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Neurovascular injuries in trauma: An under recognized entity

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ABSTRACT

Blunt traumatic cerebrovascular injury (TCVI) most often go unnoticed or they get noticed only when the associated complications surface. Timely detection of blunt cerebrovascular injury significantly improves the final outcome of the patient. TCVIs can result from extreme hyperextension/rotation, direct vascular blow, intraoral trauma, or direct laceration from bony fracture fragments. The strongest predictor of a carotid artery injury is a closed head injury (Glasgow Coma Scale score </=6) while the strongest predictor of a vertebral artery injury is a cervical spine injury. CT Angiography is a useful screening tool for TCVI but Digital subtraction angiography (DSA) remains the gold standard. Endovascular treatment of TCVI is safer than surgery although the indications for treatment should be individualized.

Traumatic vascular injuries to head and neck can be broadly classified into two categories

1. Penetrating traumatic cerebrovascular injury.
2. Blunt traumatic cerebrovascular injury.

Penetrating cerebrovascular injury is obvious and is most often managed surgically. Blunt traumatic cerebrovascular injury is covert and most often goes unnoticed or they get noticed when the effects of it appear after a few years. Timely detection of blunt cerebrovascular injury is important to improve the disease outcome of the patient.

Blunt traumatic cerebrovascular injury (TCVI) is defined as an extracranial or intracranial cerebrovascular structural defect that is directly attributable to a known high-energy non-penetrating injury. TCVIs are distinguished from spontaneous cerebrovascular dissections, which may also be associated with trivial trauma, such as coughing, by the nature of the inciting injury.

1. Extracranial blunt TCVI

1.1. *Epidemiology*

TCVI occurs in approximately 1% of all blunt force trauma patients.[1] If low-risk patients are excluded (i.e., patients with <24-h hospital stay who may have less significant injuries and therefore lower level of impact during their trauma), the incidence of TCVI increases to 2%–3% of all blunt force trauma patients.

Incidence: Blunt cervical carotid artery injury constitutes 0.1%–1.55% of all trauma patients. Blunt cervical vertebral

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artery injury is less common – 0.2% to 0.77% of trauma patients. Motor vehicle accident constitutes 41–70% of cervical TCVI. Assault, pedestrian struck by vehicle, assault and hanging constitute 10–20%, 12–18%, 5–15%, 5% respectively.[2]

1.2. *Pathophysiology and patterns of injury*

Blunt force TCVIs can result from any of the following physiological mechanisms like extreme hyperextension/rotation, direct vascular blow, intraoral trauma, or direct laceration from bony fracture fragments.

Extracranial carotid artery injuries most commonly occur in the distal cervical ICA. Vertebral artery dissections most commonly occur in the V2 or V3 segments. Aneurysms of the extracranial carotid and vertebral arteries caused by blunt trauma typically result from a disruption of the internal elastic lamina, weakening of the artery wall, and expansion of the adventitia. Traumatic aneurysms of the extracranial carotid system are present in 15%–33% of cases of TCVI (Fig. 1) and tend to occur in the mid- or upper-cervical parts of the vessel. Traumatic aneurysms of the vertebral artery are present in 4%–8% of cases. Traumatic extracranial arteriovenous fistulae most commonly involve branches of the external carotid artery and typically present as a pulsatile mass with a bruit.

1.3. *Risk factors for TCVI*

The strongest predictor of a carotid artery injury is a closed head injury (Glasgow Coma Scale score </ = 6). The strongest predictor of a vertebral artery injury is a cervical spine injury.[3]

Factors and physical findings like basilar cranial fracture, cervical bruit, cervical hematoma, facial fractures, Horner's syndrome, neurological deficit not explainable by other injuries, seat belt sign can be attributed with blunt trauma cerebrovascular injury.

