Traumatic Intracranial and Extracranial Vascular Injuries in Children

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KEYWORDS

• Trauma • Carotid artery • Vertebral artery • Dissection

TRAUMATIC INJURIES OF THE CAROTID ARTERY

Extracranial Carotid Injuries

The incidence of carotid artery dissection (CAD) in adults after blunt head and neck injury is estimated at 0.3 to 0.67%.[1–5] In children, this injury seems to be significantly less common (estimated at 0.03%).[6] However, because CAD can be clinically silent, its frequency may be underestimated. The traumatic event is usually that of hyperextension/rotation injury or a direct blow to the neck.

Extracranial CAD represents the most common location of traumatic vascular dissections in the head and neck area and is followed in frequency by the extracranial vertebral artery.[7] Arterial dissection has been associated with several conditions including fibromuscular dysplasia, Marfan syndrome, cystic medial necrosis, oral contraceptives, and drug abuse.[8] In connective tissue disease there is a structural defect leading to weakness in the arterial wall and predisposing to dissection either spontaneously or after a minor trauma. In other cases, environmental factors such as drug abuse can cause endothelial damage predisposing to this condition. On the other hand, traumatic dissection is also known to occur in otherwise healthy patients with no known risk factors.

CAD can be asymptomatic, especially in patients younger than 18 years of age. The dissection may remain in a subadventitial rather than subintimal plane, which may account for a delay in presentation[9]; a subadventitial dissection is believed to result in pseudoaneurysm with a potential for delayed presentation caused by emboli, whereas a subintimal one can lead to significant narrowing of the lumen with a more imminent clinical presentation (**Fig. 1**). A review of the literature showed that in most cases the diagnosis was suspected and then confirmed only after a focal neurologic deficit consistent with a stroke or transient ischemic attack (TIA) in the presence of a history of trauma.[10] Consequently, a high index of suspicion based on the mechanism of injury or physical signs and symptoms is of paramount importance to diagnose these lesions before the occurrence of severe neurologic deficit.

One mechanism of injury that deserves a special emphasis in children is soft palate traumatic injury. Pens and sticks are the most frequent traumatic agents and the mean age of occurrence is 4 years.[11] The proposed pathophysiology is related to an indirect compression of the internal carotid artery (ICA) against the skull base or against the upper cervical transverse process.[12] Although CAD is rare after such an injury, it is potentially associated with high mortality and morbidity. The initial symptoms are usually mild, such as minor and transient oral bleeding, small pharyngeal wound, and tenderness at the angle of the mandible. The neurologic symptoms typically appear after a silent period that can last from a few hours to several days.[13]

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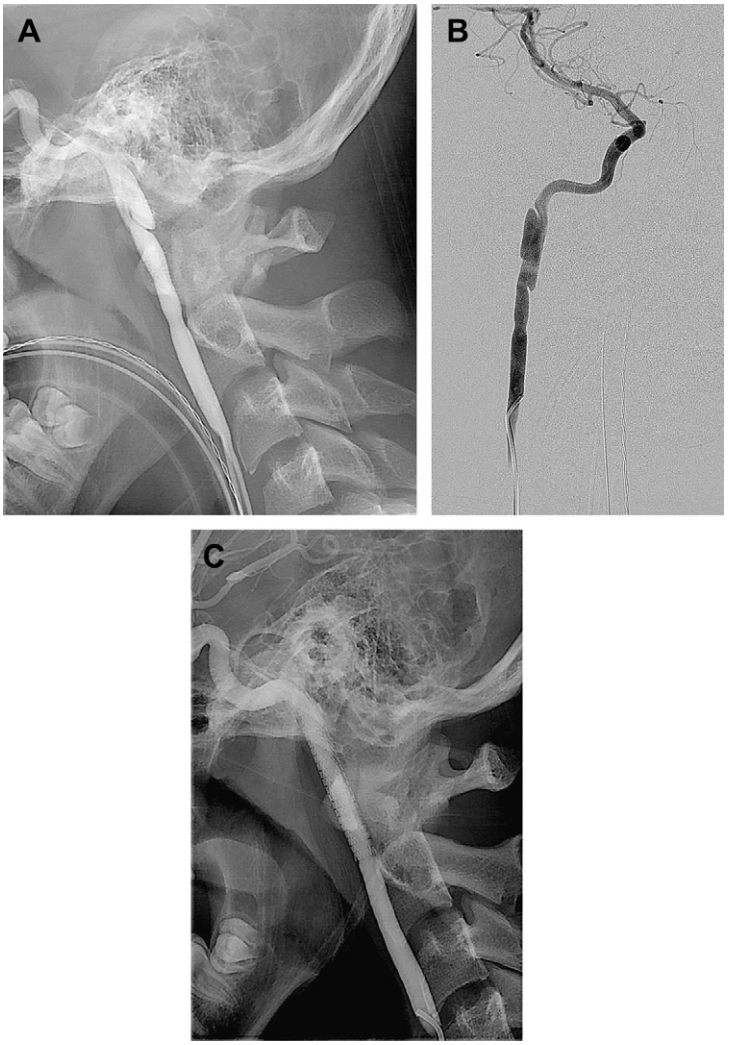

Fig. 1. 15-year-old boy with neck pain after football injury. (*A* and *B*) Lateral view of cerebral angiogram with (R) internal carotid injection shows a traumatic dissection with pseudoaneurysm of the extracranial ICA. (*C*) Lateral view of cerebral angiogram with (R) internal carotid injection at 6 months after endovascular treatment of the dissection using stent shows intimal healing and endothelialization.

