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Interventional Neuroradiology Adjuncts and Alternatives in Patients with Head and Neck Vascular Lesions

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Endovascular techniques can be applied to a variety of benign and malignant extracranial lesions of the head, neck, and skull base. We discuss the role of endovascular techniques in the management of benign and malignant head and neck neoplasms, acute and delayed presentations of vascular traumatic injury, and acute management of head and neck bleeding. We discuss endovascular options and management strategies for treatment of extracranial lesions of the head, neck, and skull base.

Neoplastic disease

Benign and malignant tumors may compromise head and neck vascular supply or derive significant neovascularity from the extracranial vasculature. In most cases, a CT scan with or without the addition of CT angiography (CTA) or MRI with MR angiography (MRA) can demonstrate the relation of the mass to the vasculature, provide a differential diagnosis, and provide an opportunity for image-guided biopsy. Catheter angiography is reserved for those cases in which significant neovascularity is expected or where the potential for major vessel compromise is present, requiring a temporary balloon occlusion tolerance test (BTO) or major vessel sacrifice before en bloc resection [1–5].

Benign neoplasms

Benign tumors of the head, neck, and skull base may compress, displace, or encase the carotid or vertebral arteries, making complete resection difficult. Successful preoperative BTO of the carotid or vertebral artery before surgery may provide the surgeon with increased assurance in surgical planning and patient counseling. Schwannoma and neurofibroma of the neck or skull base, paraganglioma (carotid body tumor, glomus vagale, or glomus jugulare), and esthesioneuroblastoma or meningioma with a significant extracranial component as well as tumors with significant cavernous sinus extension may benefit from preoperative BTO [6,7].

Preoperative BTO of the vertebral artery may be followed by preoperative vertebral artery sacrifice before resection of aggressive benign spine tumors, such as osteoblastoma or an aneurysmal bone cyst (Fig. 1) [8].

Temporary balloon occlusion tolerance test

BTO is composed of an angiographic anatomic and clinical neurologic assessment of a patient's

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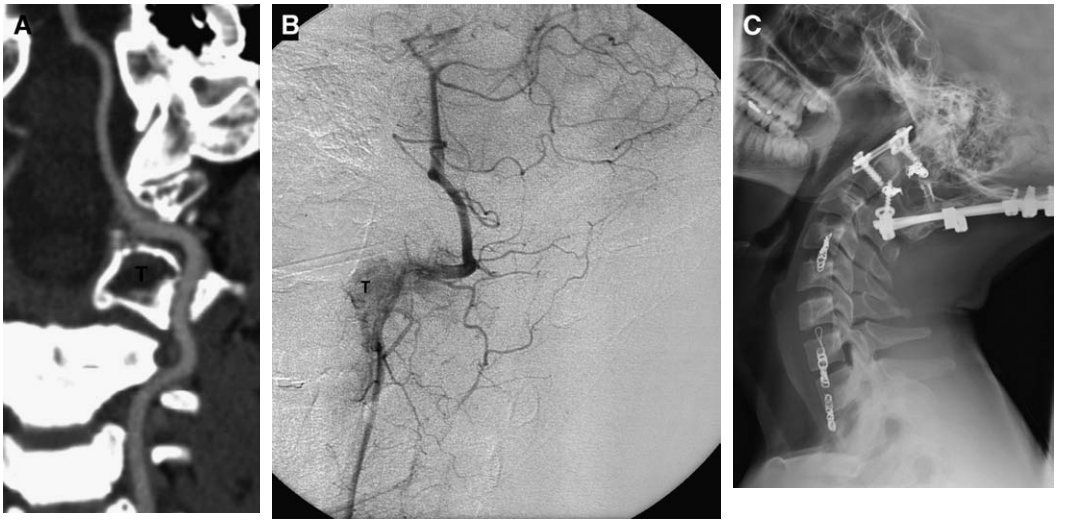

Fig. 1. (*A–C*) CTA shows patent vertebral artery encased by osseous C_2 tumor (T). Vertebral angiogram reveals a hypervascular mass. Preoperative vertebral artery sacrifice using platinum microcoils was performed in this adolescent with C1 osteoblastoma.

ability to tolerate temporary occlusion of a major vessel [6,7,9,10]. The components include a diagnostic arteriogram to assess anatomic collaterals, administration of heparin before inflation of a contrast-containing nondetachable balloon with occlusion of the major vessel, and clinical neurologic testing while the balloon is inflated for approximately 30 minutes. If the patient tolerates the normotensive temporary occlusion, hypotension may be pharmacologically induced with reduction of the mean arterial pressure (MAP) to two thirds of the baseline blood pressure and extending the temporary occlusion for an additional 15 minutes. This hypotensive challenge is analogous to a stress or exercise state and may unmask subtle hypoperfusion. For BTO of the anterior circulation, the predictive value can be improved by the injection of the radionuclide Tc99m hexamethylpropyleneamine-oxime (HM-PAO; exametazime) before brain single photon emission CT (SPECT) imaging [9]. Tc99m HM-PAO is a single-pass blood flow agent that provides a snapshot of cerebral blood flow at the time of injection. Cerebral SPECT imaging can take place up to 6 hours after injection and reflects the cerebral blood flow at the time of hypotension and BTO of the major vessel (Fig. 2). After injection of Tc99m HM-PAO, the balloon is deflated, antihypertensive medication is discontinued, and blood flow is restored to the cerebral circulation. SPECT imaging is of lesser value for assessment of the posterior fossa after vertebral artery temporary occlusion and is rarely performed.

The predictive value of tolerance BTO of the internal carotid artery with a combination of angiographic demonstration of anatomic collaterals, normotensive and hypotensive clinical tolerance of test occlusion, and satisfactory perfusion on brain SPECT is approximately 85% that the patient can tolerate permanent occlusion without infarction [10–12]. Risks of the test occlusion include stroke and local blood vessel injury in addition to the overall risks of cerebral angiography.

