17 The Role of Neurointervention in Traumatic Vascular Injury and Vascular Surgery

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Introduction

The evolution of the treatment of vascular injury and disease toward endovascular approaches has brought the trauma surgeon, vascular surgeon, and neurointerventionalist into contact with great potential synergies between the fields. In this chapter, we explore some of the more common areas in which these specialists collaborate.

Screening for Vascular Injury

Neurovascular injury associated with blunt trauma is too often recognized late in the clinical course of the trauma patient. During the acute phase of assessment and stabilization, the focus on airway protection and need for emergent surgery may lead to intubation without a preceding detailed neurologic assessment. If neurovascular injury is not considered in this early phase, the diagnosis may not be made until days later when the patient is noted to have neurological deficits in the intensive care unit. At this point, the neurological injury is often complete [1]. These injuries are associated with mortality as high as 23% and morbidity of 48%, adding to the urgency of early diagnosis [2].

The need to more rapidly diagnose neurovascular injury has led to the development of institutional and societal guidelines for screening (see Table 17.1).

CT angiography (CTA) has emerged as the preferred screening test for carotid or vertebral artery traumatic injury [3–6]. It has the advantage of rapid availability in most hospitals and short acquisition times. The latter factor is vital in the trauma population given the potential of hemodynamic instability and/or patient movement during the examination. CTA has nearly 100% sensitivity for carotid and 96% sensitivity for vertebral injuries when reviewed by experienced radiologists [4].

While highly sensitive, there is up to a 43% false-positive rate seen with CTA used for traumatic neurovascular injury screening [7]. Catheter-based angiography, thus, continues to have an important role as the definitive diagnostic

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Table 17.1 Clinical and radiological criteria to screen for neurovascular injury*

Clinical criteria	Radiological criteria
Penetrating head injury with a GCS < 13	Cervical spine fracture involving the foramen transversarium, occipital condyle, or C1-3
Closed head injuries (CHI) consistent with DAI and GCS < 6	CT or MR evidence of cerebral infarction
Arterial hemorrhage from neck, mouth, nose, ears	Displaced midface fracture (LeFort II or III)
Large expanding cervical hematoma	Skull base fracture involving the carotid canal
Cervical bruit in a patient younger than 50	30% or greater subluxation of a vertebral body at or above C5-6
Focal or lateralizing neurologic deficit	
Transient ischemic attack	
Horner's syndrome	
Oculosympathetic paresis	
Vertebrobasilar insufficiency	
Neurologic deficit that is incongruous with CT or MRI findings	
Near hanging with anoxia	
Clothesline type injury or seat belt abrasion with significant swelling, pain, or altered MS	

Adapted from Biffl et al. [3]
Kansagra et al. [8]

tool when CTA shows possible neurovascular injury and should be strongly considered when CTA findings are equivocal.

Traumatic vascular injuries of the head and neck can be dynamic for several days after injury. Dissecting injuries may improve or worsen spontaneously, with initially mild luminal narrowing progressing to vessel occlusion in some cases, and in others with injuries becoming less apparent on subsequent imaging. In addition, vessel injuries are at times associated with delayed thrombus formation which could lead to a switch from antiplatelet agents to anticoagulation. For these reasons, follow-up imaging 5–7 days after injury is initially diagnosed is generally recommended [3] (Table 17.2).

Table 17.2 Denver grading scale for blunt cerebrovascular injuries [9, 10]

Grade	Description
I	Irregular vessel wall or dissection/intramural hematoma with <25% luminal stenosis
II	Intraluminal thrombus or raised intimal flap, or dissection/intramural hematoma with 25% luminal stenosis
III	Pseudoaneurysm
IV	Vessel occlusion
V	Vessel transection