Intracranial Carotid Injuries

Although intracranial location is rare among CAD in adults,[14,15] it seems to be common in children. In their review of the literature, Fullerton and colleagues[16] found that 60% of the reported cases of CAD were intracranial. A male predominance seems to be attributable to a higher incidence of trauma among young males. Similarly, Oka and colleagues[17] found that 25 of 45 patients who presented with intracranial carotid dissection were 18 years of age or younger. Although subarachnoid hemorrhage (SAH) is a real concern in these cases, strokes and TIAs remain the most common presenting feature.

Intracranial ICA dissection in children most commonly occurs spontaneously without any history of trauma. Among all reported cases of pediatric ICA dissection, the likelihood of intracranial dissection seems to be inversely proportional to the severity of trauma reported. Following severe trauma, 25% of the reported ICA dissections were intracranial, compared with 58% in the case of mild trauma, and 86% when no history of trauma is given.[16] This observation favors a traumatic cause in extracranial dissections and a spontaneous cause in the intracranial ones (possibly precipitated by a minor trauma). A predisposing risk factor (collagen vascular diseases, connective

tissue disorders, use of oral contraceptives, smoking, hypertension, and migraine) was reported in several cases; however, in most cases the cause remains unknown. Overall, the mortality seems to be significantly higher in intracranial dissections compared with extracranial ones.[16]

Traumatic intracranial aneurysms are rare, comprising less than 1% of intracranial aneurysms in most large series.[18,19] Histologically, they can be true aneurysms (disruption of intima and media, with an intact adventia) or false aneurysms (disruption of all 3 layers with formation of a contained hematoma). False aneurysms are considered the most common, although the relative incidence of these histologic types is unknown.[20] Traumatic intracranial aneurysms in children are best categorized by mechanism of injury and location (**Table 1**).[21] Traumatic aneurysms can be caused by penetrating or nonpenetrating trauma. Aneurysms secondary to nonpenetrating trauma can be divided further into skull base and peripheral lesions. Peripheral traumatic aneurysms can again be divided into aneurysms of the distal anterior cerebral artery (ACA) secondary to trauma against the falcine edge (**Fig. 2**), distal posterior cerebral artery secondary to trauma against the tentorial edge, and distal cortical artery aneurysms frequently associated with an overlying skull fracture (**Fig. 3**). At the base of the skull, traumatic aneurysms most commonly involve the petrous and cavernous carotid artery and are almost invariably associated with a skull base fracture.[22,23] Injury to the ICA at the skull base can cause immediate rupture, leading to a carotid-cavernous fistula or to massive epistaxis.[24] Maurer and colleagues[25] stated that the triad of unilateral blindness, basal skull fracture, and recurrent severe epistaxis is diagnostic of ICA injury at the skull base.

Traumatic carotid-cavernous fistula (TCCF) is another rare entity that can occur after head injury. The estimated incidence ranges between 0.1 and 1%.[26,27] In a recent study, a skull base fracture was documented in 67% of the cases, and among 312 patients with a fracture at the skull base, TCCF was found in 3.4%.[28] TCCF most commonly results from a direct connection between the carotid artery and the cavernous sinus, leading to high-flow fistula. These lesions are unlikely to regress spontaneously and require prompt diagnosis and management. Clinically, these patients most commonly present with exophthalmos, bruit, chemosis, decreased vision, and limited ocular movements.

Table 1
Classification of traumatic intracranial aneurysms

Penetrating trauma
Nonpenetrating trauma
Skull base
Peripheral
Distal ACA: parafalcine
Distal cortical artery

TRAUMATIC INJURIES OF THE VERTEBRAL ARTERY

Extracranial Vertebral Injuries

Traumatic extracranial vertebral artery injuries may include dissections, pseudoaneurysms, or arteriovenous fistulas. Trauma remains the most common cause of dissection of the extracranial vertebral artery.[16] Other causes include mainly vasculopathy and connective tissue disease. In some cases the dissection can be spontaneous, with no history of trauma or predisposing factors identified. In accordance with the adult literature, the most common segment involved is at the mobile C1-C2 level.[29,30] The predilection for injury of this segment of vertebral artery has been observed in traumatic as well as in spontaneous cases. In most reported cases, vertebral dissections are preceded by a mild head or neck trauma.[31,32] Typically, there is a history of neck hyperextension with torsion.[33] When the dissection involves a segment below C2, an alternative mechanism must be sought because rotation between adjacent lower cervical vertebrae is minimal (**Fig. 4**). Typically, more severe trauma with cervical spine fractures is found in these cases.

