Spontaneous thrombosis of posterior cerebral artery aneurysm with angiographic reappearance

Case report

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The case is presented of a 23-year-old man suffering ischemic brain infarction from spontaneous thrombosis of a left posterior cerebral artery P1-P2 junction aneurysm. Vasospasm and/or partial parent vessel occlusion were documented by magnetic resonance (MR) imaging and angiography. Repeat cerebral angiography and MR imaging 3 months later revealed patency of the posterior cerebral artery and luminal filling of a 1-cm fusiform aneurysm, which was successfully trapped at surgery.

Key words: cerebral angiography • thrombosed aneurysm • aneurysm recurrence

Thrombosis of intracranial arterial aneurysms occasionally occurs, but thrombosis of the parent vessel is uncommon.12,18 The disappearance of aneurysms previously defined by angiography is also uncommon.4,5 However, the spontaneous angiographic disappearance followed by angiographic reappearance of an aneurysm is rare, and we could only identify six such cases in the literature.6,7,11,14,16 In those cases the aneurysm was presumed thrombosed by nonfilling at angiography; however, there may be other reasons for aneurysm nonvisualization.10 We present what we believe to be the first case report documenting aneurysm thrombosis by magnetic resonance (MR) imaging and angiography, followed by aneurysmal filling also documented by MR imaging and angiography.

Case report

This 23-year-old right-handed man, 6 weeks prior to admission, developed a continuous, severe left-sided throbbing headache lasting approximately 10 days. The headache improved over several weeks but gradually worsened again; it was most severe when he was erect. He did not seek medical attention. The evening prior to admission he retired early because of the severity of his headache and awoke at 3 a.m. with an extremely severe left-sided throbbing headache, followed by nausea and emesis. He was taken to the emergency room where he was found to be lethargic and confused.

Examination. Examination revealed a stuporous young man with significant attention difficulties and short-term memory deficits. He exhibited a dense right homonymous hemianopsia and a mild right hemiparesis. Computerized tomography (CT) showed a 1-cm high-density lesion in the left anterior perimesencephalic cistern without evidence of subarachnoid hemorrhage (SAH). Lumbar puncture revealed normal opening pressure and no evidence of hemorrhage. On MR imaging an acutely thrombosed 1-cm posterior cerebral artery (PCA) aneurysm was seen at the P1-P2 junction and a left occipital lobe infarction was visualized (Fig. 1). Repeat lumbar puncture on Day 2 again revealed no evidence of SAH. The patient was stabilized and treated with volume expansion and his level of consciousness improved. Two weeks after the ictus, he underwent cerebral angiography, revealing segmental narrowing of the P1-P2 junction and nonfilling of the aneurysm previously shown on MR imaging to be acutely thrombosed (Fig. 2). He was discharged from the hospital with a complete, dense, right-sided homonymous hemianopsia, attention difficulties, mild short-term memory deficits, and a mild right-sided hemiparesis.

Follow-up MR angiography 3 months after the ictus revealed a patent left PCA and filling of the aneurysm (Fig. 3 left). The patient was readmitted to the hospital and repeat angiography revealed a narrowed but patent PCA and 6-mm filling of the 10-mm aneurysm without
Spontaneous thrombosis, then recurrence of aneurysm

Fig. 1. Magnetic resonance images obtained 6 hours postictus. Left: T1-weighted sagittal image demonstrating a spherical mass (arrow) just superior to the left posterior clinoid. The mass is isointense with adjacent brain and consistent with acute thrombus (deoxyhemoglobin). Right: T2-weighted axial image demonstrating a markedly hypointense signal within the thrombosed aneurysm (arrow) and confirming the presence of acute clot (deoxyhemoglobin). Note changes of acute infarction in the distribution of the left posterior cerebral artery.

an identifiable neck (Fig. 3 right). He subsequently underwent surgery.

Operation. A left pterional craniotomy and transsylvian fissure approach was used. The aneurysm was identified and a small posterior communicating artery defined. This was divided at the aneurysm distal to a

Fig. 2. Selected left vertebral angiogram obtained 2 weeks postictus demonstrating marked segmental narrowing of the P2 segment of the left posterior cerebral artery (arrows) without opacification of the aneurysm.