Penetrating Injuries

Penetrating injuries of the neurovasculature are most commonly due to gunshot wounds (GSW) and stab wounds (SW). The locations of injury have been divided into three zones: Zone 1, which extends from the clavicle to the cricoid cartilage; Zone 2, from cricoid cartilage to mandible; and Zone 3, superior to the angle of the mandible. Zone 2 is the most common and the most surgically accessible location of injury. In patients with hemodynamic instability from active extravasation/bleeding or airway compromise due to a large hematoma, open surgical exploration is indicated for injuries in this location. If the patient is hemodynamically stable without hard signs of vascular trauma, further testing and medical management is recommended. In Zone 1 and 3 injuries, endovascular surgery may be preferable given the complexity of open exploration. Such interventions may include placement of covered stents or coils in the setting of ongoing hemorrhage and hemodynamic compromise or stent repair in the setting of carotid or vertebral injury with resultant stenotic lesions and hemodynamic stability. These procedures obviate the need for antiplatelet medications and intraoperative anticoagulation, which should be considered when planning intervention in trauma patients with other injuries [11].

Occasionally, facial fractures or bullet fragments lead to transection of branches of the external carotid artery. Such injuries are well addressed endovascularly with particulate embolic materials or liquid embolics.

Blunt Injuries

Carotid Artery Dissection

Internal carotid artery (ICA) dissection has been increasingly recognized as a primary cause of stroke in young patients, accounting for 20% of strokes in patients younger than 45 years of age. However, carotid dissection is an infrequent cause of stroke in the general population, accounting for only 0.4 to 2.5 percent of all strokes [12, 13]. The cervical segment of the ICA is frequently affected. While classic mechanisms of injury are described as being associated with posterior oropharyngeal injuries, such as falls while holding objects orally; there is great heterogeneity in traumatic etiology. Traumatic dissection can be caused by blunt trauma to the neck directly or in association with trauma subjecting the neck to excessive flexion, extension, or rotation. A common mechanism involves compression of the internal carotid artery against the lateral process of cervical vertebra [14]. This inciting trauma results in an intimal tear or intramural hematoma allowing for intima-media separation. Cerebral ischemia results from direct luminal narrowing by the dissection flap, subsequent thrombosis at the site of dissection, or distal embolization of micro emboli created from turbulent flow and a now thrombogenic vessel wall.

Headache and ipsilateral neck pain are the commonest clinical findings present in 75% of patients, and a minority will present with Horner syndrome. Though uncommon, the findings of pupillary constriction, ptosis, and anhidrosis in combination with trauma, minor or otherwise, should prompt investigation into a possible dissection [15–17].

Angiography, usually initially obtained via computed tomography, is the diagnostic gold standard, which shows differing radiologic patterns depending on severity of injury. Small intimal defects may be evident by a small vessel wall irregularity or minor filling defect due to associated thrombus. Larger dissections tend to create a linear filling defect that may or may not be flow limiting. Though identification of an intimal flap is specific for carotid dissection, it is only seen in 10% of patients with dissection. The most common angiographic finding being irregular narrowing with a smooth luminal taper can be seen in 65% of patients [18].

Medical treatment is the mainstay of management of flow limiting carotid dissection. There is some disagreement in the literature over the first-line antithrombotic medication. The trauma surgery literature often advocates for intravenous heparin as a first-line treatment, while neurological studies have generally shown no advantage of anticoagulation over antiplatelet agents [19, 20]. The use of either class is complicated by the patient with polytrauma, particularly in those with bleeding risk and in the absence of neurologic findings. If symptoms worsen despite medical therapy, surgical management may be offered to select patients. Surgical intervention is usually reserved for patients in extremis, either from active hemorrhage or a focal thrombosis in the context of new-onset neurologic deficits, and consists of either bypass or rarely ligation [21]. Endovascular management has generally been shown to be low risk in the stable patient who can be appropriately pre-treated with dual antiplatelet therapy and who have anatomy suitable for these intervention. Self-expanding, Nitinol, stents are typically used and sized to the diameter of the carotid segment in question [22, 23]. Adjunctive angioplasty is generally not needed but can be used if luminal narrowing persists after stent deployment [24]. Indications for treatment include impaired cerebral perfusion, infarction despite appropriate antithrombotic use, or acute, ongoing stroke [25].