Arteriovenous fistulas involving the vertebral artery are rare lesions, defined by the presence of an abnormal shunt between the extracranial vertebral artery or 1 of its muscular or radicular branches and the adjacent perivertebral venous plexus.[34,35] Approximately one-third of arteriovenous fistulas are asymptomatic,[36] discovered incidentally after auscultation of a neck bruit. However, these lesions can have ischemic symptoms of vertigo, diplopia, and cephalgia secondary to arterial steal. The presence of myelopathy or cervical neuralgia is rare but can result after arterial blood reflux into spinal pial veins, causing venous hypertension (Foix-Alajouanine syndrome) or after root compression by engorged epidural veins.[37] The main goal of treatment is closure of the arteriovenous fistula or pseudoaneurysm with preservation of the parent artery, frequently attained through an endovascular approach.[36]

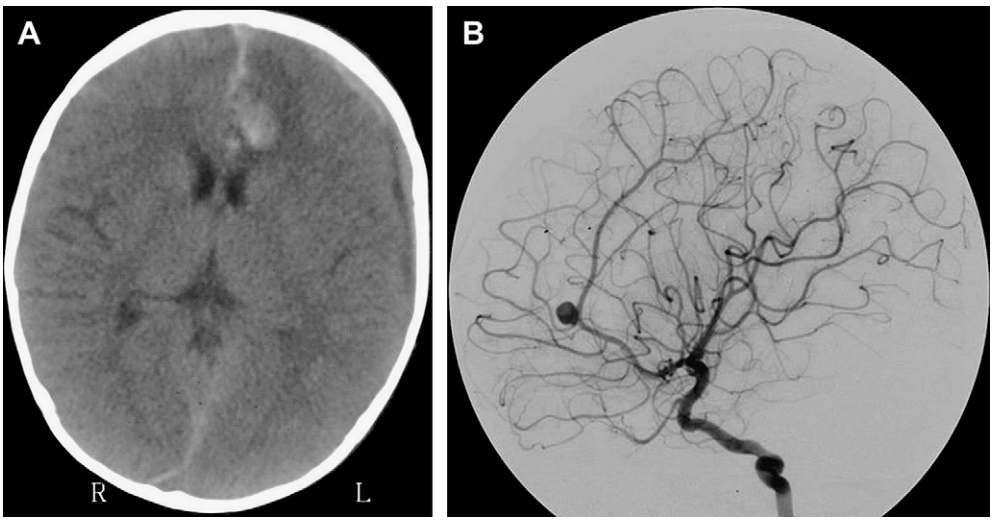

Fig. 2. A 7-year-old boy after a motor vehicle accident. (*A*) Axial CT scan of the brain without contrast shows evidence of interhemispheric subarachnoid and intraparenchymal hemorrhage. (*B*) Lateral view of cerebral angiogram with (L) internal carotid injection illustrates a traumatic distal ACA aneurysm.

The natural history of extracranial vertebral artery dissection in children remains poorly understood. Late complications in children include pseudoaneurysm formation, thrombosis, and recurrent stroke.[38] Stroke can result either from thrombosis leading to critical narrowing of the vessel or from emboli. The dynamic processes involved with vascular injury and healing may span years and result in variable outcomes. Because of the unpredictable evolution of these vascular changes, long-term clinical and radiologic follow-up are warranted.

Intracranial Vertebral Injuries

Intracranial dissection constitutes around 11% of reported vertebral artery injuries in children.[39] Unlike intracranial carotid dissections, trauma remains the most common cause in intracranial vertebral dissections.[16,40] Intracranial vertebral

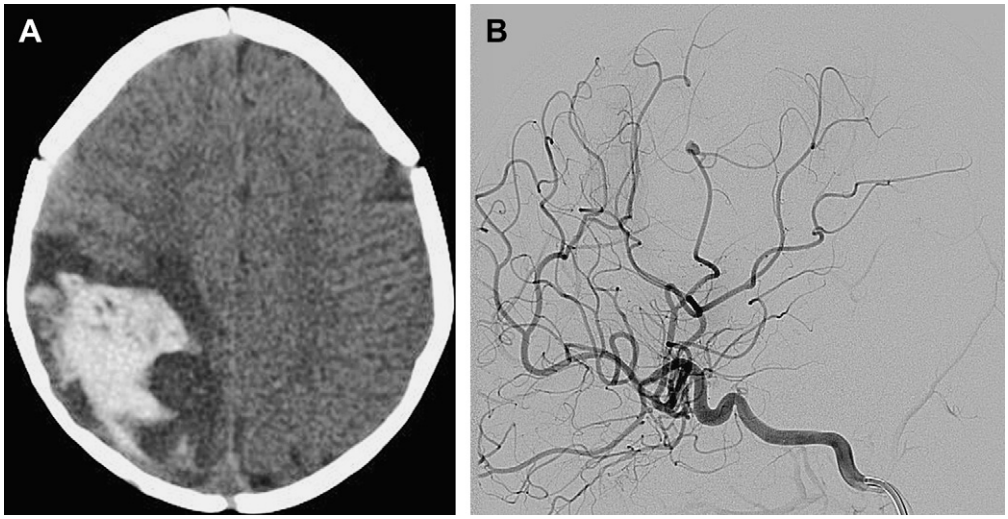

Fig. 3. A 3-month-old baby in a suspected case of nonaccidental trauma. (*A*) Axial CT of the brain without contrast shows large (R) intraparenchymal hemorrhage. (*B*) Lateral view of cerebral angiogram with (R) internal carotid injection illustrates a traumatic aneurysm of a distal cortical branch of the middle cerebral artery. Although distal cortical aneurysms are frequently associated with skull fractures, no fracture was identified in this case.