Preoperative embolization is commonly used for juvenile nasal angiofibroma, esthesioneuroblastoma, meningioma, schwannoma, neurofibroma, and paraganglioma. Preoperative particulate embolization using polyvinyl alcohol (PVA) foam particles measuring 250 to 350 μm in size 1 to 2 days before surgical excision increases safety by decreasing blood loss at the time of surgery and thus facilitates a safer and more complete resection. The embolization can be performed in the same angiographic day following BTO and SPECT (Fig. 3) [3,4].

Extracranial transarterial tumor embolization adds a small risk to the performance of cerebral angiography. The principal risks include blood vessel injury and stroke. With some tumors of the skull base, there is a risk of opening potential collaterals between the extracranial and intracranial

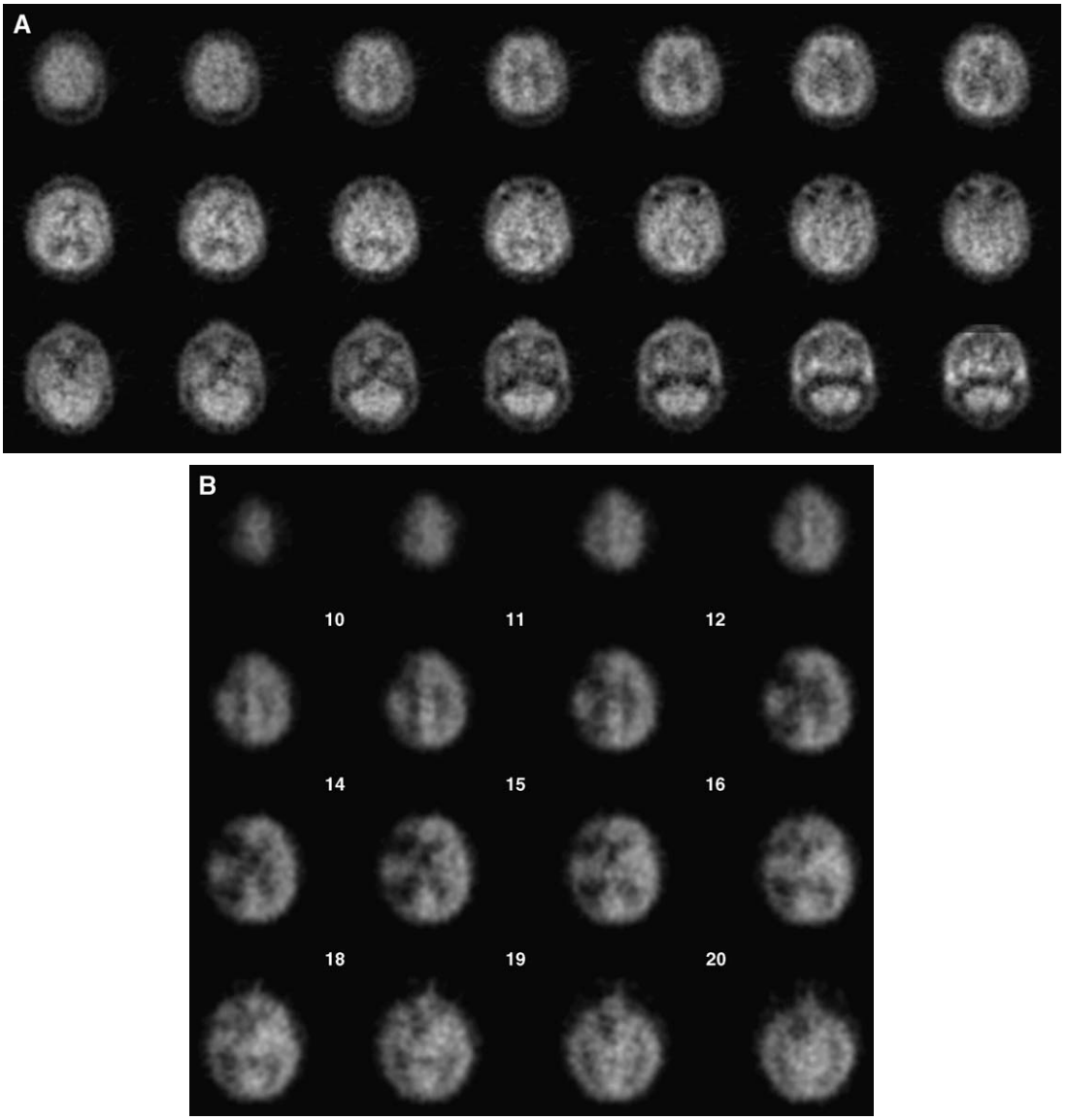

Fig. 2. Temporary carotid balloon occlusion test (BTO) is performed with a contrast-filled non-detachable balloon catheter. Tc99m HM-PAO is injected during temporary occlusion. (*A*) SPECT following carotid BTO demonstrates no differential cerebral hemisphere hypoperfusion. (*B*) SPECT demonstrates marked hypoperfusion in a patient with massive carotid bleeding following urgent endovascular occlusion.

circulations, such as the inferolateral trunk and the vidian artery (internal maxillary artery to internal carotid artery) or middle meningeal artery to ophthalmic artery during the embolization procedure resulting in cerebral emboli. Similarly, extracranial-to-intracranial anastomoses occur between the vertebral artery and the ascending pharyngeal and occipital arteries [2,5]. Extracranial tumoral embolization is well tolerated by patients, although they may experience some increase in pain in the region of a large tumor mass resulting from tumoral ischemia and edema after embolization.

