AJNR

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AJNR Am J Neuroradiol 1998, 19 (2) 386-388
http://www.ajnr.org/content/19/2/386

Spontaneous Thrombosis of a Basilar Artery Traumatic Aneurysm in a Child

Laurie A. Loevner, Tricia Y. Ting, Robert W. Hurst, Herbert I. Goldberg, and Luis Schut

Summary: Traumatic aneurysms are rare and occur most commonly in young adults; however, the relative frequency in the pediatric population is high, owing to the low prevalence of congenital saccular aneurysms in children. Traumatic aneurysms typically involve the anterior circulation, and spontaneous thrombosis is uncommon; hence, surgery is usually necessary. We present a case of a posttraumatic aneurysm in a child that occurred after a fall from a large height and that spontaneously thrombosed.

Intracranial aneurysms in the pediatric age group are uncommon. Clinical presentation is usually related to mass effect associated with large aneurysms or intracranial hemorrhage. Traumatic aneurysms are rare, constituting less than 1% of all intracranial aneurysms (1). They occur more commonly in males, with a ratio of 2:1 or 3:1 relative to females, most likely reflecting the greater frequency of trauma among males. In addition, the majority of aneurysms associted with trauma occur in young adults, with approximately 30% of reported cases occurring in children and adolescents before the age of 20 years (2). However, because congenital aneurysms are rare in the pediatric population, the relative frequency of trauma-induced aneurysms in children is high. The vast majority of traumatic aneurysms involve branches of the middle and anterior cerebral arteries (2–5) or the internal carotid artery at the level of the skull base (1, 4, 5). They rarely involve the posterior circulation (3, 6–8), and are extremely unusual in the basilar artery; we found fewer than 10 cases reported in the past three decades (3, 9, 10).

Spontaneous thrombosis of intracranial aneurysms is uncommon, but has occasionally been described (1, 11). We report a case of complete spontaneous thrombosis of a presumed posttraumatic giant aneurysm of the basilar artery.

Case Report

A 7-year-old boy was admitted to a hospital in Bosnia-Herzegovina in 1989 with a left third nerve palsy and a sudden change in mental status manifested by a depressed level of consciousness. A computed tomographic (CT) scan of the head reportedly showed a midbrain hematoma. Cerebral angiography showed a giant aneurysm arising from the distal basilar artery at the level of the superior cerebellar and posterior cerebral arteries. Angiography also showed a clot within a portion of the aneurysm (Fig 1A and B). The patient's medical history was remarkable only for a significant closed-head injury sustained from a fall while playing 9 months prior to presentation, at which time a head CT study was reportedly normal.

Owing to war in his nation, the patient was lost to follow-up and was not seen again until 13 years of age, at which time his left third nerve palsy was again noted. Because he was known to have an aneurysm and because of the high likelihood of rupture associated with these lesions, he was sent to the United States for further management. A head CT scan showed an extraaxial mass with a partially calcified rim in the prepontine and suprasellar cisterns, consistent with an aneurysm (Fig 1C). Cerebral angiography showed filling of the distal basilar artery and its branches; however, it failed to opacify the aneurysm shown previously, consistent with complete interval thrombosis (Fig 1D and E). The patient was discharged the next day in good condition.

Discussion

The vast majority of cerebral aneurysms are true aneurysms that involve all layers of the arterial wall, including the internal elastic lamina, the media, and the adventitia. Most true aneurysms are congenital in nature, although their appearance in childhood is uncommon. They usually occur in the circle of Willis at the bifurcation of arteries, with approximately 33% arising at the anterior communicating artery, 33% at the origin of the posterior communicating artery, 20% at the middle cerebral artery bifurcation, and 14% involving the vertebral and basilar circulation.