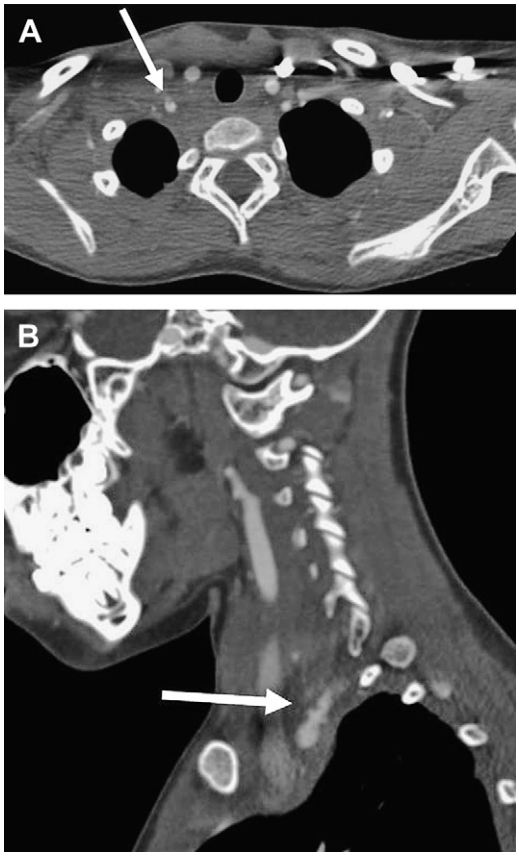

Fig. 4. A 9-year-old boy after whiplash injury from a motor vehicle accident. (*A*) Axial and (*B*) sagittal CTA shows a dissection and intimal flap (*arrow*) of the proximal part of the right extracranial vertebral artery.

artery dissections also differ from extracranial dissections, which are usually associated with strokes, as mentioned earlier.[41] Their prognosis is worse than extracranial dissections. Intracranial vertebral artery dissections are more susceptible to rupture than the extracranial segment, because the intracranial vertebral artery has thinner adventitia, and few elastic fibers in the media.[42] SAH is commonly reported in these cases with a high risk of rebleed (in up to 30%–70% of cases in some series), resulting in high mortality and morbidity.[43] Rare cases of nonaccidental trauma resulting in intracranial vertebral artery injury have been reported.[44] The presence of retinal hemorrhage in these cases should be interpreted with caution as it can be simply the consequence of SAH (Terson syndrome).

Patients with vertebral artery dissections usually have a lucid interval after trauma until they present with symptoms.[45,46] Ipsilateral headache, neck pain, dizziness, and neurologic deficits are the most common symptoms of vertebral artery dissections.[47] Patients who develop neurologic deficit may have speech deficits, dysphagia, and vision defects.

DIAGNOSTIC MODALITIES

Cerebral angiography remains the gold standard diagnostic modality (**Table 2**). It is currently the most accurate modality and provides fine detail of vascular anatomy and intimal injury near bony structures such as the skull base or the transverse foramen for the vertebral artery.[48] One of the major advantages of angiography is the ability to detect collateral circulation, which is critical when dealing with a dissected or occluded vessel. Furthermore, three-dimensional reconstructed images enable circumferential spatial evaluation of the vessel and estimation of flow compromise. However, because of its invasive nature and associated risk of iatrogenic injuries, it is advisable to reserve formal angiography for confirmation of findings detected on a screening diagnostic examination.

Magnetic resonance angiography (MRA) offers a high-resolution noninvasive approach for diagnosis and follow-up of traumatic vascular injuries. It is helpful in visualization of the arterial wall and detection of intramural hematoma.[49] However, the accuracy of MRA is limited in detecting small intimal injuries (<25% luminal stenosis) and early pseudoaneurysm formation.[50] MRA is less suited for acute unstable trauma patient, and because of logistical difficulties with access for critically ill patients. In a prospective comparative study by Biffl and colleagues[51] of trauma patients, the sensitivity and specificity of MRA for the diagnosis of cerebrovascular injuries were 75% and 67%, respectively. In a similar study by Miller and colleagues[52] the sensitivity of MRA was 50% in carotid injuries and 47% in vertebral injuries. However, the sensitivity of MR imaging/MRA is highest 2 days after dissections. The resolution of MRA now approaches that of conventional angiography. MR imaging can show not only vessel occlusion, but its effect on the brain. It is also noninvasive and should become the investigation of choice for patients in whom blunt cervical vascular trauma is clinically suspected.[53]

Because computed tomography (CT) is noninvasive and widely available, CT angiography (CTA) has been used for the screening and diagnosis of traumatic vascular injuries. Early studies using old generation scanners have been disappointing, suggesting a high rate of false-negative and false-positive results.[51,52,54] The main disadvantage of CTA is related to bony artifact limiting its ability to identify injuries in some areas such as carotid canal or transverse foramina. However, current generation 16-detector scanners are