Malignant neoplasms

Malignant neoplasms of head and neck origin predominantly include tumors of squamous cell

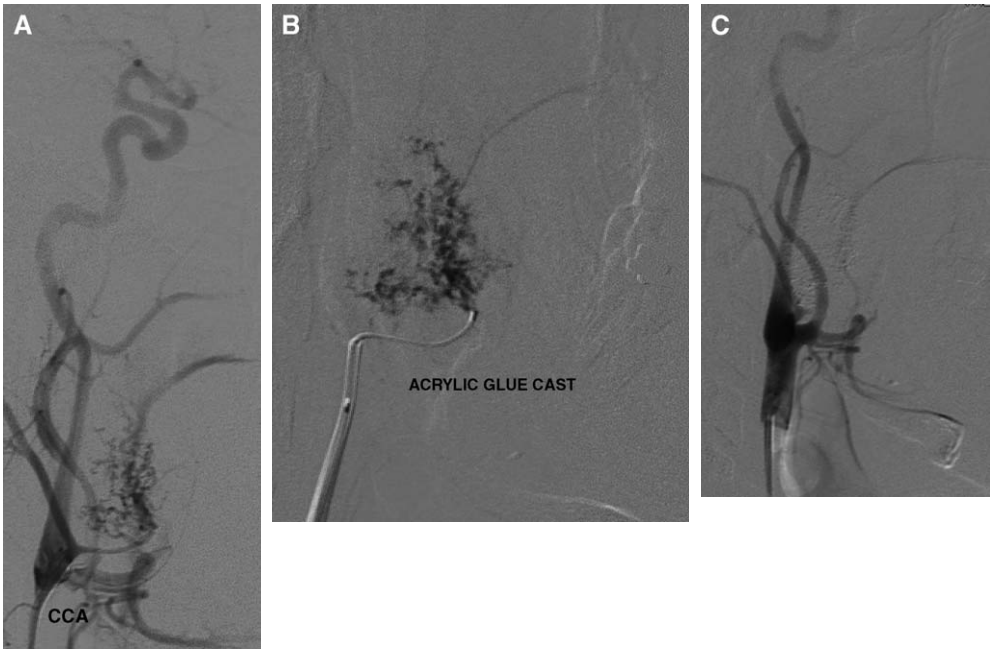

Fig. 3. (*A–C*) Preoperative embolization of a large, hypervascular, parapharyngeal space schwannoma using a combination of PVA particles and acrylic.

origin involving the paranasal sinuses, oral-digestive tract, or larynx. Adenocarcinomas and mucoepidermoid carcinomas arising from minor salivary glands as well as other more unusual tumors may arise in this region. Many of these lesions extend to involve the skull base and create symptoms in the central nervous system. Most of these malignancies are relatively hypovascular, with the exception of malignant paraganglioma; thus, most do not require preoperative embolization. In large tumors adjacent to the carotid artery, en bloc resection may require sacrifice of the carotid artery [3]. BTO followed by endovascular carotid sacrifice 3 to 6 weeks before en bloc resection is associated with a lower stroke risk than is surgical carotid sacrifice at the time of en bloc resection [6]. The interval of a few weeks allows re-equilibration of the cerebral blood flow before the stress of major surgical resection. After endovascular carotid sacrifice, these patients are managed in the neurointensive care unit with heparin therapy and control of fluid dynamics to decrease stroke risk during the equilibration period. There is limited experience with intra-arterial administration of chemotherapeutic agents into locally recurrent or locally advanced and unresectable tumors [13–17]. Despite significant local toxicity, early results suggest tumor regression that may permit salvage surgery. These techniques have not yet become the standard of care for advanced head and neck cancers.

Carotid blow-out syndrome

Many of these patients receive intraoperative brachytherapy as part of their tumor management. The combination of malignant disease recurrence, surgery, and radiation therapy may result in vascular injury with tumoral encasement, vessel wall ulceration, or pseudoaneurysm formation. Radiation injury to the carotid and or vertebral arteries in the neck may result in symptomatic stenosis and present with transient ischemic attack (TIA) or stroke, even without residual tumor (Fig. 4). Patients more commonly present with acute oral, nasal, or paratracheal bleeding weeks to months after the initial presentation and surgery. The exact site of injury relates to the location of the tumor in relation to the major vessels, the degree of intrinsic tumoral vascularity, the presence of tumoral or mucosal ulcerations, and the types of prior treatment. Localization of surgical clips or brachytherapy seeds is a good indication of the site of potential

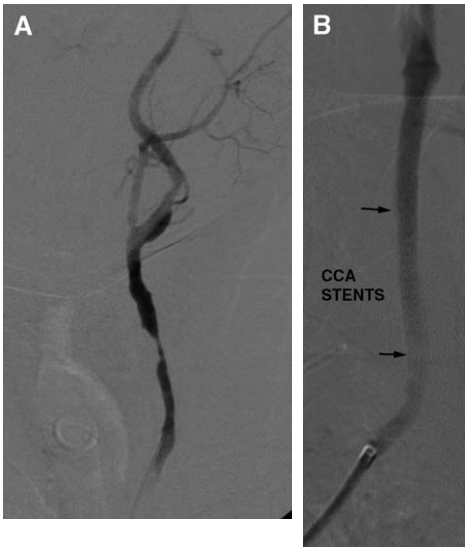

Fig. 4. (*A*) After successful treatment of left middle cerebral stroke with intravenous tissue plasminogen activator, angiography revealed left common carotid stenosis 12 years after radiation therapy for laryngeal carcinoma. (*B*) Two overlapping stents were placed.

vascular injury leading to hemorrhage. When the oral, nasal, or paratracheal bleeding is mild to moderate, it may result from erosion through the wall of a small vessel and can usually be treated with vessel-specific embolization with particles or acrylic designed to maintain patency of the major vessels.

The term *carotid blow-out* (CBO) specifically refers to catastrophic rupture of the common, internal, or external carotid artery or their branches with life-threatening hemorrhage [18–23]. Rapid initiation of resuscitation securing airway followed by oral or nasal packing and angiography with endovascular occlusive techniques may be lifesaving (Fig. 5). For carotid rupture, the standard of care is sacrifice of the carotid artery above and below the point of rupture. It is important to include areas of intimal irregularity as demonstrated on angiography or, alternatively, to span the region of vessel encasement as visualized on cross-sectional imaging. Carotid occlusion by detachable balloons has been the mainstay of therapy, although these are currently unavailable in the United States. Alternatively, platinum microcoils, usually a combination of detachable and complex helical coils, can be