A carotid artery “pseudoaneurysm” occurs when the dissection tract is between the media and adventitia and the adventitia bows outward. Arterial flow permits continued perfusion of the newly formed sac [26]. Etiologies of traumatic carotid pseudoaneurysm include blunt and penetrating mechanisms of injury as well as post-procedural (i.e., inadvertent line placement, post-endarterectomy). Traumatic pseudoaneurysms are associated with dissection in 13% to 49% of cases [27]. On conventional angiography, a pseudoaneurysm is demonstrated by contained extravasation of blood from the arterial lumen.

Symptoms common include headache and at times tinnitus when the lesion is near the skull base. At other times, they can present with neck swelling to local mass effects. Carotid pseudoaneurysms can compress adjacent nerves and local musculoskeletal structures, commonly affecting cranial nerves IX, X, and XI. Horner syndrome may also be observed from stretching of the cervical sympathetic chain [28]. Once again, observation is the mainstay of pseudoaneurysm treatment. Rupture of traumatic pseudoaneurysms is rare, and spontaneous regression at 12 months is common if the lesion is small [27]. At times, however, the lesion persists and can cause disabling head and/or tinnitus. In these instances, endovascular treatment with stenting, stent-assisted coiling, or flow-diverting devices can be considered [29, 30]. In cases with anatomy not suitable for endovascular repair, open surgical repair with interposition bypass grafting is an option.

Case Study

A 22-year-old man suffered extensive polytrauma after involvement in a head on motor vehicle collision. He was intubated by emergency medical services at the scene and upon arrival at the hospital was found to have C3 and C4 spinal fractures. He was taken emergently for operative repair of bladder and kidney lacerations. While undergoing a routine neurological check in the intensive care unit postoperatively, he was noted to have decreased movement on the left side of the body. CT angiography showed bilateral carotid dissection, and MR showed left hemisphere, low volume, watershed distribution infarcts. The risk for further bleeding was assessed to be low, and the patient was loaded with aspirin and clopidogrel. He underwent overlapping stent insertion to treat a left internal carotid artery dissection with associate narrowing of the distal cervical internal carotid artery and pseudoaneurysm of the mid-cervical internal carotid artery (Fig. 17.1a). A 6 F cook shuttle sheath was placed within the distal left carotid artery and a 5F Spider Rx filter was deployed distal to the site of stenosis. Over the filter wire, a 6 × 40 mm Xact stent was deployed with the distal end across the stenosis and the proximal end covering the entry zone of the cervical pseudoaneurysm. A second 6–8 × 30 mm Xact stent was deployed across the entry zone to "double cover" and promote flow diversion (Fig. 17.1b).

Carotid Cavernous Fistula (See Chap. 15)

Head trauma may rarely lead to traumatic injury to the intracranial carotid artery as it passes through the cavernous sinus. This dural sinus is the main venous drainage pathway for the eye. The carotid injury can lead to a high-flow/direct fistula through which arterial blood enters the cavernous sinus, leading to arterialized pressure and poor orbital drainage. The fistulous connection in such cases is most commonly located in the inferior aspect of the horizontal limb of the cavernous segment of the internal correct artery. Clinically, patients will rapidly develop ipsilateral or bilateral orbital chemosis, proptosis, and scleral edema. Ocular motility is frequently impaired. Intraocular pressure is elevated due to impaired venous drainage of the globe. If left untreated, permanent vision loss may occur. This makes the early recognition of high-flow carotid fistulas important for the trauma surgeon. Such lesions are amenable to endovascular treatment via both a transvenous and transarterial approach. See Chap. 15 for a more detailed description of these approaches [31].

Vertebral Artery Dissection

While less emphasized in the literature, vertebral artery dissection is also seen in the setting of blunt trauma to the head and neck [32, 33]. When these dissections occur, they are sometimes associated with cervical spine injuries, especially subluxations and fractures of the foramen transversarium. When not in the setting of boney injury, vertebral dissections most commonly occur at the skull base as the vertebral artery enters the dura.