Table 2
Diagnostic modalities in case of carotid and vertebral artery injury

	Doppler Ultrasound	CTA	MR Imaging/MRA	Angiography
Information				
Flow interruption	Very helpful	Very helpful	Very helpful	Sometimes helpful
Thrombus versus spasm	Very helpful	Very helpful	Very helpful	Very helpful
Thrombus extension	Useless	Very helpful	Very helpful	Very helpful
Permeability of circle of Willis	Sometimes helpful (transcranial Doppler)	Very helpful	Very helpful	Very helpful
Cerebral ischemia	Useless	Sometimes helpful	Very helpful	Useless
Advantages	Noninvasive, easy to obtain, and readily available	Rapid, high spatial resolution, usually easy to obtain	Noninvasive; no adverse effect with contrast	Most sensitive technique
Disadvantages	No visualization of intracranial vessels. Limited use for the vertebral	Risks resulting from intravenous injection of iodinated contrast; irradiation; sedation required in patients <2 years old	Sedation usually required in patients less than 5 years old; not easy to obtain; impossible in case of metallic foreign body	Invasive; may not differentiate between dissection and vasospasm; irradiation

Data from Pierrot S, Bernardeschi D, Morrisseau-Durand MP, et al. Dissection of the internal carotid artery following trauma of the soft palate in children. Ann Otol Rhinol Laryngol 2006;115:323–9.

capable of rendering high-resolution images along with high-speed data acquisition. In a recent large study, the accuracy of new generation CTA in diagnosing and excluding blunt carotid or vertebral artery injuries was evaluated by comparing it with angiography. Dissections as well as pseudoaneurysms were included in this study. The overall sensitivity, specificity, and positive and negative predictive values of CTA were 74%, 86%, 65%, and 90%, respectively; no significant difference was found between carotid and vertebral artery injuries.[55]

Doppler ultrasonography is able to provide high-resolution real-time images of the carotid artery bifurcation and proximal ICA. It has proved reliable in evaluating the presence and severity of atherosclerotic disease. Ultrasonography is also a noninvasive and widely available test. However, its role in the diagnosis of traumatic vascular injuries is hampered by several limitations. For obvious reasons related to surrounding bony structures, this modality is unable to assess intracranial injuries or high cervical vascular lesions close to the skull base. Furthermore, most of the extracranial vertebral artery cannot be assessed for the same reason. Small series reporting the use of Doppler ultrasonography have been published.[56–58] The diagnostic accuracy for identification of a vascular injury was found to be around 86% for the cervical carotid[59] and 79% for vertebral artery.[60] This modality is suboptimal for the screening and diagnosis of traumatic vascular injuries, but may have a role in the follow-up of known traumatic cervical lesions of carotid and vertebral vessels.

MANAGEMENT OPTIONS

Choosing a treatment option represents a significant challenge for the clinician in traumatic cerebrovascular injuries in children because of the

lack of high-quality clinical data comparing 1 modality with another, and the absence of a clear consensus. As a result, decisions are largely based on individualized, single-center experience supported by data from case reports and small case series. The individual application of these modalities may also be extrapolated from adult experience, but the unique features of children need to be recalled (eg, the safety profile of full anticoagulation in active toddlers).

In general, penetrating trauma is more likely to require surgical repair for control of bleeding than is the case with blunt injury, although endovascular occlusion may be used. Blunt trauma is most often managed by medical therapy or endovascular approaches.[61]

Extracranial Carotid Artery Injury

Medical therapy is based on the premise that most neurologic events are related to thrombus within the lumen and are potentially preventable with anticoagulation or antiplatelet drugs (**Fig. 5**).[62–64] Imaging studies suggest that more than 90% of infarcts caused by dissection are thrombotic rather than hemodynamic in origin.[62,63] Transcranial Doppler studies show a high frequency of intracranial microemboli.[64]

Antithrombotic therapy has been advocated since the 1970s.[65] However, there were no randomized trials to assess the effects of antithrombotic therapy or surgical treatment. As no reliable data from randomized trials were available, it is not possible to draw any definite conclusions.[66,67] Reported nonrandomized studies have, likewise, not shown evidence of a significant difference between anticoagulant and antiplatelet agents.

Based on some open-label studies and anecdotal experience, several investigators favor anticoagulation with heparin followed by coumadin therapy as a reasonable approach in symptomatic CADs.[68,69] Treatment with coumadin for 6 months

