. Laryngoscope 124:2292-2296, 2014

57. Koenigsberg RA, Grandinetti LM, Freeman LP, et al: Endovascular repair of radiation-induced bilateral common carotid artery stenosis and pseudoaneurysms: A case report. Surg Neurol 55:347-352, 2001
58. Chan SHV, Woo YMP, Wong KSA, et al: The angiographic and clinical outcomes of intracranial aneurysms following irradiation in patients with nasopharyngeal carcinoma: A 13-year experience and literature review. J Neuroradiol 45:224-229, 2018
59. Girishkumar HT, Sivakumar M, Andaz S, et al: Pseudo-aneurysm of the carotid bifurcation secondary to radiation. J Cardiovasc Surg (Torino) 40:877-878, 1999
60. Chen YJ, Wang CP, Wang CC, et al: Carotid blowout in patients with head and neck cancer: Associated factors and treatment outcomes. Head Neck 37:265-272, 2015
61. Chang FC, Lirng JF, Luo CB, et al: Carotid blowout syndrome in patients with head-and-neck cancers: Reconstructive management by self-expandable stent-grafts. AJNR Am J Neuroradiol 28:181-188, 2007
62. Cheng SW, Ting AC, Lam LK, et al: Carotid stenosis after radiotherapy for nasopharyngeal carcinoma. Arch Otolaryngol Head Neck Surg 126:517-521, 2000
63. Xu J, Cao Y: Radiation-induced carotid artery stenosis: A comprehensive review of the literature. Interv Neurol 2:183-192, 2014
64. Huang TL, Hsu HC, Chen HC, et al: Long-term effects on carotid intima-media thickness after radiotherapy in patients with nasopharyngeal carcinoma. Radiat Oncol 8:261, 2013
65. Gujral DM, Shah BN, Chahal NS, et al: Clinical features of radiation-induced carotid atherosclerosis. Clin Oncol (R Coll Radiol) 26:94-102, 2014
66. Upile T, Triaridis S, Kirkland P, et al: The management of carotid artery rupture. Eur Arch Otorhinolaryngol 262:555-560, 2005
67. Fankhauser GT, Stone WM, Fowl RJ, et al: Surgical and medical management of extracranial carotid artery aneurysms. J Vasc Surg 61:389-393, 2015
68. Powitzky R, Vasan N, Krempl G, et al: Carotid blowout in patients with head and neck cancer. Ann Otol Rhinol Laryngol 119:476-484, 2010
69. Shichita T, Ogata T, Yasaka M, et al: Angiographic characteristics of radiation-induced carotid arterial stenosis. Angiology 60:276-282, 2009
70. Tallarita T, Oderich GS, Lanzino G, et al: Outcomes of carotid artery stenting versus historical surgical controls for radiation-induced carotid stenosis. J Vasc Surg 53:629-636, 2011. e621-625