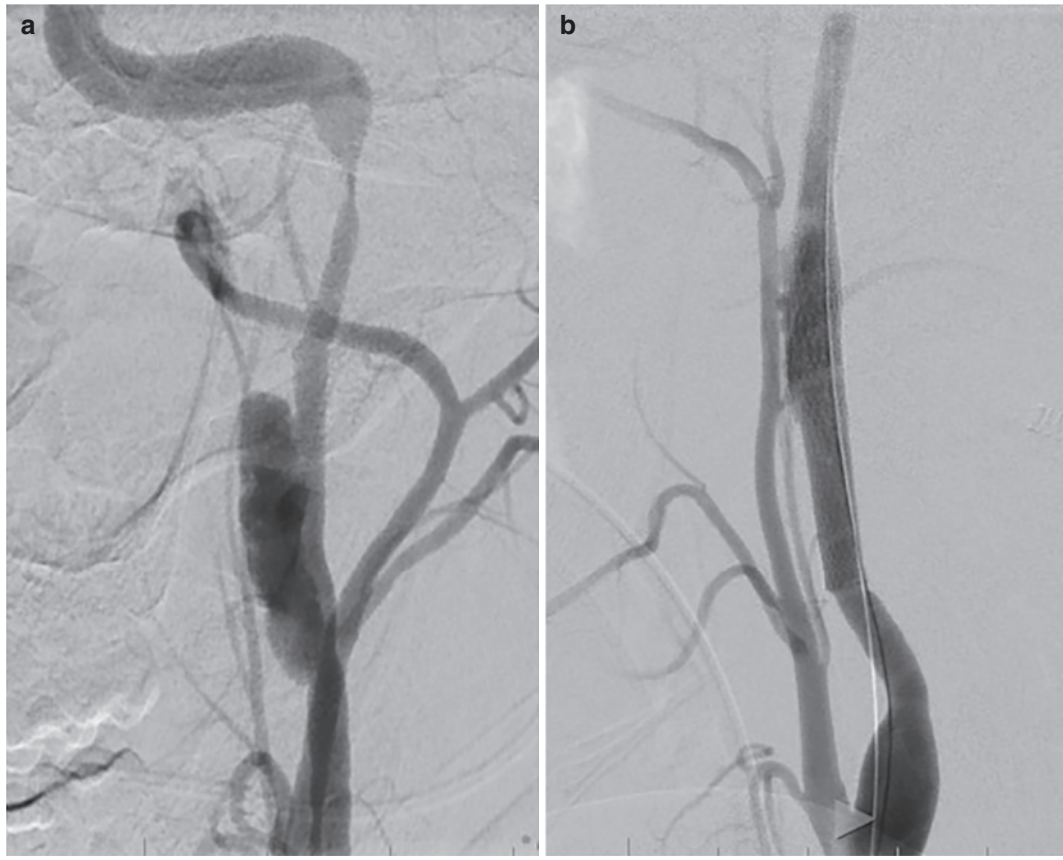

Fig. 17.1 Carotid dissection and psuedoaneurysm

Presenting symptoms often include neck pain that radiates to the skull base. Symptoms and signs of posterior circulation ischemia such as vertigo, nausea, diplopia, hemibody anesthesia or weakness, limb ataxia, dysarthria, and/or hemianopsia should also prompt consideration of this diagnosis [34].

Unlike carotid dissection, vertebral artery dissection that extends intracranially can also be associated with vessel rupture and subarachnoid hemorrhage, a potentially lethal complication.

Management of patients without signs of symptoms of TIA or stroke usually focuses on antiplatelet use and anticoagulation if there is associated thrombus. However, in patients with brainstem flow compromise and stroke symptoms, stenting or open surgical repair can be considered. In those with subarachnoid hemorrhage, vessel preserving techniques such as stent-assisted coiling or flow-diverting device insertion can be considered. As a last resort, vessel sacrifice may be needed [35].

