Vessel Injuries

28

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Recommendations

Level I

There are insufficient data to support a Level I recommendation for this topic.

Level II

There are insufficient data to support a Level II recommendation for this topic.

Level III

The radiological investigation of choice is CT angiography. The goal of therapy is to prevent further neurological injury by maintaining sufficient cerebral blood flow and preventing embolic incidents.

28.1 Overview

Vessel injuries are uncommon but potentially devastating to the patient. Both blunt and penetrating trauma to the head can result in vessel injury. We will here divide vessel injuries into extracranial and intracranial vessel injuries.

They are both difficult to diagnose without special contrast-enhanced radiological investigations, and a high index of suspicion must be entertained (Vertinsky et al. 2008). The vessel pathology itself can be divided into:

- Dissection—a tear in the intima leaves a highly thrombogenic intimal flap in the vessel lumen and a haematoma can develop in the vessel wall. Hence, blood flow can be impeded by both thrombosis and mural haematoma. Distal blood flow can be influenced due to embolic phenomena from the local thrombosis.
- True aneurysms—focal outpouching from an injured vessel where at least one layer of the vessel wall is intact.
- Pseudoaneurysms—focal outpouching from an injured vessel where all vessel wall layers are compromised, leaving just an organized haematoma as the barrier to further bleeding.

Both true aneurysms and pseudoaneurysms have turbulent blood flow and can serve as a nidus for thrombosis and subsequent embolization.

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Tips, Tricks and Pitfalls

- Maintain a high index of suspicion for vessel injury when there is:
  - Discrepancy between plain CT findings and the clinical state of the patient
  - Subarachnoid haemorrhage in the interhemispheric fissure
  - Fractures in close proximity to vessels

28.2 Background

28.2.1 Extracranial Vessel Injury

The incidence of blunt carotid or vertebral injury (BCVI) in hospitalized trauma patients is 1–2% (Weber et al. 2018; Esnault et al. 2017). In patients with severe head injury (initial GCS < 9), the incidence may be as high as 9% and BCVI is also clearly associated with major facial injury, as well as cervical spine fractures. The usual mechanism of injury is dissection with formation of a thrombus, wall hematoma or pseudoaneurysm. These processes may narrow the vessel lumen or shed an embolus to a more peripheral brain artery and result in a stroke.

The true risk of a stroke in patients with extracranial vessel injury is unknown, but the literature indicates around 11% with a mortality rate of approximately 26% (Weber et al. 2018). The time interval from injury to onset of cerebral ischemia itself is difficult to pinpoint, but from 1 to 72 h has been documented. Penetrating injuries more often transect the vessel or parts of the vessel and thereby expose the patient to compromised blood flow and significant blood loss. Explosions and ballistic injuries can cause dissections due to the shock wave resulting in an intima tear.

Several screening criteria have been developed for the detection of BCVI of which we recommend the expanded Denver criteria (Table 28.1) (Geddes et al. 2016). The most important risk factor for BCVI is a high-energy trauma mechanism in combination with severe TBI (GCS < 9), cervical spine fracture, severe facial injury (Le Fort 2 and 3) or skull base fractures.

Table 28.1 The expanded Denver screening criteria for BCVI

Signs/symptoms of BCVI
Arterial haemorrhage from the neck/nose/mouth
Cervical bruit in patients <50 years
Expanding cervical haematoma
Focal neurological deficit
Neurological exam incongruous with head CT findings
Stroke on secondary CT scan
Risk factors for BVCI High-energy transfer mechanism with:
Le Fort II or III
Mandible fracture
Complex skull fracture/basilar skull fracture/occipital condyle fracture
Severe traumatic brain injury (TBI) with GCS < 6
Cervical spine fracture, subluxation or ligamentous injury at any level
Near-hanging with anoxic brain injury
Seat belt abrasion with significant swelling, pain or altered mental status
TBI with thoracic injury
Scalp degloving
Thoracic vascular injury
Blunt cardiac rupture
Upper rib fracture

CT angiography is indicated if one or more of the criteria are present

The radiological investigation of choice is CT angiography (CTA) (Roberts et al. 2013). It is readily available, fast and easily performed in a trauma setting. Usually, this is sufficient to exclude extracranial vessel injury, but MRI can provide further confirmation and also information on possible infarction.

The goal of therapy is to prevent further neurological injury by maintaining sufficient cerebral blood flow and preventing embolic incidents. Therapeutic options are anticoagulation/platelet inhibition or endovascular intervention. Both heparin/low-molecular-weight heparin (LMWH) and platelet inhibitors are options for preventing further thrombosis and embolic events. There is insufficient documentation to recommend one over the other, but a reasonable algorithm is to start with medical therapy such as LMWH with transition to acetylsalicylic acid (Harrigan et al.

2013; Brommeland et al. 2018). Endovascular intervention is usually reserved for cases with embolic events despite medical therapy. The natural history of these lesions shows a strong tendency towards spontaneous healing within the first couple of months. Treatment length for traumatic dissections has not been determined, but 3 months may be sufficient. A control CTA should be performed before stopping medical therapy.