1.4. *Screening protocol for blunt trauma cerebrovascular injury*

The Denver screening criteria[4] and Memphis criteria[5] has been developed to aid in diagnosis and treatment of blunt cerebrovascular injury. They list the following risk factors

1. Presence of Le forte II or III fractures
2. Cervical spine fractures involving subluxation
3. Cervical spine fractures involving C1–C3
4. Cervical spine fractures extending into the transverse foramina
5. Basilar skull fractures with carotid canal involvement
6. Diffuse axonal injury with a Glasgow Coma Scale of 6 or less
7. Near hanging injuries with anoxic brain injury
8. Horner's syndrome
9. Neck Soft tissue injury

1.5. *Presentation*

Classic clinical triad of dissection of cranial vessels is 1. Head-ache/neck pain, 2. Horner syndrome, and 3. Brain ischemia.

Unilateral vertebral artery injuries often remain completely asymptomatic because only 12%–20% will present with symptoms of vertebrobasilar ischemia. An important

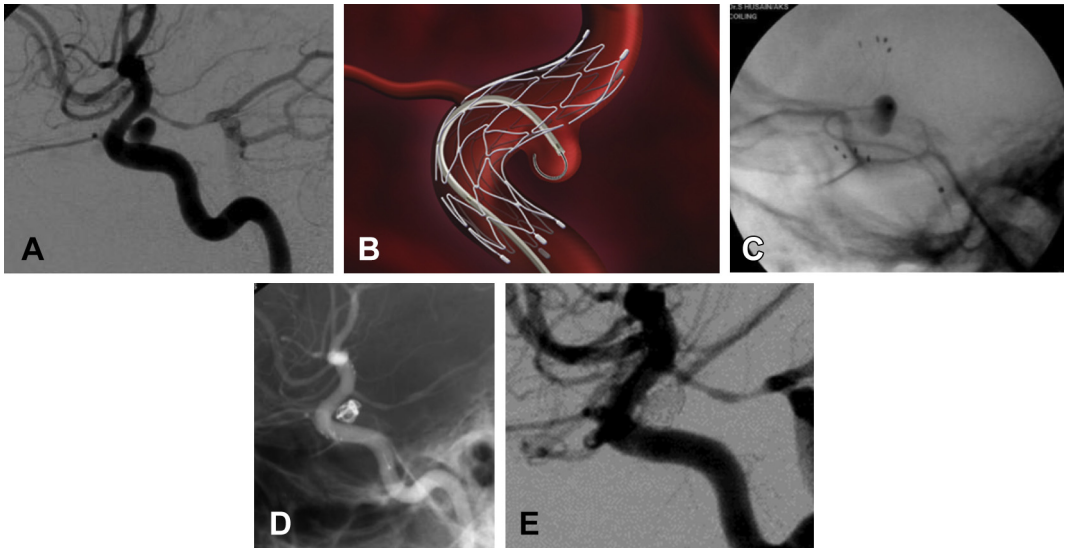

Fig. 1 – Endovascular Stent assisted repair of a dissecting pseudo-aneurysm of para-clinoid LICA. (A) cerebral DSA LICA in lateral projection delineating the rent in the posterior wall of supraclinoid LICA on its posterior wall with a medium seize, mushroom shaped pseudo-aneurysm. (B & C) deployment of a self-expanding stent Neuroform (Stryker US) across the rent and micro-catheter positioned in the pseudo-aneurysm. (D) Placement of an undersized framing coil loosely and then (E) progressive packing of the pseudo-aneurysm by smaller coils to achieve a complete occlusion of the rent in the artery with remodeling of the lumen.

difference between spontaneous and traumatic dissections is the timing of symptom presentation because the presentation of ischemic symptoms is frequently delayed in patients with TCVI.

1.6. *Imaging*

CT scan of the head and neck is a standard imaging procedure for the initial workup of most trauma patients. Noncontrast CT scans can identify clues to the presence of a TCVI, such as cranial base fractures or cervical spine injury.

Cerebral DSA is widely considered as the gold standard for imaging of TCVI. It can produce high resolution images of the site of injury, detect subtle intimal injury and identify intraluminal thrombus and intracranial branch occlusions more accurately than any other modality. Cerebral Angiography is usually indicated in patients with unclear imaging findings or with a clear need for Neurointerventional management. CT Angiography has a sensitivity ranging from 74 to 90%.

Magnetic resonance angiography can be useful because of the anatomic resolution of magnetic resonance imaging (MRI) and the ability of MRI to identify ischemic brain injury. The accuracy of MRA is limited. Compared with angiography, MRA has sensitivity to detect carotid and vertebral artery injuries of 50% and 47%, respectively.

