True traumatic aneurysm of the vertebral artery

Case report

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A patient is reported in whom a true aneurysm of the vertebral artery developed following head trauma. The histology of the aneurysm and parent vessel is presented, and its implications regarding the mechanism of formation of posttraumatic true aneurysms are discussed.

KEY WORDS • head injury • traumatic aneurysm • vertebral artery aneurysm

T R A U M A T I C aneurysms of the intracranial arterial vasculature are an unusual but well recognized complication of severe head trauma, and are often associated with a high mortality rate. They usually occur on vessels of the anterior circulation, but have been reported on the posterior cerebral artery, the superior cerebellar artery, and the basilar artery. They have not been described in other vessels of the posterior circulation.

Most traumatic aneurysms are “false” aneurysms, which are postulated to be formed from a through-and-through rent in the vessel wall at the time of the trauma. In unusual circumstances, however, “true” aneurysms, have been reported; these are thought to be formed from partial traumatic disruption of the vessel wall. Unfortunately, the pathology reported in these cases has not always been well described with regard to associated changes in the parent vessel, aside from the area of the breach, so this hypothesis is somewhat speculative.

The following case report describes a child with a head injury which was followed 3 weeks later by two episodes of subarachnoid hemorrhage, leading to death from a ruptured right vertebral artery aneurysm. This case is unusual because of the location of the aneurysm, the fact that it is a true aneurysm, and the demonstration of multifocal histological lesions in the wall of the parent vessel adjacent to the aneurysm, which were most likely the result of trauma.

Case Report

This 8-year-old boy fell 25 feet from a tree, landing on his right arm and the right side of his head. He lost consciousness as a result of the fall, and had an open fracture of the right humerus.

Examination. He was comatose on admission but had no cranial nerve or lateralizing motor or sensory findings. Reflexes were symmetrical, with bilateral flexor toe signs. X-ray studies showed the fractured humerus and a normal cervical spine and skull. Urinalysis revealed evidence of hematuria, and an intravenous pyelogram and cystogram showed a ureteropelvic junction obstruction with hydronephrosis on the left, which was believed to be a preexisting problem.

Course. The humeral fracture was stabilized, and over the next 2 days the patient gradually returned to his normal mental status. Delayed primary closure of the fracture site kept him hospitalized for the next 7 days, and he was discharged 9 days after the accident without neurological sequelae; however, he did complain of headache which responded to minor analgesics. Over the next week, he described several episodes of transient diplopia lasting from 10 to 15 seconds. On the 17th day after the initial trauma, he suddenly developed severe bifrontal headache which spread to the occiput and down his spine, and was accompanied by vomiting. He was readmitted to the hospital.
Examination revealed normal vital signs, a supple neck, and a normal neurological examination. Computerized tomography (CT) of the head was normal on the day of the admission. Meningismus developed the following day, and a lumbar puncture revealed an opening pressure of 60 mm H2O, 46,000 red blood cells with a xanthochromic supernate, 770 white blood cells, a protein content of 50 mg%, and a glucose content of 60 mg%. The patient was placed at bed rest. Before an arteriogram could be performed, he suddenly deteriorated and became comatose. Examination revealed normal pulse and blood pressure, irregular respirations, pupils fixed in the midposition, absent extraocular movements and corneal reflexes, and flaccid extremities. After intubation, hyperventilation, and mannitol administration, extensor and flexor posturing developed. A CT scan of the head now revealed blood in the ventricular system which was not dilated. The patient deteriorated further over the next day and died 21 days after the initial head trauma.

Neuropathological Examination. No fractures of the skull were found. The brain, which was examined after fixation in 10% buffered formalin for 2 weeks, weighed 1420 gm; the brain stem and cerebellum weighed 195 gm. The dura was normal, and the superior sagittal sinus was patent. A moderate amount of subarachnoid hemorrhage diffusely covered the cerebrum and cerebellum, filling the basal cisterns. The cerebral arteries were normal except for a saccular aneurysm, 5 mm in diameter, on the lateral surface of the right vertebral artery, 2 mm proximal to the origin of the posterior inferior cerebellar artery (PICA) (Fig. 1). Externally, the brain showed marked edema with flattening of the gyri. Coronal sections of the cerebral hemispheres revealed that the lateral and third ventricles were compressed, containing a small amount of blood clot. Horizontal sections of the brain stem and cerebellum revealed blood clot within the fourth ventricle.

Microscopic sections of the brain revealed mild anoxic changes of neurons in the hippocampus, inferior olives, and cerebellum. Subarachnoid hemorrhage was present over the brain and spinal cord.

Serial sections of the right vertebral artery including the aneurysm revealed several abnormalities, represented diagrammatically in Fig. 2. The aneurysm arose from the artery near the origin of the PICA, but at a point 90° to 120° around the circumference of the artery from it. Focal intimal fibrosis was present both on the aneurysm side of the vessel and on the opposite side, including around the origin of the PICA. The internal elastic membrane was disrupted in two
Fig. 3. A cross section of the vertebral artery distal to the aneurysm showing a hematoma surrounded by fibrotic scar between the media and adventitia, intimal fibrosis, a focal interruption of the internal elastic membrane with fibrosis, and a focal fibrosis of the media. As noted in Fig. 2, this is well above the aneurysm site. Verhoff-van Gieson, X 33.