Less often, patients present with an extracranial carotid or vertebral aneurysm. They can demonstrate symptoms secondary to embolization, rupture or local volume effect. Therapy is controversial; their natural history shows a clear tendency towards regression, but anticoagulation is often indicated to prevent embolization. However, they should be subject to repeated radiological investigations. Enlargement, embolization and severe compressive symptoms should mandate consultation with expertise proficient in either flow-sparing therapy (suturing the aneurysm sac, resection and anastomosis or endovascular stent therapy) or flow-stopping therapy (balloon occlusion test with subsequent ligation/coiling or bypass).

28.2.2 Intracranial Vessel Injury

These are divided into arteriovenous fistulas (AVF), traumatic aneurysms, thrombosis and sinus injuries.

28.2.2.1 Arteriovenous Fistulas

The most common traumatic AVF is the carotid-cavernous fistula. Symptoms are related to arterialization, volume and increased venous pressure and include headache, chemosis, ptosis, ophthalmoplegia and visual loss. CTA can give hints to the presence of an AVF (arterialized cavernous sinus and increased size of ophtalmic vein), but digital subtraction angiography (DSA) is mandatory to further elucidate the flow pattern. If it is a direct carotid-cavernous fistula (direct connection between ICA and the cavernous sinus), occlusion of the fistula is warranted to prevent neurological deterioration; however, if it is an indirect fistula (a fistula within the leaves of the cavernous sinus that are fed by intracavernous branches of the internal and/or external carotid artery), spontaneous thrombosis of the fistula can occur and conservative treatment may be sufficient. Usually, endovascular embolization of the fistula is the method of choice in treating both direct and indirect carotid cavernous fistulas.

28.2.2.2 Traumatic Aneurysms

These are rare, comprising less than 1% of all intracranial aneurysms (Semple 2004). Compared with 'spontaneous' aneurysms, they have a larger propensity for bleeding: 50% rupture within the first week. This can be explained by the fact that these aneurysms are pseudoaneurysms. They also have a clear predilection for more distal localizations than spontaneous aneurysms, especially the A3 + 4 segments of the anterior cerebral arteries. They also show a clear tendency for rapid growth, and regular controls are warranted if indications for surgery have not already been made. A high index of suspicion must be maintained in patients admitted with penetrating injuries, where the offending object has been in close vicinity of the vessel; in patients with localized subarachnoid clots; in patients with large bleeds in the basal cisterns; in patients with fractures of the clivus, sphenoid sinus or medial temporal bone; and in patients exposed to shock waves. Data on the sensitivity of CTA compared with DSA is lacking with regards to traumatic aneurysms. However, if there are any irregularities on the CTA, we recommend DSA, as it better reveals the hallmarks of traumatic aneurysms: delayed emptying and filling of the aneurysm, irregular contours and absence of a neck. Obliteration of these aneurysms is usually recommended.

The method of choice has traditionally been open surgery due to the lack of a neck and the poor strength of the aneurysm wall making traumatic aneurysms poor candidates for endovascular treatment. However, there are now several reports claiming good results using combined endovascular stentassisted coiling (Cohen et al. 2008). During surgery, these aneurysms have been notorious for their intraoperative tendency for rupture and difficult clip reconstruction, necessitating wrapping, ligation or trapping with or without bypass (Semple 2004).

28.2.2.3 Traumatic Occlusion

The most common intracranial vessel to be occluded is the proximal intracranial ICA where the vessel is in intimate contact with bone. Fractures in the vicinity of the ICA should arouse concern and mandate an angiographic examination. CTA is usually sufficient, but if there are any irregularities, we advocate DSA. The occlusion is usually secondary to dissection with subsequent mural haematoma and thrombosis. Rates of 70–85% suffering from massive hemispheric infarction have been reported. However, the patency of the circle of Willis clearly plays a role, and previous reports are hampered by obvious selection bias. We advocate anticoagulation and perfusion studies to elucidate whether the cross-flow is adequate. In case of perfusion asymmetry, we recommend increasing the blood pressure and preferably utilize some form of metabolic monitoring in the affected hemisphere. There are also case reports on vascular augmentation procedures. In case the patient has already sustained a massive infarction, we generally discourage decompressive hemicraniectomy.

28.2.2.4 Dural Sinus Injury

Fractures extending across major dural sinuses can potentially tear the vessel wall and produce an extracerebral haemorrhage, usually an epidural haematoma. If an indication for evacuation is found, one should make arrangements for a potentially large haemorrhage when the bone flap is raised. Usually, the sinus tear is found and can be plugged with a finger while contemplating the next move. Depending on the size of the tear, a small piece of Tachosil® or similar material and slight pressure might be sufficient, but if large parts of the sinus are torn, reconstruction is recommended. Temporary occlusion using aneurysm clips to visualize the rent while reconstructing the vessel using sutures and an overlay of dura can be done, but depending on the sinus involved, occlusion can increase the pressure with subsequent herniation. A few case reports indicate that if the patient does not tolerate temporary sinus occlusion, a Fogarty® catheter can be inserted and used as a bypass vehicle while the sinus is reconstructed.

28.3 Specific Paediatric Concerns

There are no specific paediatric concerns for this subject.

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