

# CEREBRAL ANEURYSM THROMBOSIS, SHRINKAGE, THEN DISAPPEARANCE AFTER SUBARACHNOID HEMORRHAGE

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The case is presented of a 37-year-old man with spontaneous thrombosis of a 10-mm left posterior communicating artery aneurysm, following presumed subarachnoid hemorrhage with negative angiography. Sequential magnetic resonance angiography and magnetic resonance source imaging (at 2 weeks, 3 months, 2 years, and 2 years, 5 months) showed progressive shrinkage and disappearance of the aneurysm.

## KEY WORDS

*Cerebral aneurysm, thrombosis magnetic resonance imaging, angiography, subarachnoid hemorrhage, sequential studies.*

Spontaneous thrombosis of an intracranial aneurysm is an infrequent event [1,2,4-7,11,13,14,17,18]. We report an unusual case of posterior communicating artery aneurysm thrombosis following presumed subarachnoid hemorrhage with subsequent shrinkage and then disappearance of the aneurysm on serial magnetic resonance angiography (MRA) and magnetic resonance imaging (MRI) examinations over a 2½-year follow-up period.

## CASE REPORT

A 37-year-old man complained of a dull ache in the left temporal and posterior ocular area initially felt to represent tension-type headache. He had a history of similar episodes 16 years earlier while under stress in law school. The next day, the headache became incapacitating, to the extent that the patient locked himself in a dark quiet room for 3 days. Seven days later, the patient returned to the clinic

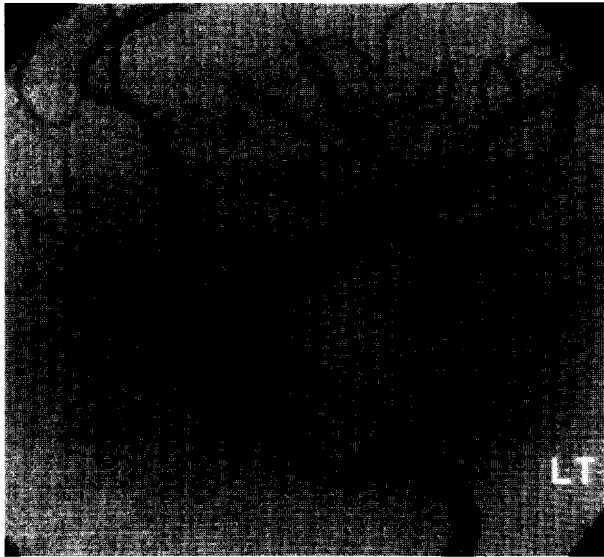
complaining of less severe, but persistent headache. He was seen by a neurologist and referred for MRI. Neurologic exam was negative at that time. MRI performed 3 days later showed a 10-mm left posterior communicating artery aneurysm with bright signal on T<sub>1</sub> weighted precontrast images, suggesting either slow flow or thrombosis. MRA was not available at that time. A three-vessel cerebral angiogram (Figure 1) with cut film and digital subtraction was performed, including multiple oblique magnification views. No evidence of a cerebral aneurysm, nor of vasospasm, was seen. The patient elected to have the aneurysm followed by MRA, and was referred to a sister institution for baseline study. A three-dimensional (3-D) phase contrast MRA showed no evidence of flow within an aneurysm (Figure 2 A). Three-dimensional time-of-flight MRA performed at the same time did show bright signal in the aneurysm, consistent with recent thrombosis (Figure 2 B-C). Repeat MRA 3 months later showed the left posterior communicating artery aneurysm to have reduced in size to 6 mm, with no evidence of flow within the aneurysm (Figure 3 A-B). The 3-D time-of-flight MRA source image which had previously shown bright signal within the aneurysm now showed dark signal consistent with thrombus evolution (Figure 3 B).

Repeat MRA 2 years after the initial presentation showed no evidence of an aneurysm. T<sub>1</sub> weighted spin-echo MRI showed a tiny residual nubbins adjacent to the left posterior communicating artery origin, with no evidence of a flow void. Magnetic source imaging (MSI) performed with magnetoencephalography showed diffuse spontaneous slow-wave activity [12,13] over the left hemisphere, indicative of possible cortical dysfunction.

Repeat MRA at 2 years and 5 months after the ictus showed no evidence of an aneurysm on 3-D

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**1** Left internal carotid arteriogram. Left anterior oblique projection.

time-of-flight (Figure 4 A) nor on 3-D phase-contrast MRA, and showed further disappearance of the aneurysm on the source images (Figure 4 B). The patient has continued to do well 3 years and 2 months after the aneurysm was initially discovered, but he does have occasional mild left-sided headaches. He does not wish to undergo repeat conventional angiography.