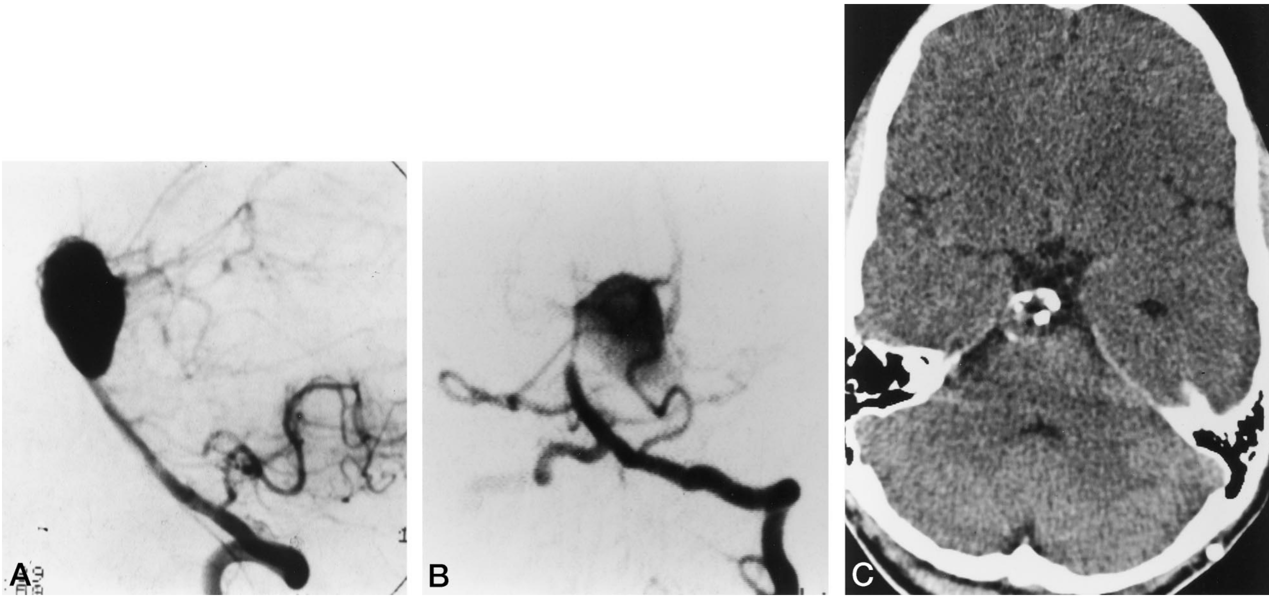
In contrast to true aneurysms, pseudoaneurysms may result from rupture of a true aneurysm, or typically are mycotic or posttraumatic in nature. They result from injury to one or more layers of the arterial wall (3). Although uncommon, traumatic pseudoaneurysms may occur after penetrating or blunt head trauma. In penetrating trauma, the pathogenesis is usually related to direct arterial injury from projectiles, to bone fragments associated with depressed skull fractures, or to iatrogenic causes (2, 3). In closed head injuries, the mechanism of arterial injury may involve vessel stretching and torsion, or compression against the dura or bony prominences, such as the anterior clinoid process or clivus (9).

Traumatic intracranial aneurysms most commonly involve the anterior circulation. Ventureya (2) et al found that of 131 traumatic aneurysms, the majority involved the anterior cerebral (38%), internal carotid (29%), and middle cerebral (25%) arteries. Only 8% occurred in the posterior circulation (vertebrobasilar system). In a review of 250 traumatic intracranial aneurysms, Quattrocchi et al (6) found that approximately half involved the internal carotid artery and almost half involved branches of the middle and anterior cerebral arteries. Less than

Received March 6, 1996; accepted after revision January 28, 1997.

From the Departments of Radiology (L.A.L., R.W.H., H.I.G.), Neurosurgery (L.S.), and the University of Pennsylvania School of Medicine (T.Y.T.), Hospital of the University of Pennsylvania (Pa).

Address reprint requests to Laurie A. Loevner, MD, Department of Radiology, Neuroradiology Section, Hospital of the University of Pennsylvania, 3400 Spruce St, Philadelphia, PA 19104.



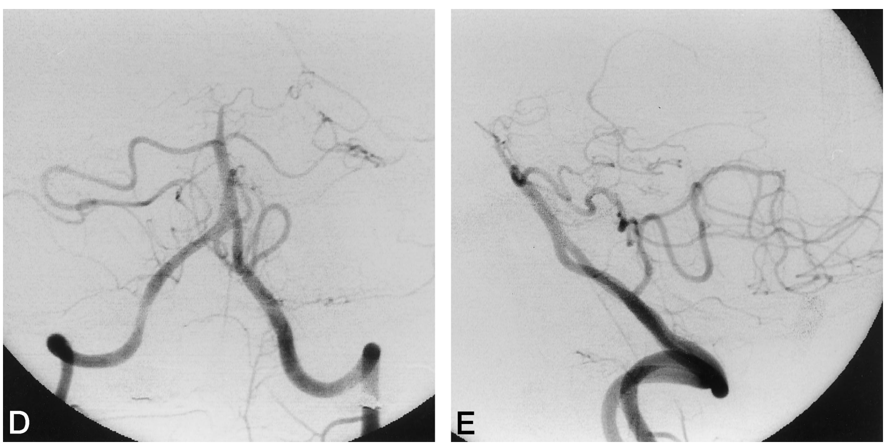


FIG 1. A 7-year-old boy with a left third nerve palsy and a history of head trauma.