Postoperative Large Vessel Stroke (See Chaps. 6 and 7)

While rare, artery-to-artery embolism occasionally occurs post-carotid stent insertion, carotid endarterectomy, or transcarotid artery revascularization. If the volume of embolic material is large, major stroke can result [36]. Mechanical thrombectomy may be a viable treatment modality in this setting, even following open carotid revascularization. If the initial procedure was done in a hybrid operating room and identified by intraoperative assessment via post repair angiogram done to assess the repair, subsequent neuro-

interventions may be expeditiously done in that same setting. Mechanical thrombectomy has been well studied and shows great potency in large vessel occlusive stroke with one in three patients treated returning to a nondisabled state [37]. Undergoing MT requires immediate recognition of the stroke syndrome followed by activation of the stroke team and CT angiography of the head and neck to identify the site of occlusion. If an occlusion of the intracranial carotid or middle cerebral artery is seen, mechanical clot retrieval via a suction thrombectomy catheter or retrievable stent is recommended. See Chap. 7 for procedural details.

Incidental Intracranial Abnormalities

Vascular disease is rarely confined to a single vascular bed. Therefore, it is common for vascular specialists to encounter "incidental" disease of the cervical and intracranial vessels in their patient populations.

Atherosclerotic Stenosis (See Chaps. 4 and 5)

The most common underlying form of vascular pathology throughout the body is atherosclerosis. Frequently, patients with atherosclerotic stenosis of the iliofemoral arteries, coronary arteries, or other systemic vascular beds will also have cervical or intracranial stenosis [38].

Extracranial carotid revascularization is of established utility in patients with symptomatic stenosis ≥50% in severity [39]. With evolving medical therapy, the treatment of asymptomatic severe disease has been brought into question but is still performed. Please refer to Chap. 4 for a more detailed discussion of these revascularization modalities.

A less commonly recognized site of extracranial atherosclerosis is the vertebral artery. These lesions most often affect the ostium of the vertebral artery but can also involve the more distal vertebral artery [40]. While there are few randomized clinical trials (RTC) of vertebral artery surgery, several small European studies have been published [41, 42]. Both studies showed excellent safety for extracranial vertebral artery stenting, with one (VAST) showing a potential efficacy signal for stenting despite its small size and premature termination.

Intracranial atherosclerosis (ICAD), unlike extracranial disease, is primarily managed with medication [43]. This is based on the results of the SAMMPRIS trial of stenting with the Wingspan intracranial stent system (Stryker Neurovascular) plus best medical therapy (BMT) versus BMT alone. In this trial, BMT was superior to stenting primarily due to the perioperative stroke risk associated with the procedure. In the best medical arm, patients were treated with aspirin and clopidogrel for 90 days along with general risk factor modification. This protocol has largely been adopted as the first-line therapy for patients with ICAD. Patients who fail BMT can be considered for stent revascularization, based on the excellent outcomes seen in the nonrandomized WEAVE trial [44].

Aneurysms (See Chap. 10)

There is growing recognition that the vascular weakening underlying intracranial aneurysms is related in some if not most cases to the risk factors that underlie atherosclerotic disease [45]. *When* encountered, such lesions warrant careful risk stratification based on aneurysm size and morphology along with patient age and comorbid conditions. Given the importance of morphological details, cerebral catheter angiography should be considered when the findings may sway decision-making and/or choice of surgical modality. Generally, intracranial aneurysms of the anterior circulation greater than or equal to 7 mm in size and those 5 mm or larger in the posterior circulation should be considered for surgery [46]. The majority of unruptured aneurysms in the United States and Europe are currently treated endovascularly with catheter-based platforms

such as detachable coils and flow-diverting devices. Occasionally, morphological features may make open surgical clipping the best option. See Chap. 10 for details of endovascular aneurysm repair.

