Traumatic Intracranial Aneurysm Formation following Closed Head Injury

Abstract

Background: Traumatic intracranial aneurysms are rare conditions that can be a result of non-penetrating head trauma. We report the occurrence of intracranial aneurysms in patients with traumatic brain injury.

Methods: All diagnostic cerebral angiograms performed in patients with traumatic brain injury at a level I trauma center from January 2006 to July 2007 were reviewed.

Results: Diagnostic cerebral angiography was performed in 74 patients with the diagnosis of closed head injury. A total of 4 traumatic intracranial pseudoaneurysms were found in 4 patients, two in the supraclinoid segment of the internal carotid artery, one in the cavernous segment of the internal carotid artery and one in the parapiphthalamic segment of the internal carotid artery. Two patients were treated with coil embolization. One patient had follow up imaging on which there was no change in the size and morphology of the aneurysm.

Conclusion: Intracranial aneurysms can develop in patients with closed head injury presumably related to shear or rotational injury. It is unclear whether these aneurysms should be classified as traumatic intracranial aneurysms or pseudoaneurysms, but the pathological findings frequently reveal disruption of the three vascular layers fulfilling the definition of pseudoaneurysm. For these reason we favor the name of post-traumatic intracranial pseudoaneurysms.

Keywords: Intracranial aneurysm; traumatic brain injury; cerebral angiogram; shear injury; pseudoaneurysm

Intracranial aneurysms are classified as congenital, atherosclerotic, infectious and post-traumatic. The traumatic formation of the aneurysms can occur as a consequence of penetrating and non penetrating injuries\(^1\) and overall are considered to be responsible for less than 1% of all aneurysms.\(^4\) Traumatic aneurysms as result of closed head injury without direct injury to the arteries are rarely found and poorly characterized.

Abbreviations: ICA, internal carotid artery; AP, antero-posterior

Methods

We reviewed all diagnostic cerebral angiograms performed in patients admitted to a level I trauma center with the diagnosis of non penetrating traumatic brain injury between January 2006 and July 2007. All patients underwent computed tomography (CT) of the head and cervical spine upon initial trauma evaluation. In those considered high risk for vascular injuries\(^7\)\(^9\), diagnostic cerebral angiography was performed.

Results

A total of 74 diagnostic cerebral angiograms were performed in patients with non penetrating head and or cervical trauma. In 26 patients angiographic vascular injuries were detected, including 4 patients with traumatic intracranial aneurysms.

Cases

Case 1:
A 40-year-old pedestrian man was involved in a motor vehicle accident, his initial Glasgow Coma Scale (GCS) was 3 and intubation
was performed on the scene. Examination revealed a large con-
tusion and scalp hematoma in the occipital area. Pupils were
equal and reactive. No limb movements were reported. Imag-
ing demonstrated bilateral subdural, subarachnoid, and intra-
ventricular hemorrhages with bifrontal parenchymal hemor-
rhages. Non-displaced fractures of the posterior wall of the
right foramen lacerum and medial wall of the left foramen lac-
erum were also seen on CT scan. On hospital day one, cerebral
angiography revealed a right internal carotid artery paraoph-
thalmic aneurysm measuring 4mm x 4 mm (Figure 1, A and B).
Early in the hospital course, the patient developed intractable
increased intracranial pressure (ICP) despite aggressive hy-
perosmolar therapy and external ventricular drainage (EVD)
progressing to brain death on hospital day 6.

Case 2:
A 15-year-old man was involved in a motor vehicle accident.
Initial GCS was 6. He was intubated on the scene and initial
examination revealed multiple facial lacerations with minimal-
lly reactive pupils and non-purposeful extremity movements.
Trauma imaging demonstrated subarachnoid, intraventricular,
subdural and bifrontal parenchymal hemorrhages and multiple
skull fractures involving bilateral lacerum foramina. Exter-
nal ventricular drainage was performed and later underwent
bifrontal decompressive craniectomy for increased ICP. On
hospital day 21, loss of movement in his right upper extremity
was reported. For this reason CT angiography was performed
and demonstrated an unruptured left internal carotid artery
intracranial aneurysm. On cerebral angiography, the presence
of a left internal carotid artery supraclinoid pseudoaneurysm
measuring 4.5mm x 9mm x 8.5mm (Figure 2) was confirmed.
Treatment with neuroform stent-assisted coiling followed. The
patient was discharged to rehabilitation on hospital day 54.

Case 3:
A 57-year-old woman, helmeted motorcycle driver, was admit-
ted following a road traffic accident. Initial GCS was 11 and
initial examination was significant for paraparesis. Imaging in-
cluding CT angiography demonstrated multiple cervical spine
fractures with anterior dislocation of the C6 vertebral body,
left vertebral artery occlusion and a right internal carotid ar-
tery cavernous segment aneurysm. On hospital day one, cere-
bral angiography demonstrated left vertebral artery occlusion
between the levels of C4-C6 and a 7mm x 6mm right internal
carotid cavernous segment aneurysm (Figure 3, A and B). Prox-
imal and distal coil embolization of the left vertebral artery
stump was performed. On hospital day 21, she was discharged
to rehabilitation. In a six month follow up CT angiography no
change in the size and morphology of the aneurysm was dem-
onstrated.