A and *B*, Lateral and anteroposterior projections from a subtracted angiogram of the left vertebral artery show a giant aneurysm arising from the narrowed distal basilar artery at the level of the superior cerebellar and posterior cerebral arteries. The aneurysm has an irregular contour and projects lateral to and along the course of the parent vessel (basilar artery).

C, Axial unenhanced head CT scan shows an extraaxial mass in the prepontine/suprasellar cisterns, at the expected location of the distal basilar artery. The mass is heterogeneous with course rim calcification, in particular along the anterior margin. In addition, high attenuation is seen in the posterior portion of the aneurysm, consistent with clot.

D and *E*, Anteroposterior and lateral projections from a digital subtraction arteriogram of the left vertebral artery show opacification of the basilar artery with reflux down the right vertebral artery. The aneurysm seen in the initial angiogram is no longer present, consistent with interval spontaneous thrombosis. There is narrowing of the distal basilar artery. A bilateral carotid arteriogram (not shown) showed large posterior communicating arteries, which provided flow to the posterior cerebral arteries.

10% involved the posterior circulation, with involvement of the basilar artery rare, constituting only 2% of all cases.

Catastrophic rupture of pseudoaneurysms occurs in up to 31% of reported cases (4). Clinical presentation is typically related to subarachnoid, intraparenchymal, or intraventricular hemorrhage (1, 4). Aneurysms of the cavernous internal carotid artery may be accompanied by cranial nerve palsies, ophthalmoplegia, or massive epistaxis (12). Neurologic deterioration may occur over the course of hours, or symptoms may be slowly progressive over weeks, months, or even years (2, 13). Many patients, however, present within several weeks after injury (2). Because of the high risk of rupture, it is imperative that the clinician have a high index of suspicion and consider the diagnosis in the appropriate clinical setting; for example, a history of head trauma (especially penetrating trauma) with neurologic deterioration, progressive cranial nerve deficits, or recurrent epistaxis (2, 4, 13).

There is controversy over the timing of angiography in patients who are thought to have vascular injuries resulting from trauma. Initial angiograms obtained within 1 or 2 days of trauma may be normal; however, follow-up angiograms obtained weeks to months later may reveal an aneurysm (1). This observation prompted recommendations to delay angiography in order to optimize visualization of aneurysms after brain injury (5). However, a recent prospective study comparing the outcome of posttraumatic patients undergoing early and delayed angiography concluded that it was not safe to postpone angiography in patients who were highly likely to have a vascular injury (14). When angiographic findings in the acute period are normal, a repeat study is necessary, given the frequency of delayed aneurysmal formation.

The high mortality associated with rupture necessitates early, definitive intervention. Pseudoaneurysms generally do not have discrete necks and are often friable so that surgical clipping may not be an option. Trapping, in which clips are placed on the parent vessel both proximal and distal to the aneurysm, followed by aneurysmal excision, is a preferred method of treatment. Interventional radiologic techniques, including placement of Guglielmi detachable coils within aneurysms, or occlusion of the parent vessel with detachable balloons or coils for aneurysms located at the skull base or intracavernous aneurysms, have gained acceptance (12). Recently, liquid thrombotic material, such as cellulose acetate polymer, which attaches to irregular vessel walls, has been used successfully to embolize pseudoaneurysms (15).

Complete, spontaneous occlusion of cerebral aneurysms, as occurred in our patient, is relatively rare (1, 11). Fewer than 15% of traumatic cerebral aneurysms heal spontaneously (11). Definitive proof of trauma as the cause of an intracranial aneurysm is often difficult, owing to lack of a pathologic report, as in the present case. However, given our patient's history of significant closed head trauma with a reportedly normal head CT study at the time of injury, the interval development of a calcified giant aneurysm in the prepontine cistern on follow-up CT, and the angiographic appearance, a diagnosis of posttraumatic aneurysm seemed likely.

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