Fig. 3. Left: Magnetic resonance angiogram obtained 3 months after admission using the time-of-flight technique. Flow is demonstrated within the left posterior cerebral artery and aneurysm (arrow). No neck was identified. Right: Selected left vertebral artery angiogram obtained at the same examination with filling of the 1-cm fusiform aneurysm (arrow) and distal opacification of the left posterior cerebral artery. No aneurysm neck was identified.
leash of anterior thalamoperforating vessels between two 2-mm Sundt-Kees No. 2 ultramicroclips (Fig. 4). A large single-trunk candelabra-shaped posterior thalamoperforating artery was dissected from the medial fundus wall of the aneurysm and a straight 6-mm Sundt-Kees aneurysm clip was placed between the aneurysm and the posterior thalamoperforating trunk. The aneurysm softened considerably so it could be dissected; it was found to be fusiform without an identifiable neck. Because of the small working area, the aneurysm was trapped by placing a slim 6-mm Sundt-Kees miniclip immediately distal to it. The aneurysm was then incised and decompressed. No filling of the exenterated aneurysm from posterior-wall perforating vessels could be identified. No revascularization via bypass grafting or vessel lumen repair via aneurysmal reconstruction was attempted as the patient had suffered a complete infarction in the territory of the vessel entrapment.

**Postoperative Course.** Postoperatively, the patient suffered a transient left oculomotor nerve palsy which resolved over several months. Temporal lobe seizures occurred, which were controlled with carbamazepine. His previous right-sided homonymous hemianopia, mild right-sided hemiparesis, attention difficulties, and mild short-term memory deficits remained unchanged. He was discharged from the hospital and returned to rehabilitation treatment.

**Discussion**

Some degree of spontaneous thrombosis is identified in 9% to 13% of intracranial aneurysms found at autopsy,\(^9\)\(^1\)\(^3\) and partial or complete thrombosis within giant aneurysms is well known.\(^1\) It is suggested that 1% or 2% of ruptured aneurysms will show spontaneous and complete thrombosis on subsequent angiography.\(^5\) Spontaneous disappearance of ruptured aneurysms on repeat angiography was found by Fodstad and Lilequist\(^1\) in 3% of patients with SAH who received antifibrinolytic therapy; however, Dávila, et al.,\(^4\) found only 12 cases documented prior to 1983 of angiographically confirmed spontaneous thrombosis of aneurysms that were previously patent on angiography and not surgically treated. Only 8% of patients in the latter review had received antifibrinolytic therapy, and in 38% the parent vessel was not visualized and presumed thrombosed as well. However, spontaneous thrombosis of aneurysms may be transient; rupture with catastrophic consequences may occur, and parent vessel occlusion or embolization have also been documented.\(^1\)\(^2\)\(^7\)\(^17\)

In our literature search, we were able to find only six cases of angiographic reappearance of a known aneurysm that had previously not filled on angiography and was presumed thrombosed.\(^5\)\(^7\)\(^11\)\(^14\)\(^16\) Patients presenting with SAH are suspected of harboring aneurysms based on the CT appearance, location, and volume of the hemorrhage. Approximately 20% of patients with SAH have negative angiograms; this lack of angiographic demonstration may be due to other vascular etiologies, technical errors, vasospasm, transient thrombosis, or a combination of these.\(^10\) The subsequent appearance of an aneurysm in such patients after an initial negative study is rare; although they are suspected of having an aneurysm, it is not proved.\(^7\) If an aneurysm is revealed in these patients, the most frequent correlation is for anterior communicating artery aneurysms and a large amount of interhemispheric blood on CT scan.\(^10\)

Our case differs from the previous six cases in that the latter were proved angiographically to have a patent aneurysmal lumen, followed by subsequent angiographic nonvisualization, then angiographic patency. However, these aneurysms may be presumed only transiently thrombosed. In our case, thrombosis was proved transient, with MR images demonstrating acute clot within the aneurysm, then nonvisualization at angiography and subsequent MR imaging and angiographic depiction of luminal filling. This case emphatically supports periodic follow-up review of patients with known aneurysms that for whatever reasons are not surgically excluded from the circulation. The natural history of aneurysms that spontaneously thrombose is unknown; however, it is known that spontaneously thrombosed aneurysms\(^17\) or aneurysms occluded via endovascular techniques\(^6\) may subsequently rupture. Continued follow-up monitoring of thrombosed aneurysms will further add to information on their natural history and aid in their overall management.

**References**

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