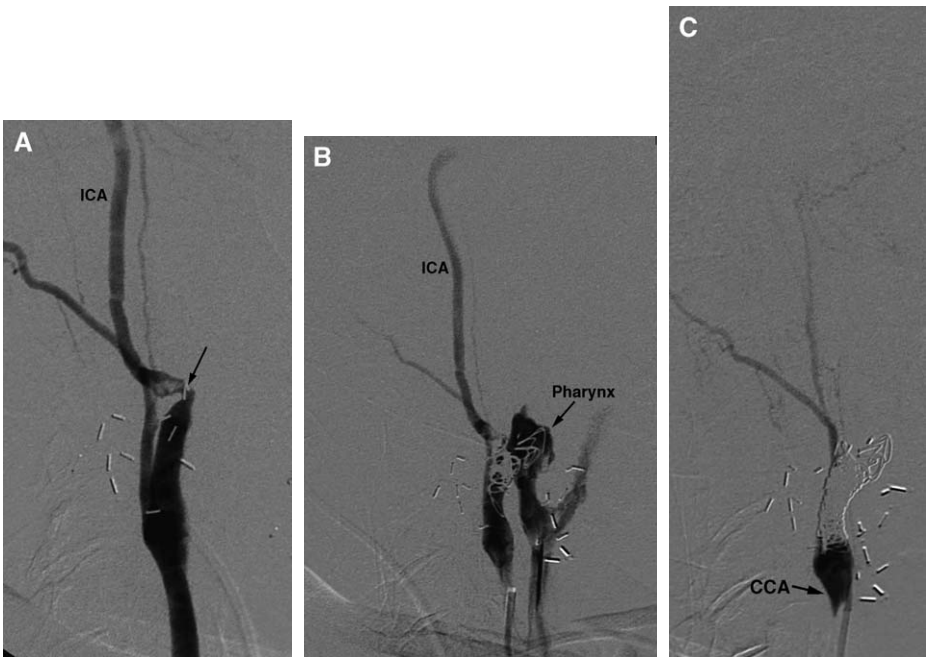

Fig. 5. (*A–C*) CBO with permanent coil occlusion. Marked intimal irregularity ICA and frank extravasation from the internal cartoid into the pharynx in a patient with recurrent squamous cell carcinoma of the hypopharynx.

used for effective carotid occlusion. A combination of platinum and fibered platinum coils can be used to promote effective and rapid occlusion. Even when frank rupture is not demonstrated angiographically, the treatment of a pseudoaneurysm and ulceration in the common or internal carotid artery usually requires carotid sacrifice. Resuscitation and packing usually allow enough time for rapid angiographic assessment of the circle of Willis before endovascular occlusion. When life-saving carotid sacrifice must be performed without preocclusion testing, HM-PAO SPECT may be performed after the occlusion to assist in planning for aggressive hypertensive and hypervolemic management [6,7,10].

We have had limited experience with good success at our institution in treating patients without adequate circle of Willis collaterals with covered stents to cover the area of vascular injury and to preserve cerebral blood flow. This technique is experimental, and there are significant limitations in terms of the size and length of available stents (generally approved for biliary or tracheobronchial use). Increased availability of stents in smaller (<5 mm) diameters and longer (>5 cm) lengths may allow more widespread application of this cerebral perfusion–saving technology to this group of critically ill patients (Fig. 6) [18,20,21].

Traumatic vascular injuries

Vascular injury from blunt or penetrating trauma may present acutely with expanding hematoma or oral or nasal bleeding or may present in a delayed fashion, such as in the development of a pseudoaneurysm or carotid cavernous fistula [24–28]. A CT scan, with or without adjunctive CTA, is useful for defining the location of fracture fragments or the path of a projectile and may demonstrate the injury before endovascular or surgical treatment. Angiography may demonstrate vessel laceration, vasospasm, dissection, occlusion, or pseudoaneurysm formation, with or without distal embolization. Oral or nasal bleeding secondary to facial fractures may initially be treated by packing in the emergency room, followed by angiographic evaluation of the common, internal, and external carotid arteries and their branches. Transarterial embolization is performed similar to that described for idiopathic epistaxis. Temporary agents, such as Gelfoam pledgets (Pharmacia & Upjohn, Kalamazoo, Michigan) or larger PVA foam particles (250–350 μm or 300–500 μm), are useful to provide immediate hemostasis and to allow healing without compromising facial arterial supply

Vertebral artery injury most commonly results from cervical spine fractures caused by blunt or

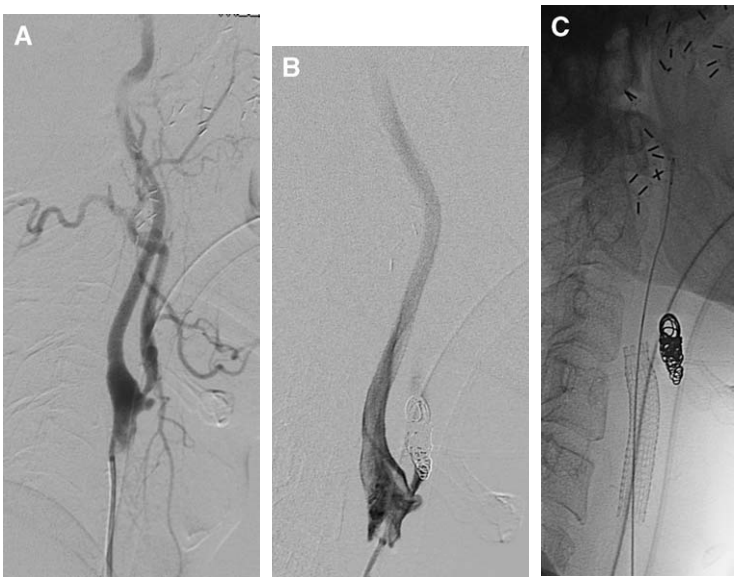

Fig. 6. (*A–C*) CBO with massive oral bleeding and pseudoaneurysm at the carotid bulb. Fibered coils were placed to exclude the proximal external carotid artery before covered stent placement in the common and internal cartoid artery with cessation of bleeding.

penetrating injury with resultant dissection, occlusion, pseudoaneurysm, or fistula formation. The vertebral artery is especially vulnerable to injury at the anatomic points of fixation, such as the entrance into the foramen transversarium at C5 to C6, the junction of C1 and C2, and above C1, where the vertebral artery pierces the dura. CTA at the time of the initial CT scan may demonstrate loss of integrity of the transverse foramen and demonstrate the concomitant vascular injury. Persistent antegrade flow in dissected or partially occluded vertebral arteries may lead to distal embolization and stroke. Heparin with or without antiplatelet therapy has become the mainstay of treatment for such patients, although vertebral artery sacrifice remains a viable treatment strategy to prevent distal embolization in patients with adequate posterior circulation collateral flow (carotid arteries and contralateral vertebral artery). Stent technology is not an important form of treatment for vertebral artery injury. This is largely related to the small size of the vertebral artery and the difficulty in maintaining stent patency in small vessels.