TCD has been advocated as a faster and less inexpensive means for screening of trauma patients. TCD findings suggestive of TCVI include asymmetry in mean middle cerebral artery blood flow velocities by more than 20 cm/s, dampened velocity waveform in the middle cerebral artery, signs of collateral cross flow.

1.7. *Management*

Management of blunt cerebrovascular injuries is controversial. No prospective, randomized trials have been completed, and most of the published data come from only 2 centers (Denver and Memphis).

2. Medical therapy

Most authors agree that some form of antithrombotic treatment is appropriate for most patients with TCVI. The rationale for antithrombotic therapy is based on the belief that most cerebral ischemic events in TCVIs are thromboembolic in origin.[6] The most widely reported antithrombotic regimen has been anticoagulation with intravenous heparin. Hemorrhagic complication rates of anticoagulation in this setting have ranged from 8% to 16%.[7] About 30%–36% of patients with TCVI are not candidates for systemic anticoagulation because of concomitant injuries. Recent recommendations describe a target partial thromboplastin time of 40–50 s. Antiplatelet therapy (either aspirin alone or dual antiplatelet therapy with aspirin and clopidogrel) is an alternative to anticoagulation that may carry a more favorable risk of complications. Several retrospective studies have reported that antiplatelet therapy is equivalent to or superior to anticoagulation in terms of neurological outcomes.

Endovascular treatment of TCVIs is now considered the nonmedical treatment of choice ahead of surgery in many centers.[8] Endovascular options include: stenting with balloon expandable, self-expanding or covered stents[9]; embolization of traumatic aneurysms with coils or liquid embolics; intra-arterial thrombolysis; and Endovascular vessel occlusion.

Surgical options for the treatment of TCVI include Vessel ligation, thrombectomy, direct suturing of intimal injury, replacement of injured vessels with interposition grafts, and bypass grafting. Potential risk of carotid ligation is an elevated risk of hypertension and de novo intracranial aneurysm formation. Direct surgical repair of carotid injuries carries a significant risk of cranial nerve injury.[10]

2.1. *Choice of management strategy*

No organized guidelines exist for the treatment of TCVI. All authors seem to agree that antithrombotic medical therapy is appropriate for most patients, and antiplatelet therapy appears to be gaining popularity as a safer alternative to anticoagulation.

Cervical Artery Dissection in Ischemic Stroke Patients (CADISP)[11] study recommends antiplatelet treatment in most patients with TCVI, but advocates anticoagulation in patients with recurrent symptoms while on antiplatelet medications, with vessel occlusion, or with injuries associated with free-floating intraluminal thrombus.

Endovascular treatment is safer than surgery for most TCVI lesions and has become the treatment modality of choice in most centers.

2.2. *Prognosis*

Early series found rates of permanent neurological deficits of 40%–80% and mortality of 20%–40%. Series published within the past decade have reported overall neurological morbidity of extracranial TCVI of 0–31%. Neurological morbidity rates are higher with carotid injuries compared with vertebral artery Injuries. Traumatic aneurysms are present at the time of diagnosis in 8%–33% of patients with TCVI, and carotid traumatic aneurysms are more common than vertebral artery aneurysms.

In contrast to spontaneous dissecting aneurysms, which frequently stabilize or resolve over time, traumatic aneurysms tend to persist and enlarge in the majority of cases.

2.3. *Intracranial TCVI*

Data on the overall incidence of intracranial cerebrovascular injuries is lacking, although they appear to be substantially less common than extracranial carotid and vertebral artery injury. In patients with blunt trauma, a Glasgow Coma Scale score of 8 or lower and the presence of facial fractures are independent risk factors for intracranial arterial injury.

Intracranial carotid artery injury is present in 11% of patients with carotid canal fractures. Intracranial arterial injuries manifest primarily as arterial occlusions, aneurysms, and arteriovenous (most often carotid-cavernous) fistulae.