Dural Arteriovenous Fistula (See Chaps. 13 and 15)

Arteriovenous fistulas (AVFs) are rare vascular malformations. Their basic angioarchitecture involves arteries directly filling a vein without an intervening capillary bed. The most common form of AVF is the dural AVF (dAVF), in which arteries normally supply the dura mater, instead directly empty into one of the dural sinuses. These lesions are of interest to the trauma surgeon because one speculated etiology is as sequelae of traumatic injury. Thus, dAVFs are sometimes discovered in the follow-up of the traumatic brain injury patient. Presenting symptoms include tinnitus, bruit, dizziness, headache, and progressive cognitive decline. Diagnosis starts with noninvasive vascular imaging such as MR angiography, but if the clinical suspicion is high, the workup is not complete without a catheter angiogram. The small caliber of the involved arteries means that the higher resolution of an invasive angiogram is often required [47]. See Chap. 13 for a complete discussion of the endovascular management of this disorder.

Role of Transcarotid Artery Revascularization (TCAR) with Flow Reversal (See Chap. 4)

Transcarotid artery revascularization is a minimally invasive surgical modality for treating carotid stenosis. It involves open exposure of the common carotid artery via supraclavicular incision along with the use of the ENROUTE transcarotid neuroprotection system (Silk Road Medical, Sunnyvale, CA, USA) [48]. This relatively new procedure may be of interest to neurointerventionalists and vascular surgeons faced with patients with high-risk features for both stenting and carotid endarterectomy.

See Chap. 4 for a discussion of high-risk features for carotid endarterectomy. Features that make transfemoral or transradial stenting high risk include severe aortic arch angulation, excessive redundancy and tortuosity of the common carotid artery, and/or internal carotid artery and heavily calcified composition of the stenotic lesion [49]. Although carotid endarterectomy and carotid stenting are the most frequently used procedures for carotid revascularization, TCAR has shown excellent early results in nonrandomized prospective cohort studies, particularly in high-risk patients [50].

Several multicenter studies compared TCAR to CEA and found that both modalities had a comparable pre-procedural stroke and all-cause mortality rates immediately following the procedure and 1 year later. However, TCAR was associated with a lower risk of cranial nerve injury. According to a recent meta-analysis, the risk of CN injury was less than 0.4 percent [48]; therefore, TCAR may be a viable option for patients at high risk for open surgery, including those with surgically inaccessible lesions, prior neck surgery, neck irradiation, laryngeal nerve palsy, and tandem lesions [51]. The TCAR procedure has a favorable safety profile in nonrandomized prospective registries, with a reported periprocedural stroke rate of 1.4 percent and a 1-year follow-up stroke rate of less than 0.6 [51]. However, randomized controlled trials comparing TCAR with CEA and/or CAS have not been published to date.

A significant constraint on the use of TCAR is the technical challenges it presents: it is a hybrid procedure requiring both open surgical and interventional management. The infrastructure and operator skill set are not available in all centers [52]. Additionally, to be eligible for TCAR, patients must have amenable anatomy. Patients must have an ICA > 4 mm, CCA free of plaque and >6 mm and a distance from the clavicle to carotid bifurcation of at least 5 cm [53].

Procedural Steps

TCAR can be performed under general or local anesthesia with intra-procedural heparin. A small supraclavicular incision is made to allow for dissection of the proximal common carotid artery. A micropuncture kit is used to cannulate the arterial wall followed by the advancement of a guidewire and arterial sheath without crossing the lesion. Venous access is then obtained by percutaneous puncture of the contralateral common femoral vein. Both the arterial and venous sheaths are connected to a "flow controller" to form a flow circuit that includes an embolic protection filter. The common carotid artery is occluded proximal to the arterial puncture site with a vascular clamp which initiates flow reversal is initiated. The goal of flow reversal is to pull all potential embolic debris away from the intracranial circulation and toward the filter before blood is returned to the venous system. A guidewire is used to cross the stenotic carotid segment under fluoroscopy. This is followed by stent deployment and balloon angioplasty in a similar sequence to traditional carotid stenting [48, 51].

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