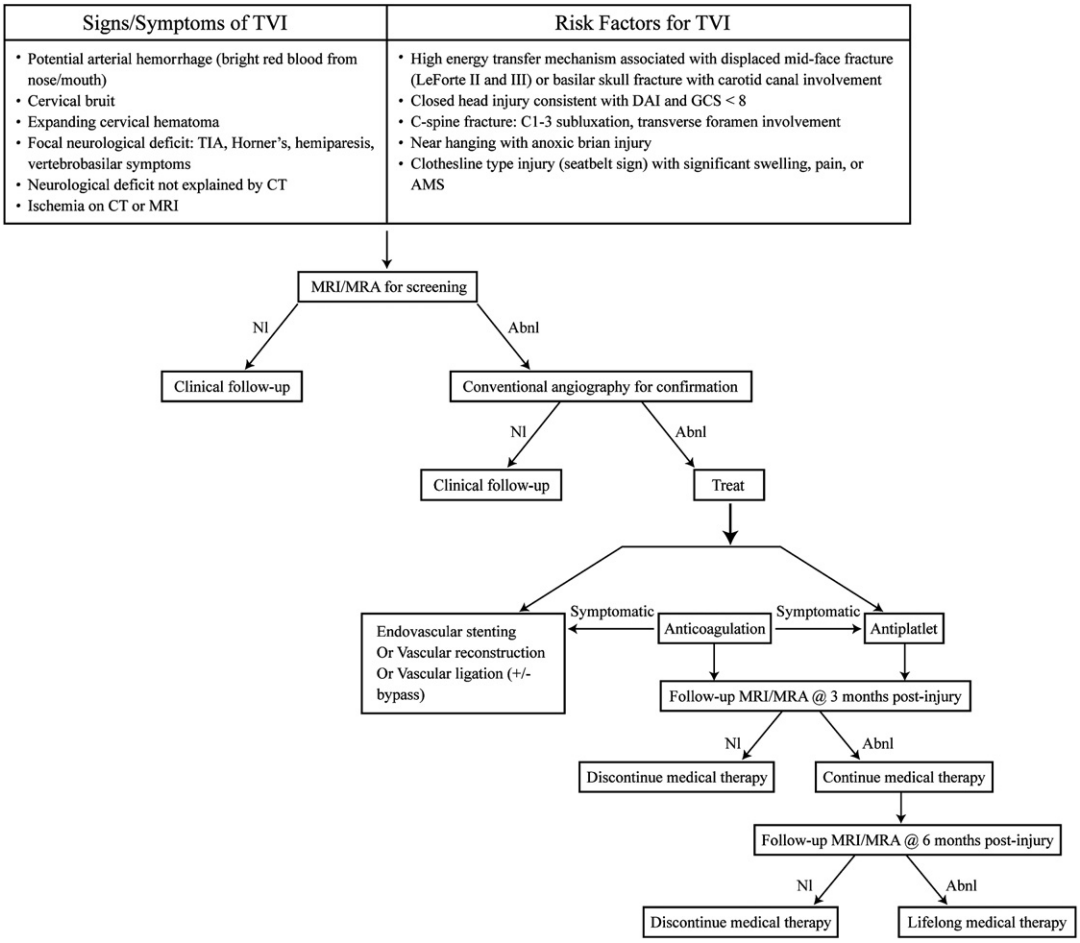

Fig. 5. An approach to the diagnosis and treatment of extracranial carotid and vertebral artery injuries. AMS, altered mental status; TVI, traumatic vascular injuries.

to 1 year has traditionally been advocated with a target international normalized ratio of 2 to 3.[63,70] In a study of the treatment of a blunt carotid artery injury, anticoagulation was suggested to be the treatment of choice when dissection or pseudoaneurym was diagnosed.[71] There are no data from controlled studies on the safety of heparin, although several consecutive series showed no significant side effects.[72,73]

Caution should be exercised when anticoagulant agents are used in patients who have concurrent intracranial dissections, because of the risk of SAH and a worsening of deficits after early anticoagulation.[74] The fear that anticoagulation or tissue-plasminogen activator therapy will extend the dissection seems to be unfounded.[14,75]

Antiplatelet agents have been used in the management of dissection, but there is less information about their efficacy.[76] If anticoagulation is contraindicated, many investigators recommend antiplatelet therapy.[63]

In studies comparing antiplatelet and anticoagulation treatment, there were no statistically significant differences in primary outcome measures. In a 1-year follow-up study, patients who were treated with aspirin had higher event rates (TIAs, stroke, or death) compared with patients treated with anticoagulants (12.4% compared with 8.3%)[66]; however, this was statistically not significant. Therefore, it cannot be concluded that antiplatelet therapy is less effective than anticoagulation therapy in preventing stroke occurrences and recurrence in patients with extracranial CAD.[67]

Antithrombotic therapy is recommended as initial medical treatment in extracranial carotid dissection. Antiplatelet therapy with its safer pharmacologic profile for children in terms of administration, maintenance, and complications may be favored over anticoagulation (see **Fig. 5**).[10]

Surgical intervention for carotid dissection is reserved for patients with recurrent TIAs or progressive neurologic deficits secondary to hypoperfusion or embolic phenomenon despite maximal medical therapy. Eligibility for surgery is determined by the patient's clinical status as well as the duration of the occlusion as suggested by imaging criteria. In general, patients are excluded who have severe fixed deficits after a completed stroke or TIA for an extended period with a chronic occlusion. Those patients with recent onset of multiple TIAs, transient monocular blindness, and acute occlusion may benefit from endarterectomy and thrombectomy or carotid ligation.[77]

One review indicated that the presence of a large or expanding pseudoaneurysm is also an indication for surgical intervention.[78] Chronic carotid dissections have also been treated with surgical reconstruction to prevent further ischemic or thromboembolic complications, if medical treatment with 6-month anticoagulation fails or if carotid aneurysms and/or high-grade stenosis persist.[79]

