Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/ijnt

Neurointervention

Neurovascular injuries in trauma: An under recognized entity

S.P. Goutham, H. Husni, S. Vanchilingam, Shakir Husain *

Department of Interventional Neurology and Stroke, Saket City Hospital, Institute of Neurosciences, Saket, New Delhi 110017, India

ARTICLE INFO

Article history:
Received 1 December 2014
Accepted 12 December 2014
Available online 20 December 2014

Keywords:
Traumatic-SAH
Pseudo-aneurysm
Cerebrovascular injury
Dissection
Endovascular treatment

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

* *Corresponding author*. Tel.: +91 98 101 20942.
E-mail address: drshakir@gmail.com (S. Husain).
http://dx.doi.org/10.1016/j.ijnt.2014.12.009

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

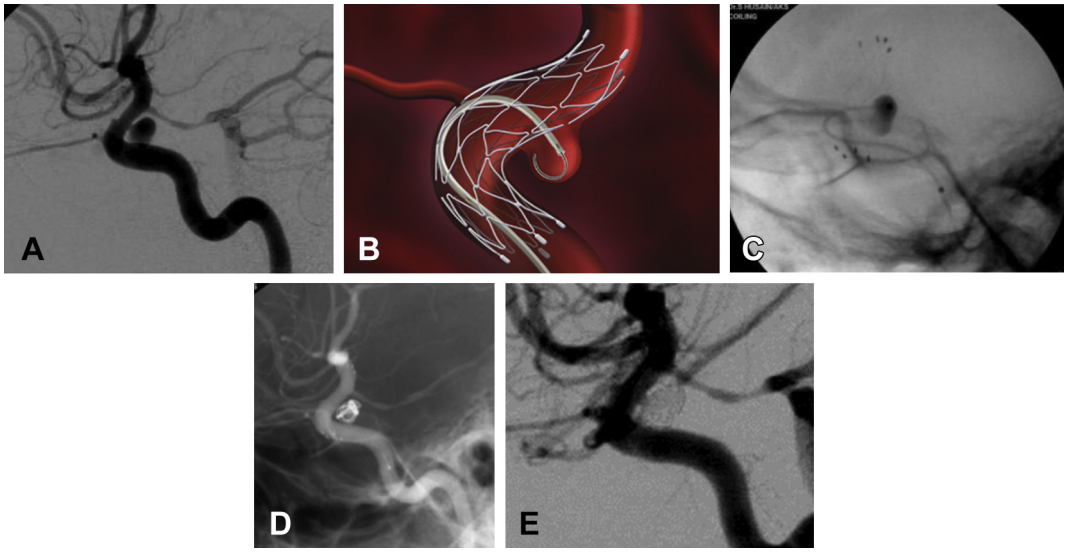

Fig. 1 – Endovascular Stent assisted repair of a dissecting pseudo-aneurysm of para-clinoid LICA. (A) cerebral DSA LICA in lateral projection delineating the rent in the posterior wall of supraclinoid LICA on its posterior wall with a medium seize, mushroom shaped pseudo-aneurysm. (B & C) deployment 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.