A vertebral-jugular fistula most commonly results from penetrating trauma and may be associated with local cervical hematoma and hemodynamic instability. Transarterial embolization, usually resulting in sacrifice of the ipsilateral vertebral artery, may be curative and is well tolerated if adequate collateral circulation is present. Rarely, a venous endovascular approach or direct surgical repair is necessary for control.

Carotid artery injury may result from direct injury to the carotid and neck or may be the result of a skull base fracture with arterial dissection [24–29]. As with the vertebral artery, fixation points, such as the skull base, where the carotid pierces the dura, and within the cavernous sinus, render the carotid at the skull base more vulnerable to injury. Mandibular fractures are commonly associated with common or proximal internal carotid artery injury. Direct trauma to the common carotid artery may result in dissection or pseudoaneurysm formation with or without distal embolization (Fig. 7). Similarly, dissection leading to occlusion or pseudoaneurysm formation may occur with injuries of the internal carotid artery. A pseudoaneurysm of the internal carotid artery may be treated expectantly with antiplatelet agents, with or without heparin as for vertebral injuries. A pseudoaneurysm of the common carotid artery is generally treated by surgical repair or by endovascular treatment with bare or covered stents to maintain distal intracranial supply [30–35]. Surgical repair of pseudoaneurysms may be curative, but access may be difficult when the aneurysm is located near the skull base or behind the mandible. Endovascular treatment, particularly with covered stent technology, can be useful in this setting. The use of antiplatelet therapy at the time of stent placement is critical to diminish platelet adhesion in the setting of vascular injury. A pseudoaneurysm of the internal carotid artery may also present in a delayed fashion as a localized mass, with local pain or Horner's syndrome. We have found CTA useful for following patients with a pseudoaneurysm on antiplatelet therapy before intervention (Fig. 8).

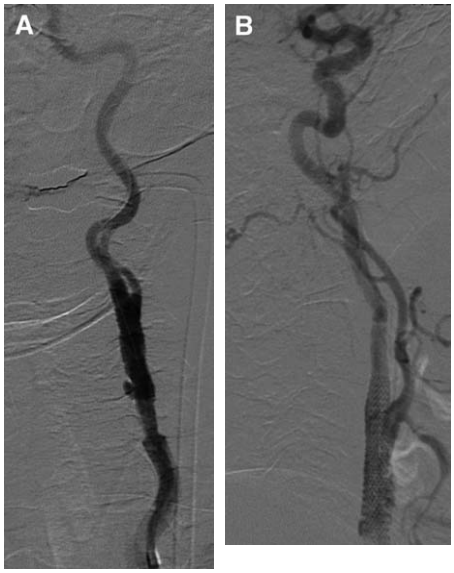

Fig. 7. (*A*) Common and internal carotid dissection secondary to seatbelt injury during a motor vehicle accident. (*B*) The patient was treated with two overlapping stents and antiplatelet therapy.

Traumatic carotid cavernous fistula

Delayed development of a traumatic carotid cavernous fistula is a known complication of closed-head injury. Unlike patients with vertebral-jugular fistulas, these patients do not present with hematoma or hemodynamic instability; instead, they present with proptosis or chemosis, with or without diplopia. Patients may develop increased intraocular pressure leading to visual impairment. Emergency treatment is rarely

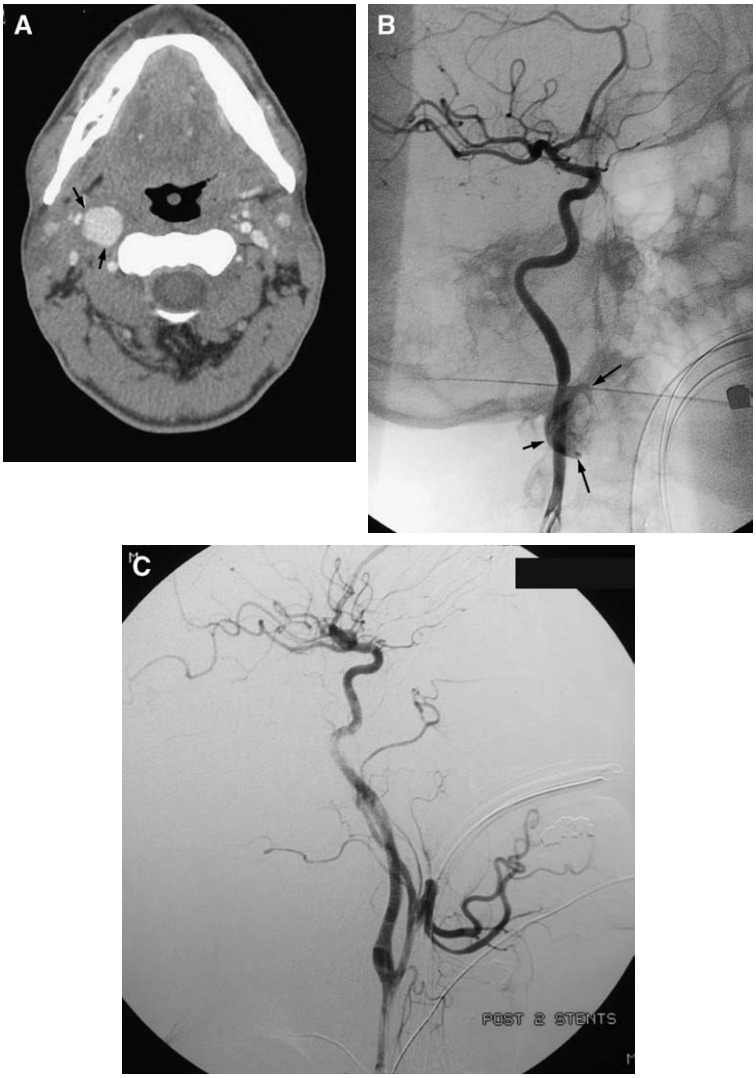

Fig. 8. (*A–C*) Intimal dissection and pseudoaneurysm (*arrows*) 1 month after motor vehicle accident seen on CT for neck mass. Angiography was followed by overlapping stent placement with exclusion of the pseudoaneurysm.