## DISCUSSION

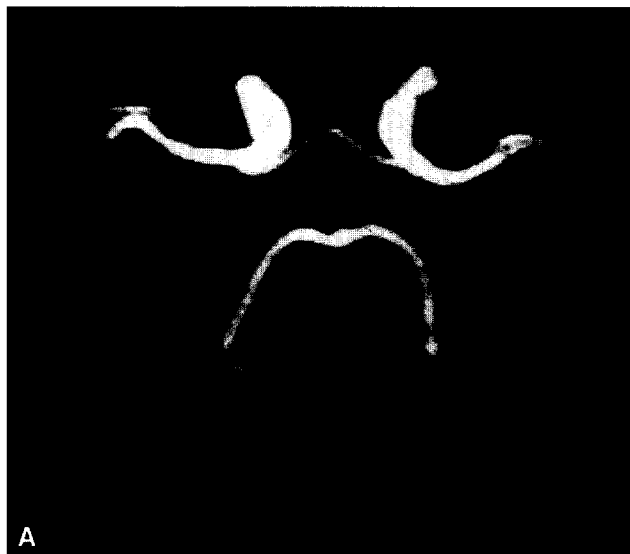
The reported incidence of complete thrombosis of an intracranial aneurysm following a subarachnoid hemorrhage varies widely [1,2,4-8,11,13]. In 1944, Dandy stated, "It is impossible to more than guess at the percentage of certain seeming cerebral hemorrhages of this origin that are permanently cured by thrombosis. My guess would be that it is not more than 15% or 20%" [4]. He also stated that one must look upon so-called cures with considerable skepticism, since delayed rupture may occur many years later. Autopsy studies have reported spontaneous thrombosis of ruptured intracranial aneurysms to occur in 9%-13% of patients [2,13]. Although our patient did not have documentation of subarachnoid hemorrhage by either lumbar puncture or computed tomography, his clinical symptoms strongly suggested its occurrence. Edner [6] found that 1 of 78 ruptured aneurysms visualized initially were not visualized at subsequent angiography due to thrombosis. Davila [5] reviewed the literature prior to 1984 and found only 12 cases of spontaneous thrombosis of ruptured aneurysms

that were initially patent at angiography, but failed to visualize at subsequent angiographic study. Only 7 of these 12 cases had patent parent arteries. Fodstad [7] reported 3 cases of complete aneurysm thrombosis out of 100 cases treated with antifibrinolytic therapy for subarachnoid hemorrhage. In 1 of these 3 cases, aneurysm patency reappeared 1 month later. In 1992, Hamilton [11] reported a patient with a ruptured 5-mm anterior communicating artery aneurysm that was not repaired initially due to the patient's poor clinical status. Repeat angiography 2 years later, when the patient requested surgery, was negative. Kumar [14] described a patient with an 8-mm posterior cerebral-posterior communicating artery junction aneurysm that was shown by initial angiography 16 days following subarachnoid hemorrhage. Repeat angiography at 30, 40, and 60 days failed to demonstrate the aneurysm.

Failure to visualize an aneurysm by conventional angiography may be due to either vasospasm or faulty technique in addition to complete aneurysm thrombosis. Furthermore, if an aneurysm is thrombosed at angiography, does this constitute a cure? Nishioka [15] reported 13 of 72 patients with normal three-vessel angiography who were found to have an aneurysm at subsequent angiographic study, whereas Forster [8] found an aneurysm in 1 of 56 patients on a second angiogram not seen on initial study. Spetzler [17] reported a patient with severe vasospasm who received antifibrinolytic therapy for a left frontopolar artery aneurysm that was visualized on initial angiography, disappeared, and then reappeared on repeat angiographic study 21 days later. Atkinson [1] found 6 cases in the literature of aneurysms that were initially shown angiographically to have a patent lumen followed by non-visualization, and then revisualization on serial angiographic follow-up studies. He reported an additional case without subarachnoid hemorrhage of a left posterior cerebral P1-P2 junction aneurysm associated with left occipital lobe infarction. Magnetic resonance imaging at 2 weeks after the ictus showed acute aneurysm thrombosis, and angiography showed nonfilling of the aneurysm. Both repeat MRI and conventional angiography 3 months later showed a patient aneurysm lumen.

Our case differs from those previously reported in that not only was the aneurysm not visualized at conventional angiography, but serial MRA and MRI studies over 2½ years showed progressive shrinkage and then disappearance of the aneurysm. Continued long-term follow-up will be necessary to determine if this represents a complete cure.