Fig. 4. A cross section of the vertebral artery just above the aneurysm showing a dissecting aneurysm surrounded by fibrotic thickened scar tissue in the media. Verhoff-van Gieson, X 33.

locations, one well above and remote from the site of the aneurysmal breach (Fig. 3), and one at the site of the breach (Fig. 4). Coils of elastin were seen throughout the latter site, and within the vertebral artery lumen. The media was thinned and fibrotic on the aneurysm side throughout the area sectioned, as well as circumferentially in several places. A small hematoma surrounded by an organized fibrotic scar was present between the media and adventitia on and above the aneurysm (Fig. 3); at the level of the distal break in the internal elastic membrane, a fibrotic break in the media also occurred (Fig. 3). At the site of the aneurysm breach, the media was absent (Figs. 4 and 5); it was present below the breach, but very fibrotic.

The adventitia provided part of the wall of the aneurysm (Fig. 5); the remainder was formed by the clot lying within the aneurysm, which was partially organized. Several areas of focal hemorrhage distal to the aneurysm were present within the adventitia. A

Fig. 5. The aneurysm at the rupture site showing the aneurysm to be partially covered with adventitia. Laminated clot fills the aneurysm and adjacent vertebral artery lumen. Gomori trichrome, X 23.
laminated and partially organized clot was present within the lumen of the vertebral artery at the level of the aneurysm breach.

Discussion

An intracranial aneurysm directly or indirectly produced by trauma is a rare, but well established entity. Traumatic intracranial aneurysms frequently occur at the base of the brain where "congenital" saccular aneurysms are common, and their gross appearance often appears identical, so that the differential diagnosis between the two may be quite difficult at times.

The diagnosis of a traumatic aneurysm is indisputable when it can be proved that a patient did not have an aneurysm before head injury but developed one after head injury. A patient can have head injury as a result of a ruptured berry aneurysm, or a coincidental berry aneurysm can occur in a patient who had head injury. Since such proof is usually unavailable, the following criteria are used for distinguishing traumatic aneurysms from ordinary saccular aneurysms: 1) history of the trauma; 2) site of the aneurysms; 3) age of the patients; and 4) the histopathological findings.

History of Trauma. The reported time between trauma and the diagnosis of aneurysm ranges from a few hours to 10 years, with an average lapse of 2 weeks. Although a history of severe head injury, especially with evidence of skull fracture or penetrating wound near the site of aneurysm, is essential for the diagnosis of traumatic aneurysm, such a history alone is insufficient in ruling out a coincidental saccular aneurysm.

Site of Aneurysms. Most "congenital" saccular aneurysms arise at a junction of two or more branches of major arteries in the circle of Willis and the trifurcation of the middle cerebral arteries. Traumatic aneurysms tend to arise in the extracranial portion of the internal carotid artery as the result of basal skull fracture, from small peripheral arteries adjacent to the edge of the falx and tentorium as the result of arterial contusion against these structures, and from small arteries on the surface of the brain as the result of fracture of the skull vault, or from a penetrating wound or arterial contusion against prominences of bone. Aneurysms arising from small peripheral arteries or superficial arteries are diagnostic of traumatic or inflammatory origin. However, traumatic aneurysms arising at the base of the brain are difficult to differentiate from ordinary saccular aneurysms.

Age of Patients. Although "congenital" saccular aneurysms can occur in young patients, this is uncommon. Aneurysms arising in young children can be suspected to be of traumatic or inflammatory origin if the history and site of the aneurysm are consistent. This criterion is not useful if a patient is an adult.

Histopathological Findings. Traumatic aneurysms can be either true or false aneurysms. Practically all "congenital" saccular aneurysms are true aneurysms, so that false aneurysms are most likely of traumatic origin. Histopathological findings at the site of the arterial rent can be identical in true traumatic and "congenital" saccular aneurysms; however, multifocal injuries of the arterial wall are frequently found in the wall separate from but adjacent to the site of rent of traumatic aneurysms. These lesions include old focal hemorrhages in the arterial wall, dissection of the arterial wall, focal fibrotic scar of the media, focal intimal fibrosis, or foci of ruptured internal elastic lamina. There is very little doubt that an aneurysm was induced by trauma when the estimated age of multifocal scars in the arterial wall matches the duration between the trauma and death.

In spite of the lack of adjacent skull fracture or penetrating wound and the unusual location of the aneurysm, in this case the presence of multifocal scars in the adjacent arterial wall of an age dating back to the time of the trauma strongly suggests that the aneurysm in the right vertebral artery was indeed caused by the head injury that occurred 21 days prior to death.

Vascular injury leading to the formation of traumatic aneurysms can occur by direct trauma with compression of the vessel against the relatively fixed edges of the falx or tentorium, by laceration of the vessel by edges of a skull fracture, bone fragments, missiles, or surgical instruments, or by torsion and overstretching of the vessels at the time of the trauma. Shaw and Alvord noted that vascular injury by stretching and torsion was an infrequent event due to the relative immobility of the intracranial vessels, but that a fracture in the vicinity of the vessel could cause enough additional vessel displacement to enable stretching and torsion injury to occur. In the absence of a skull fracture or close proximity of the aneurysm to dural edges in this patient, it is reasonable to assume that the vessel trauma was due either to a contusion of the vertebral artery against a bone prominence, or to stretching of the artery associated with torsion of the brain at the time of the trauma.

References


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