indicated, except in the setting of impending visual loss. Endovascular treatment may include closure of the fistula via an arterial approach (detachable balloons or platinum microcoils) or, alternatively, closure of the fistula via a venous approach (petrosal sinus or superior ophthalmic vein approach with platinum microcoils). The endovascular approach is dictated by the anatomy of the fistulous communication. In some cases, the severity of the carotid injury may lead to parent artery occlusion at the time of fistula treatment (Fig. 9) [36–38].

Intraoperative and perioperative vascular compromise

Intraoperative and perioperative vascular compromise may occur either during direct vascular surgery, such as an endarterectomy, or may occur in any setting in which tumor surrounds major vascular structures, that often alters the normal anatomic position and relations to adjacent structures [37–42]. The sphenoid sinus has a close relation to the internal carotid artery at its lateral and superior walls. The bone can be particularly

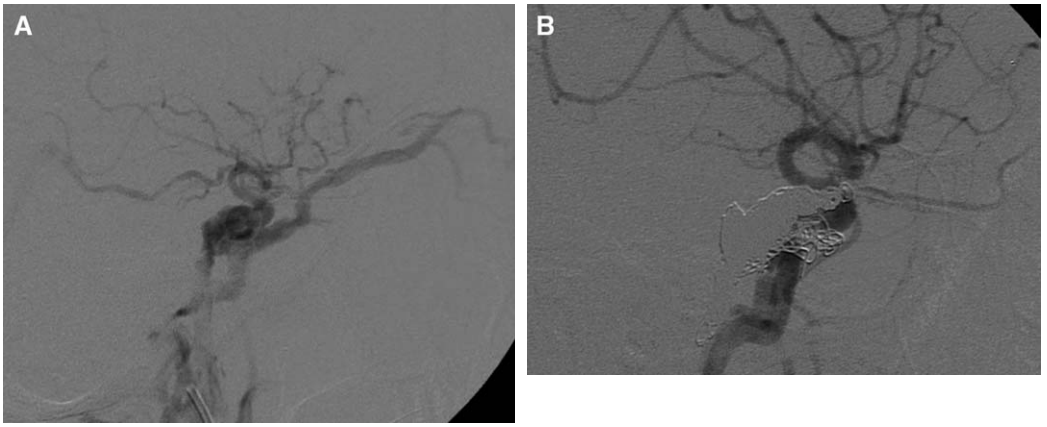

Fig. 9. (*A,B*) Carotid cavernous fistula developing after a fall was treated transvenously with coils via the petrosal sinus.

thin overlying the carotid and may be distorted in the presence of tumor or inflammatory disease. Trans-sphenoidal surgery for intrinsic sphenoid pathologic findings or for pituitary tumor removal may result in injury to the adjacent internal carotid artery, leading to hemorrhage, pseudoaneurysm, or occlusion (Figs. 10 and 11) [40]. Infection or tumor invasion involving the internal carotid at the skull base may result in similar pathologic findings (Fig. 12). Intraoperative measures to control bleeding locally are instituted in addition to sphenoid sinus packing before angiographic assessment. Endovascular repair may require carotid artery sacrifice after assessment of the remaining cerebral vasculature for adequacy of the circle of Willis. In most cases, the incomplete wall of the pseudoaneurysm in the face of injury is too fragile for coil embolization to prevent the possibility of rupture. Currently available covered stents may not be small enough or flexible enough to negotiate the curves of the petrous and cavernous internal carotid segments routinely [38]. Parent vessel occlusion remains the mainstay of treatment for most postsurgical vascular injuries.

After placement of screws and plates for facial fracture fixation or, occasionally, after tracheostomy insertion, oral or peritracheal bleeding may occur. In the acute postoperative period (5–10 days after surgery), vascular injury with laceration

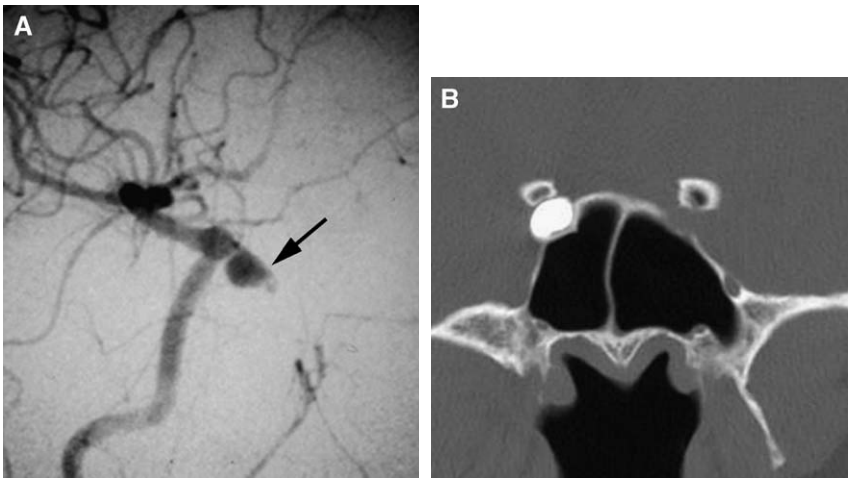

Fig. 10. (*A,B*) Intraoperative bleeding encountered during sphenoid sinus surgery. After packing, the angiogram revealed a pseudoaneurysm (*arrow*) treated with carotid occlusion. CT demonstrates the occlusive coil pack occlusion balloon position relative to the sphenoid sinus.