In most cases, endovascular treatment has supplanted open surgery as the initial treatment of choice once medical therapy fails in adults.[80,81] Endovascular stenting for carotid atherosclerotic disease was not in use before 1989; studies published before 1989 may not have reflected endovascular stenting as an available treatment option for carotid dissection. The decreased tortuosity of pediatric vessels makes stent placement feasible in the extracranial carotid artery.[82] Nonetheless, the long-term results and effects of carotid stenting in children are unknown (eg, post-stenting restenosis), and the treatment of stent-related complications can be complex.[83]

Extracranial Vertebral Artery Injury

The natural history of vertebral artery dissection is unknown (see **Fig. 5**). It can heal spontaneously, develop occlusion, or form a pseudoaneurysm. Like extracranial carotid dissection, treatment of extracranial vertebral artery dissection is controversial; it is not clear whether patients must be heparinized, be treated with antiplatelets, or treated at all. Hasan and colleagues[39] in their review of 68 children found that the most common treatment of extracranial vertebral artery dissection was antiplatelet therapy. These investigators found that asymptomatic recovery occurred in 12 of 15 (80%) children who received antiplatelet therapy compared with 4 of 15 (27%) patients who received anticoagulation therapy with or without antiplatelet therapy. Once thrombus occurs, it is also controversial whether anticoagulation or antiplatelet therapy should be the treatment of choice. Beletsky and colleagues[66] showed that the recurrence rate for embolization is decreased significantly in patients on anticoagulation compared with those on antiplatelets (8.3% vs 12.4%). The difference in outcome at 1 year was not significantly different, however. It is therefore prudent to consider prophylactic treatment with antithrombotic therapy (unless contraindicated) because the consequence of brainstem ischemia is so poor.

Attempts at primary repair of an injured extracranial vertebral artery are rarely successful. Because of its location deep within the posterior triangle of the neck, the vertebral artery is difficult to approach surgically. Osteotomy of the middle portion of the transverse process may be required. Furthermore, craniotomy may be necessary for exposure of the distal portion.[84]

Open surgical ligation or endovascular occlusion (via balloon occlusion or coil embolization) of an injured artery with progressive dissection, pseudoaneurysm, arteriovenous fistula, or thromboembolic events despite antithrombotic therapy is frequently the procedure of choice. Only 10% of cerebral blood flow is from the vertebrobasilar system. A hypoplastic left vertebral artery occurs in 3.1% of individuals; on the right, 1.8% are hypoplastic. Thus, the risk of neurologic deficit from unilateral ligation is small.[85] Controversy persists in the literature as to whether proximal ligation alone is adequate; some believe that proximal and distal ligation must be done to avert distal thrombus propagation. Most agree that the risk of this complication is reduced by prophylactic heparinization in the acute phase for any patient who has undergone vertebral artery ligation.[86]

The surgical and endovascular procedures described earlier involve the occlusion of the arterial lumen, which is the main disadvantage of these methods. For the patients whose contralateral vertebral artery is hypoplastic, it is often impossible to perform an arterial occlusion because of lack of an adequate intracranial collateral circulation and consequent ischemia.[87] In these cases, the use of a stent graft placed by endovascular means may halt progressive dissection, seal a pseudoaneurysm, or occlude an arteriovenous fistula, yet preserving the parent artery.[88]

In the very young pediatric population, the femoral artery can accommodate only a 4-French catheter system. Placement of a stent, which typically requires larger guide catheters, may not be an option in this population. Moreover, stent placement is associated with a risk of thrombosis that increases significantly with decreasing arterial diameter; for this reason, stents are rarely, if ever, placed when the patient is younger than 1 year of age.[40]

Intracranial Carotid Artery Injury

Dissecting aneurysm

The goal of treatment is to exclude an intracranial dissecting aneurysm from the circulation by surgical or endovascular methods. In 1975, Fleischer and colleagues[89] reported 41% mortality in patients treated conservatively compared with 18% mortality in surgically treated patients. Other investigators also described poor outcome in conservatively treated pediatric patients.[21] Given its poor natural history, aggressive surgical management with clipping, resection, or trapping of intracranial dissecting aneurysms of the carotid artery and its branches seems the most appropriate treatment.[21,90–94]

Endovascular techniques such as trapping, in which detachable balloons or embolization with detachable coils are used, have also been performed in the treatment of traumatic aneurysms.[21,95] The advantages of surgical clipping include the ability to isolate the aneurysm with the opportunity to reconstruct the parent artery, and allowing the opportunity to evacuate the associated intracranial hematoma.[20] On the other hand, endovascular therapy avoids prolonged anesthesia, eliminates retraction of an inflamed and irritated brain, and allows diagnostic angiography to be performed throughout the case.[96]

Carotid-cavernous fistula

Rupture of a large or giant aneurysm of the intracavernous carotid artery can result in sudden and massive epistaxis, requiring emergent packing of the nose as a life-saving measure. Parkinson first proposed a direct surgical repair of intracavernous aneurysms with preservation of the carotid artery by a transcavernous approach, but the morbidity was high from multiple cranial nerve dysfunction, and it subsequently fell out of favor.[97]