The MSI finding of spontaneous slow-wave activity in our patient on the same side of the aneurysm



**2** MRA 2 weeks after presentation. Three-dimensional phase-contrast MRA does not show the aneurysm due to the absence of flow (A, *axial*). Three-dimensional time-of-flight MRA shows bright signal (*white arrow*) within the left posterior communicating artery aneurysm due to recent thrombosis (B). Source image from 3-D time of flight MRA shows a 10-mm aneurysm with bright signal (C, *white arrow*).

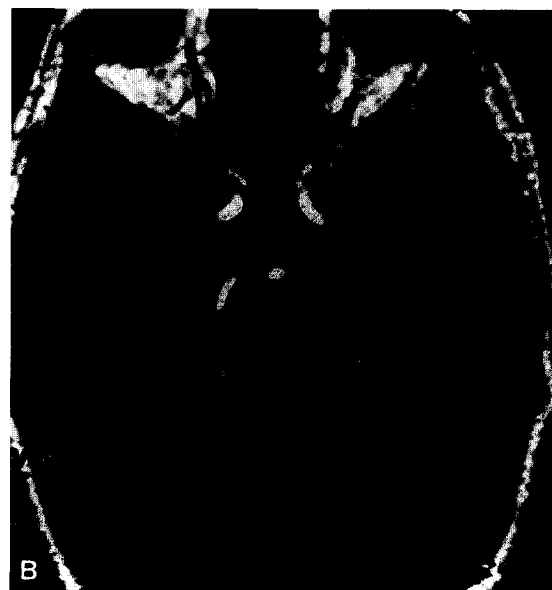
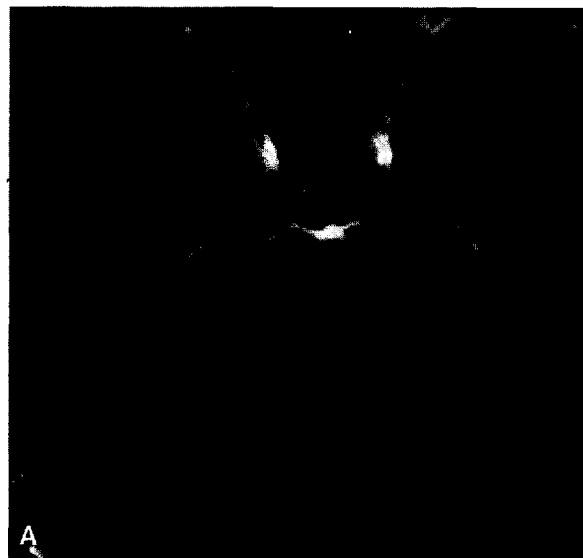
is of interest. We hypothesize that this represents mild cortical dysfunction related to the presence of subarachnoid hemorrhage and associated meningeal irritation and/or vasospasm present prior to performing the cerebral angiogram. No evidence of left hemispheric infarction was seen at MRI. We have not observed similar findings in patients with migraine headaches. Further MSI studies of additional patients with subarachnoid hemorrhage will be necessary to determine the significance of this finding.

Finally, this case raises the question: how sensi-

tive is MRA in detecting cerebral aneurysms? Ross [16] reported an 86% sensitivity in detecting cerebral aneurysms greater than 3 mm in size when combining MRA and spin-echo MRI as compared to intraarterial digital subtraction angiography. Curnes [3] reported an unusual case of a ruptured 4-mm posterior communicating artery aneurysm in which conventional angiography was negative, 3-D phase contrast MRA performed immediately after the angiogram showed a patent aneurysm, and repeat conventional angiography 7 days later confirmed the aneurysm. MRA is insensitive to aneurysms less



**3** MRA 3 months after presentation. Neither 3-D phase contrast MRA (not shown) nor 3-D time-of-flight MRA (A) showed evidence of an aneurysm. Magnetic resonance source image from the time-of-flight MRA now shows the left posterior communicating artery aneurysm to be reduced in size to 6 mm and to contain dark signal due to thrombus evolution (B, white arrow).



**4** MRA and magnetic resonance source image, 2 years and 5 months after presentation. Axial 3-D time-of-flight MRA through the circle of Willis (A) and axial magnetic resonance spoiled grass (B) source image both show no evidence of a posterior communicating artery aneurysm.

than 3 mm in size and should be used only as a screening procedure [12]. In the presence of subarachnoid hemorrhage, catheter angiography remains the diagnostic procedure of choice.

In conclusion, we report a case of complete spontaneous thrombosis following presumed subarachnoid hemorrhage, with subsequent shrinkage, and then complete disappearance on serial MRA studies.

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