Case 4:
A 24-year-old man (unhelmeted cyclist) was ejected from his
bicycle during a collision with another cyclist. Initial GCS was

Figures 1-3. The arrows point at the aneurysm.
Figure 1A. Right ICA injection, AP projection.
Figure 1B. Right ICA injection, 3-D reconstruction, lateral
projection.
Figure 2. Left ICA injection, AP projection.
Figure 3, A and B. Right ICA injection, AP (A) and oblique
(B) projection

Abbreviations: ICA, internal carotid artery; MCA, middle
cerebral artery; ACA, anterior cerebral artery; OA, ophthalmic
artery
3. Examination was remarkable for left facial lacerations, unresponsiveness, no spontaneous extremity movements and left pupillary midriasis. Trauma imaging revealed subarachnoid and subdural hemorrhage with a 7mm left-to-right midline shift, multiple vertebral, facial and skull base fractures including a right carotid canal fracture (Figure 4C). The patient underwent decompressive craniectomy, left subdural hematoma evacuation and EVD placement. On hospital day 1, CT angiography revealed a left internal carotid artery supraclinoid aneurysm. Cerebral angiography demonstrated a 2mm x 4.5mm x 4.0 mm pseudoaneurysm (Figure 4A and 4B) that was treated with endovascular coil embolization. The patient was discharged to rehabilitation on hospital day 33.

Discussion

Traumatic intracranial aneurysms can be classified histologically as true or false aneurysms (pseudoaneurysm). In true aneurysms, the intima, internal elastic lamina and media layers are disrupted but with an intact adventitia. In false aneurysms, there is disruption across all layers of the arterial wall and it is a hematoma in the surrounding tissue that prevents blood extravasation. The latter, is considered the most common form of traumatic intracranial aneurysms.1, 10 The location of traumatic intracranial pseudoaneurysms varies, but these are commonly found in the anterior circulation. Reported locations are the vertebral artery11-13, internal carotid artery14-17, anterior cerebral artery14,15,19,21,22, middle cerebral artery14,15,19,21 and middle meningeal artery23,24. These intracranial arteries are thought to be vulnerable to injuries when they travel adjacent to skull fractures15,23,25 or even normal structures like the falx (seen in the anterior cerebral artery)14,18,26 or sphenoid wing (seen in middle cerebral artery).22 Our four cases involved the intracranial internal carotid artery and more specific, two supraclinoid, one cavernous and one paraophthalmic.

The intracranial space into which the hemorrhage occurs depends on the vessel and the segment of the vessel involved. Once an extracranial artery becomes intradural, the external elastic lamina disappears transforming the arterial wall into a relative weak vessel.27 For this reason, the traumatic involvement of the adventitia in a vessel can result in subarachnoid hemorrhage12,11,10,21, epidural28,29, epistaxis14,15 or intraparenchymal21,26 hemorrhage. Aneurysm rupture did occur in one of our cases (Case #4).

The mechanisms of injuries are vast but commonly reported as consequence of sports30,31, assault15 or traffic accidents, such as in pedestrians14,16 or in vehicle passengers.16,22,26 No imaging screening recommendations for intracranial injuries have been published, but probably the already established guidelines for blunt trauma7-9 are the most appropriate for posttraumatic intracranial aneurysm detection. Some features in the clinical course following a head injury should raise suspicion of a posttraumatic intracranial pseudoaneurysm such as occurrence of a delayed onset of a cranial nerve palsy, stupor, coma or severe epistaxis. The reported risk of hemorrhage in posttraumatic pseudoaneurysms is 19%, with a peak incidence of rupture at 2-3 weeks20,21 after the injury, resulting in 32-54%10,20,21 mortality. Therefore, an early diagnosis and treatment is recommended. Treatment options include surgical repair and endovascular interventions. Treatment approach is often carried out on a case to case basis since atypical locations and variable shapes are often encountered. Surgical clipping, resection, or trapping of the aneurysm are considered to be surgical treatment options.26

Figure 4, A-C.
Figures 4A, 4B. Left internal carotid artery injection, oblique projections. The arrow points at the pseudoaneurysm.
Figure 4C. Computed tomography of the skull, 3-D reconstruction, reveals multiple fractures.
Endovascular interventions include embolization with liquid agents, detachable balloons, or coils which may require assistance with balloon or a stent placement. Patient’s clinical condition may also limit treatment options, for example unstable patients may not be suitable to undergo general anesthesia or in patients with active bleeding that cannot be placed on dual antiplatelet therapy in anticipation of stent assisted aneurysm coil embolization.

Conclusion

It is unclear whether these aneurysms should be classified as traumatic intracranial aneurysms or pseudoaneurysms, but the pathological findings frequently reveal disruption of the three vascular layers fulfilling the definition of pseudoaneurysm. For these reason we favor the name of post-traumatic intracranial pseudoaneurysms.

We propose the hypothesis that the traumatic formation of intracranial carotid artery aneurysms that are not immediately adjacent to fractures occur in segments where the artery fixes to the skull and other elements. These points include are the dura and carotid canal caudally; the ophthalmic artery anteriorly; posterior communicating artery posteriorly; and the origin of anterior and middle cerebral arteries cranially.

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References