Contemporary management of carotid-cavernous fistula is nearly always accomplished by endovascular techniques.[98–100] An endovascular approach to these lesions is attractive in that the procedure can usually be performed using local anesthesia in older children and adolescents. Test occlusion and antiaggregant medication for at least 3 months are recommended to avoid secondary thromboembolic complications.[101] Balloon test occlusion, however, is often not technically feasible in children. Few data are available in children regarding the correlation of collateral artery caliber and the tolerance to cerebral circulation occlusion.[40]

Occlusion

Management of traumatic intracranial carotid artery occlusion is problematic, with death or severe neurologic deficit resulting in up to 85% of patients.[101] Steroids, revascularization, and anticoagulation have all been tried with limited success. Control of intracranial pressure (ICP) is essential in patients in whom distal ischemia and infarction can be expected to incite cerebral swelling, but maintenance of blood pressure and even hypervolemia are most effective in limiting the neurologic deficit and extent of infarction from thrombotic vascular occlusion.[101]

Vasospasm

Although rarely symptomatic in children, treatment of traumatic vasospasm when it does become symptomatic remains problematic. Blood pressure support with the goal of normotension, establishment of normovolemia, and surveillance with

transcranial Doppler ultrasound may be reasonable first steps in treating symptomatic traumatic vasospasm. The standard prophylactic course of artificially maintained hypertension, hypervolemia, and hemodilution used in vasospasm following aneurysm rupture and SAH may be counterproductive in patients whose injury is often complicated by cerebral edema, increased ICP, and tenuous tissue perfusion. Calcium channel blockade with calcium antagonists may be a more benign regimen, and good results have been reported in vasospasm associated with aneurysm rupture.[102] However, calcium channel blockers have been shown to interfere with autoregulation, to undermine the integrity of the blood-brain barrier, and to increase the sodium content of cerebral edema fluid. Their indiscriminant use in the context of increased ICP from trauma or radiographic signs of brain swelling is, therefore, dangerous and not recommended until further information is available for children.[103]

Angioplasty has been proposed by Higashida and colleagues[104] for management of cerebral vasospasm secondary to aneurysmal bleeding. However, this technique has not been used in post-traumatic vasospasm in children, and its safety and efficacy in this background remain to be proved.[104] Endovascular infusion of vasodilatory medications such as papaverine, or of calcium channel blockers such as verapamil, is also used in the treatment of vasospasm in adults. The safety and efficacy of this treatment in children is unknown.

Intracranial Vertebral Artery Injury

Traumatic dissections isolated to the intracranial vertebral artery approaching the vertebrobasilar junction are rare. At the level of the pontomedullary sulcus, the vertebrobasilar junction is tethered by thick trabeculae, the medial pontomedullary membrane, separating the premedullary and prepontine cisterns. The tethered vertebrobasilar junction may therefore represent another intracranial site prone to motion-induced arterial injury.

For intracranial dissections presenting with ischemia, spontaneous healing is regularly observed, and therefore anticoagulation or antiplatelet therapy with close follow-up suffice in most cases. On the other hand, dissecting aneurysms presenting with SAH portend a high risk of rehemorrhage (30%–70% in adults and unknown in children[88,105]).

The most definitive treatment of arterial dissection involves excluding the aneurysm and the injured segment of the parent vessel from the circulation, using either surgical or endovascular trapping procedures. When only the aneurysm sac is obliterated, the associated injured parent vessel segment remains vulnerable to rerupture, particularly in the acute phase. Combined occlusion of the aneurysm and the parent vessel is not feasible in every case, and the treatment plan must be formulated case by case.[40]

Introduction of a microcatheter and placement of coils into the friable aneurysm sac may be associated with a high risk of intraprocedural perforation. In addition, packing coils against the pseudoaneurysm wall, composed largely of fibrin, thrombus, and collagen, may be associated with a high risk of delayed recanalization.[40]

SUMMARY

Traumatic vascular injuries are uncommon in children. The clinical experience with this entity is limited and there is a lack of clear recommendations regarding diagnosis and treatment practices. Because of the rarity of this condition in children, it is unlikely that recommendations based on high-level evidence will be available soon. With these limitations in mind, several points can be concluded:

1. These injuries are frequently missed, and a high index of suspicion needs to be maintained for diagnosis before the occurrence of severe neurologic deficits
2. The natural history of these injuries is unique and stratified on the type of vessel injured (carotid vs vertebral) and location of injury (extracranial vs intracranial)
3. Noninvasive screening with MRA or CTA for cases of high suspicion, reserving catheter angiography for definitive diagnosis
4. Use of antiplatelet therapy rather than anticoagulation in children as first-line medical therapy in asymptomatic extracranial vascular injury without pseudoaneurysm formation, because of lack of evidence in favor of the latter, and the safer pharmacologic profile of the former
5. Endovascular treatment seems safe and efficacious and preferred to open surgery in failed medical treatment of extracranial vascular injuries
6. Surgical and/or endovascular approaches represent primary treatment rather than medical therapy for intracranial vascular injuries.

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