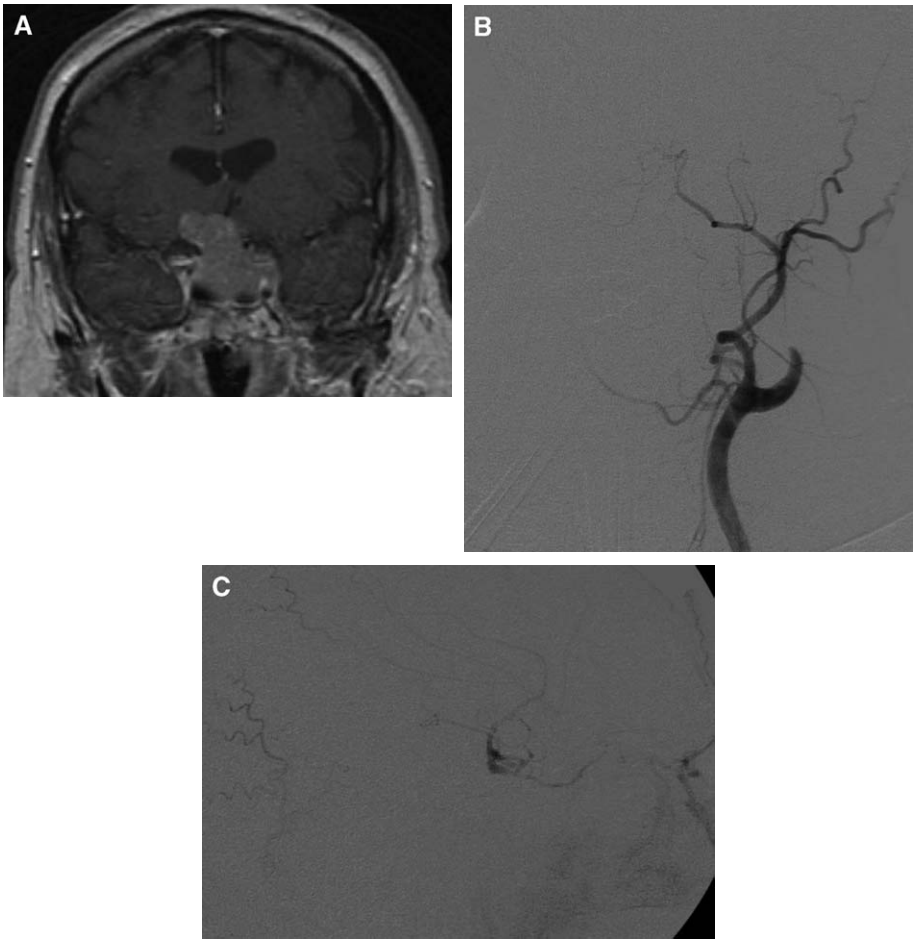

Fig. 11. (*A*) Intraoperative bleeding encountered during transphenoidal hypophysectomy for a large pituitary tumor seen on MRI. After packing, angiography revealed carotid occlusion (*B*) and poor circle of Willis collaterals on the intracranial view (*C*).

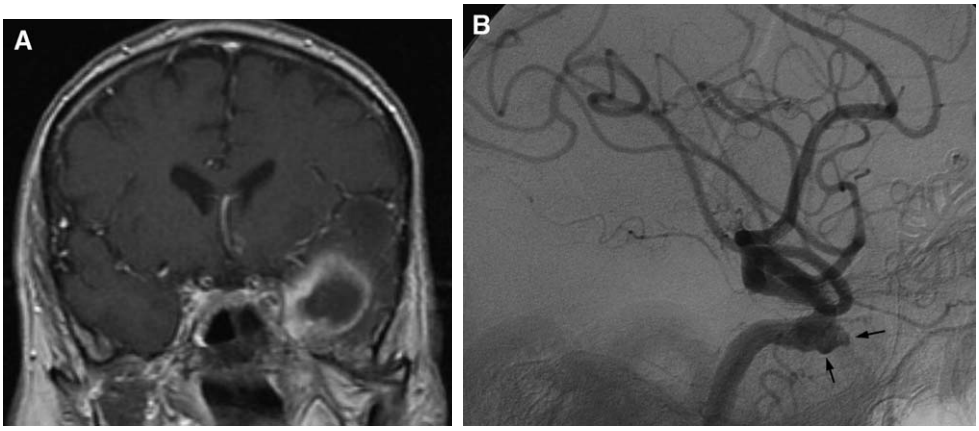

Fig. 12. (*A*) Controlled intraoperative bleeding encountered during removal of a medial temporal lobe abscess, seen on MRI, associated with mucoepidermoid carcinoma. Angiography showed minor intimal irregularity (not shown). (*B*) Massive oral bleeding occurred 5 months later; revealed a pseudoaneurysm (*arrows*) which was treated by carotid occlusion.

