

Case Report

Spontaneous Partial Thrombosis of a Basilar Artery Giant Aneurysm in a Child

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Summary: We report the complete, spontaneous obliteration of a partially thrombosed dissecting giant aneurysm in the basilar artery by occlusion of both the lumen of the aneurysm and the parent artery in a 15-year-old girl.

Giant dissecting aneurysms of the posterior intracranial circulation are a rare finding in the pediatric population. We report a partially thrombosed, giant dissecting basilar artery (BA) aneurysm in a young female patient that spontaneously and asymptotically resolved by complete thrombosis of both the giant aneurysm and the BA. We briefly review the pathophysiology, clinical presentation and MR imaging and angiographic findings associated with partially and completely thrombosed dissecting giant aneurysms.

Case Report

A 15-year-old female patient was admitted to our hospital for angiographic follow-up and possible treatment of a partially thrombosed BA aneurysm extending from the midbasilar portion to the origin of the superior cerebellar arteries (SCAs) that had been discovered at another hospital 11 days before. The patient originally complained of sudden nuchal headaches 1 week before her first hospitalization. During the following week, headaches became intermittent and disappeared without any treatment. Headaches were sometimes associated with nausea, vomiting, and visual changes. No history of previous trauma was reported. Findings of the physical and neurologic examinations performed during the patient's hospital admissions were within normal limits. A cerebral CT scan obtained during the first hospital admission showed an aneurysmal dilatation of the BA with no sign of bleeding. Four-vessel cerebral digital subtraction angiography (DSA) performed 11 days after the CT scan confirmed the presence of the BA aneurysmal dilatation measuring 5 mm in its patent portion with an extension of 2 cm in length (Fig 1). Stenosis of the BA was recognizable proximal and distal to the aneurysm. Both SCAs were injected from the BA. MR imaging performed 1 day later visualized the dissecting basilar aneurysm and small ischemic areas on the left lower part of the pons and in the left cerebral peduncle (Fig 2). The patient was discharged on antiplatelet therapy (ticlopidine 250 mg/dL) to prevent further thrombotic events.

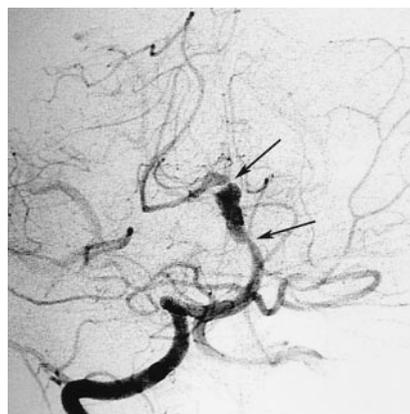


FIG 1. Left vertebral artery angiogram, obtained 11 days after the initial symptoms, demonstrates the presence of the BA aneurysm. At both edges of the aneurysm, stenosis of the BA is recognizable (arrows).

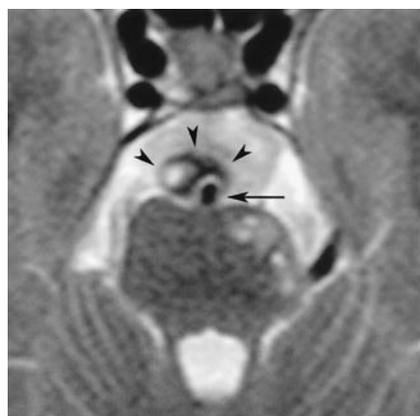


FIG 2. MR image, obtained concurrently with the DSA image, demonstrates the thrombotic component located peripherally (arrowheads). The patent lumen is indicated (arrow). Small ischemic areas on the left side of the pons are also visible.

The stenosis at the distal edge of the BA aneurysm had slightly progressed at the 2-month follow-up cerebral MR imaging with MR angiography (MRA), with an irregular T1 hyperintense bulging consistent with a newly formed clot at the cranial portion of the aneurysm. The antiplatelet therapy was maintained. The 4-month follow-up MR imaging with MRA showed further progression of the BA stenosis. Finally, the follow-up angiogram (Fig 3) obtained 11 months after the initial symptoms demonstrated complete obliteration of the BA at the level of the dissection without any evidence of filling of the aneurysmal pouch and adequate collateral flow to the posterior communicating arteries and SCAs provided by the internal carotid artery via the posterior communicating arteries. MR imaging performed at the same time demonstrated shrinkage of the aneurysm without new lesions. At the present

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FIG 3. Follow-up angiogram, obtained 11 months after the onset of symptoms, shows complete obliteration of the BA at the level of the dissection, without any evidence of residual filling of the aneurysm.

time, the patient remains clinically stable and neurologically intact. The 19-month follow-up MR imaging ruled out the presence of new ischemic areas.

Discussion

Intracranial aneurysms in children account for 0.5–4.6% of all aneurysms (1). In this population, 20% are giant aneurysms. Aneurysms located in the posterior circulation are more numerous in children than in adults (1, 2). Dissecting giant aneurysms of the vertebral and basilar intracranial arteries are relatively rare in the pediatric population. They do, however, carry a severe morbidity and mortality rate because of the tendency to cause distal thromboembolism (3). The mechanisms determining the behavior of partially thrombosed giant aneurysms are not completely understood. These aneurysms probably grow by intramural hemorrhage, as seen on their MR images, which typically show concentric layers of blood in different stages with varying signal intensities (4, 5). Regardless of age or location, the percentage of reported spontaneous total thrombosis of giant aneurysms ranges from 13–20% (6). It has been proposed that thrombosis is not the usual final stage of the aneurysm evolution, but rather an ongoing dynamic process that has potential for further growth and mass effect. Aneurysmal growth may be due to accumulation of thrombotic materials, recurrent intramural hemorrhage, or development of intrathrombotic cap-

illary channels, which may, in turn, thrombose or bleed. Among children, trauma represents the most common predisposing factor for vertebrobasilar (VB) dissecting aneurysms (3). Strokes are quite frequent in children with VB dissecting aneurysms because of secondarily reduced distal flow or embolic phenomena. The angiographic signs of a dissection are not always evident in the acute phase (5); for this reason, when a dissection is suspected, repeat angiography is mandatory, also in light of the frequency of delayed aneurysm formation. The natural history of most untreated giant aneurysms is extremely dismal. The literature contains very few cases of complete spontaneous occlusion of partially thrombosed giant BA aneurysms (1, 7, 8).

In the two cases described in the pediatric population in which spontaneous occlusion occurred, a few similarities with our patient were recognizable (1, 8). First, none of the patients experienced subarachnoid hemorrhage. Second, all of them remained clinically stable or improved during the follow-up period. Third, the aneurysms were located at the same level of the BA, between the midbasilar portion and the origin of the SCAs. Finally, at the diagnostic DSA, tapered narrowing of the parent artery close to the neck of the aneurysm was demonstrated in all three patients. In our opinion, this condition may have altered the dynamic of the jet stream of blood, promoting the progression of the thrombosis of both the aneurysm and the parent artery.

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