2.4. *Traumatic intracranial aneurysms*

They account for less than 1% of all intracranial aneurysms.[12] Although blunt trauma is a significant source of traumatic intracranial aneurysms, traumatic aneurysms caused by penetrating trauma are the most frequently reported. Blunt trauma–related aneurysms result from rapid deceleration, causing sudden brain movement and arterial wall injury from stationary structures such as the cranial base or the falx cerebri. Injury to the distal anterior cerebral artery by the edge of the falx accounts for the finding that the pericallosal artery is a common location of traumatic aneurysms. Traumatic aneurysms of the basilar artery and petro cavernous segment of the ICA are associated with cranial base fractures.

Cranial fractures are a clue to the presence of a traumatic aneurysm and are present in 90% of traumatic intracranial aneurysms. Aneurysms caused by blunt trauma typically appear in a delayed fashion, ranging from days to weeks after the initial injury. They are also unstable lesions because 50% rupture within the first week after diagnosis.

Traumatic aneurysms of the cavernous ICA may present with life-threatening epistaxis caused by bleeding into the sphenoid sinus. Traumatic aneurysms are sometimes smaller than 3 mm in size and are frequently located in peripheral arterial branches. Because traumatic aneurysms may appear in a delayed fashion after injury, some authors have recommended delaying angiography by 10 days to 2 weeks after the injury.

Treatment is recommended for all traumatic intracranial aneurysms when feasible because conservative management is associated with a mortality rate of nearly 50% (Fig. 1). Recent series of 13 traumatic intracranial aneurysms treated with endovascular techniques, there were no procedure-related complications or delayed hemorrhage over a mean follow-up of 2.6 years.

2.5. *Traumatic intracranial arteriovenous fistulae*

Direct carotid-cavernous fistulae (CCFs) are the most common intracranial traumatic arteriovenous fistula and are present in 4% of all patients with blunt TCVI. 75%of direct CCFs are caused by trauma and result from cranial base or facial fractures, although iatrogenic injury, such as during endoscopic sinus surgery or trans sphenoidal pituitary surgery, represents an important subset of cases.

3. Conclusion

TCVIs occur in approximately 1% of blunt trauma patients. 20% of patients with TCVI do not have an established risk factor. CTA is a useful screening tool for TCVI but DSA remains the gold standard. Early recognition of craniocervical vascular injury is of value in early planning of management and avoidance of secondary ischemia or long-term sequalae of these vascular injuries. Optimal antithrombotic regimen has not yet been identified. Endovascular treatment of TCVI is safer than surgery although the indications for treatment should be individualized.

Case study

- Mr. MK 20 years.
- *Presenting complaints:* RTA followed by unconsciousness for a few min and continuous headache.
- *On Examination:* Patient was conscious, GCS score was 15/15, pulse was 90/min; BP-140/90 mm of Hg; there was mild neck stiffness present.
- *CT Scan Head:* Diffuse SAH.

Cerebral DSA: 1st Cerebral DSA reveals no aneurysm, but there was a severe spasm of LICA in its supraclinoid segment. A 2nd Cerebral DSA after one month revealed a dissecting pseudo-aneurysm of LICA para-clinoid region over the posterior wall about 5–6 mm proximal to a dominant PcomA. Its location and morphology is pathognomonic of dissection of LICA.

- *Diagnosis:* Traumatic dissection of para-clinoid LICA with pseudo-aneurysm.
- *Procedure:* Endovascular repair of LICA dissection (Fig. 1) and obliteration of pseudo-aneurysms under GA.
- *Angiographic outcome*: Raymond grade –1
- *Clinical Outcome*: mRS-0.

Conflicts of interest

In last three years Dr. Husain have received: educational grants from Stryker Neurovascular, Abbott Vascular, and Covidien to conduct Delhi Course on Neurointervention under the aegis of Stroke & Neurointervention Foundation (SNIF) of which I am the chairman; speaker fees from Stryker Neurovascular; honoraria from scientific advisory boards from Stryker Neurovascular, Abbott vascular; Proctorship fees from Stryker Neurovascular; and travel support from Stryker Neurovascular. I am chairman of subsection of Interventional Neurology of Indian Academy of Neurology and serves on the editorial board of Frontiers in Endovascular and Interventional Neurology, Journal of Neurology and Stroke, and Internet Journal of Neurology.

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