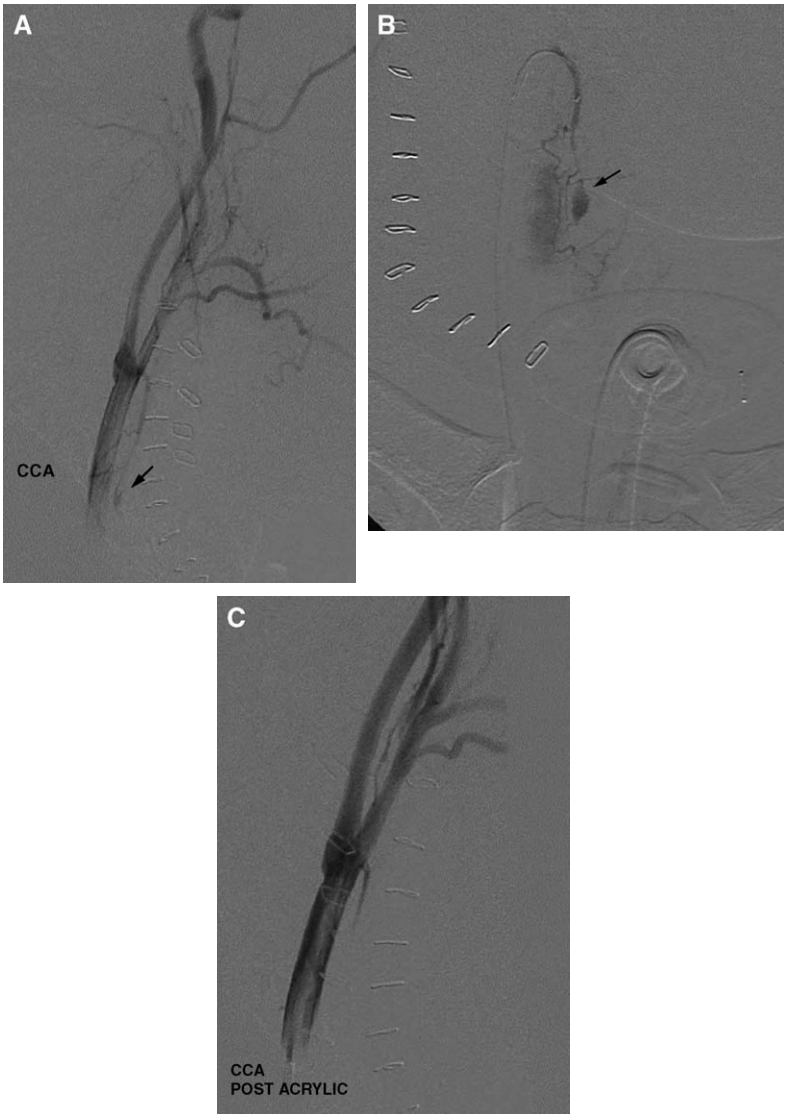

Fig. 13. Peritracheal bleeding occurred 9 days after bilateral radical neck surgery and tracheostomy. Common carotid angiography (*A*) faintly demonstrates a superior thyroidal pseudoaneurysm better seen on microcatheterization (*B*). (*C*) It was embolized with acrylic.

and a secondary pseudoaneurysm is the most commonly identified angiographic abnormality. Embolization of the lesion using particulate embolic material (PVA foam particles, Gelfoam, or coils) or, in the setting of an end artery, acrylic glue is usually efficacious for bleeding control (Fig. 13).

TIAs or hemiparesis after carotid endarterectomy may result from carotid occlusion, often requiring reoperation. Emergent angiography or, more currently, CTA may be performed to establish the cause of the symptomatology and the nature of the vascular compromise before surgical re-exploration. Stent technology might also be applied in this postsurgical setting.

Epistaxis and other endovascular interventions in the head and neck

When no traumatic, neoplastic, or intrinsic vascular cause for epistaxis is identified, it is referred to as idiopathic. Epistaxis may require

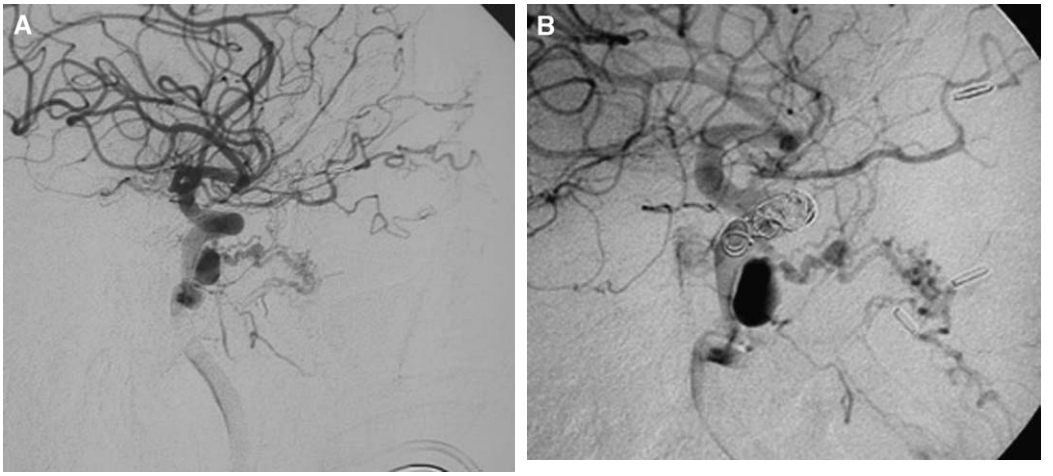

Fig. 14. (*A,B*) CBO with massive bleeding in an 80-year-old patient with HHT and multiple prior external cartoid branch ligations (clips). He had had multiple prior embolizations and developed this internal carotid pseudoaneurysm and extensive nasal collaterals, seen most clearly after partial carotid occlusion. Internal carotid coil occlusion resulted in cessation of bleeding.

endovascular treatment when posterior packs fail to control the bleeding [3,4,43]. Transarterial embolization consists of distal particulate embolization (PVA foam particles measuring 250–355 μm) in the distal internal maxillary artery and distal facial artery on the ipsilateral side and embolization of the distal internal maxillary or facial artery on the contralateral side, reserving a single branch to maintain adequate collateral blood supply to the nasal tissues. Transarterial embolization is well tolerated and highly effective in the idiopathic epistaxis patient population, with a low complication rate (2%).

Hereditary hemorrhagic telangiectasia (HHT) is an autosomal dominant vascular dysplasia with telangiectasia of the nasal and gastrointestinal mucosa, skin, and respiratory tract as well as arteriovenous malformations of the lung, liver, and brain [44]. Epistaxis may be difficult to control endovascularly (Fig. 14). Septal dermoplasty has been found to be more effective and durable in the management of transfusion-dependent epistaxis [45].

Summary

Vascular lesions of the head and neck include a variety of neoplastic and traumatic lesions that may cause local neurologic symptoms or may compromise the carotid or vertebral arteries, leading to ischemic deficits. Management of lesions involving vascular structures at the skull base may require BTO or endovascular transarterial embolization as part of the preoperative evaluation. Endovascular techniques can be used as a salvage measure for severe head and neck bleeding and can assist with the management of vascular injury occurring in the operative or perioperative setting. Familiarity with the role of endovascular techniques in this group of patients may favorably influence patient management and outcome.

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