Case Report

Cerebral Aneurysms Associated with Seizures but Without Clinical Signs of Rupture: Seemingly Distinctive MR Imaging Findings in Two Patients

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ntracranial aneurysms typically have a clinical presentation of symptoms related to subarachnoid hemorrhage. Less commonly, aneurysms can present with headache in the absence of aneurysm rupture or cranial neuropathy secondary to cranial nerve compression. Because seizures are an uncommon presenting feature of intracranial aneurysms, aneurysm is generally not considered in the differential diagnosis of lesions causing seizures. Aneurysms can produce seizures by a number of mechanisms, including compression or infarction of adjacent brain tissue as well as irritation by blood products from subclinical hemorrhage [1-3].

We present the MR imaging findings in two patients with cerebral aneurysms and seizures in the absence of clinical features of aneurysm rupture. In both cases, we saw abnormal perianeurysmal MR signal within the adjacent temporal lobe, and the MR signal abnormality was associated with findings at surgery of gliotic brain tissue. In one of these patients, who had thrombus found outside the aneurysm at surgery, the abnormal MR signal also indicated subclinical leakage of the aneurysm. We therefore suggest that

the finding of perianeurysmal MR signal abnormality should raise the possibility that an aneurysm has caused structural changes in the adjacent brain and serve as evidence to support the role of an aneurysm in producing seizures in individual patients.

Case Report

A 65-year-old man in good health awakened from sleep and experienced a generalized seizure. Neurologic examination a few hours later had nonfocal findings. MR imaging performed the same day showed a small rounded region of hypointense signal within the medial left temporal lobe on all pulse sequences, consistent with the lumen of an aneurysm (Figs. 1A and 1B). On T1-weighted and T2weighted images, abnormal signal was seen in a circumferential pattern around the periphery of the aneurysm lumen, consistent with partial thrombosis of the aneurysm. Also, we noted a more peripheral and irregularly marginated region of abnormal signal that hypointense on T1-weighted images hyperintense on T2-weighted images. This region extended through much of the temporal lobe (Figs. 1A and 1B). MR angiography showed the aneurysm to project laterally from the supraclinoid segment of the left internal carotid artery (Fig. 1C). This projection was later confirmed by catheter angiography.

The patient underwent aneurysm clipping the next day by a pterional approach. A 1-cm aneurysm was found. No evidence of hemorrhagic products was found within the surrounding subarachnoid space or adjacent brain to indicate prior rupture. The medial left temporal lobe was seen to have a yellowish hue and abnormally firm texture secondary to gliosis, which we supposed to be a reaction to the aneurysm. We thought the gliosis most likely was the cause of abnormal signal within the temporal lobe on MR imaging. The patient, who was placed on anticonvulsants at the time of hospital admission, has experienced no seizures during a 6month follow-up.

Similar findings of abnormal perianeurysmal MR signal were found in another patient with seizures that were associated with a cerebral aneurysm (Fig. 2).

Discussion

Seizures are an uncommon, but not rare, presenting feature of intracranial aneurysms.

Received November 2, 1995; accepted after revision December 7, 1995.

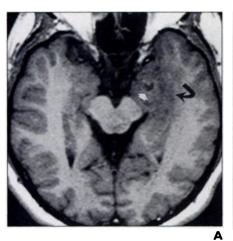
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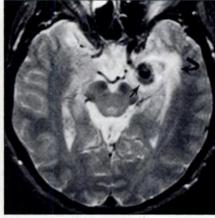
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Cerebral Aneurysms Associated with Seizures





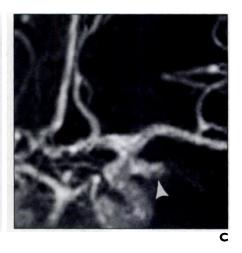


Fig. 1.—MR imaging in 65-year-old man who had recently suffered first seizure.

A, Unenhanced T1-weighted axial image (500/11/1 [TR/TE/excitations]) shows rounded region of hypointense signal representing patent lumen of aneurysm (straight arrow). Extensive region of abnormal signal (curved arrow) extends through much of left temporal lobe.

B, T2-weighted axial image (2500/80/0.75) shows lumen of aneurysm surrounded by rim of abnormal signal (*arrow*) that is hypointense to brain, representing intraaneurysmal thrombus. At surgery, actual size of aneurysm correlated with external diameter of rim of hypointense signal. That correlation was taken as evidence that aneurysm was, indeed, partially thrombosed. Hyperintense signal abnormality (*curved arrow*) extends through much of medial left temporal lobe, which was correlated with gliosis at surgery.

C, Three-dimensional time-of-flight MR angiogram (48/5.6/1; flip angle, 30°) shows aneurysm (arrowhead) arising from supraclinoid segment of left internal carotid artery and projecting laterally.

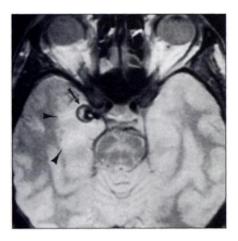


Fig. 2.—MR imaging study of 39-year-old man who suffered olfactory hallucinations due to temporal lobe seizures and had long-standing history of right-sided headache. Long TR/short TE axial image (2500/30/1) shows partially thrombosed aneurysm (arrow) with hyperintense signal within adjacent mesial temporal lobe (arrowheads). At surgery, 1.2-cm posterior communicating artery aneurysm that was densely adherent to medial right temporal lobe was found. Organized thrombus was also seen outside dome of aneurysm in right temporal lobe, indicating previous subclinical aneurysm leak. Moderate gliosis was also noted at surgery in part of temporal lobe that exhibited abnormal signal. Patient experienced no further seizures following surgery.

The strongest association appears to be between complex partial seizures and aneurysms of the middle cerebral artery because of the proximity of such aneurysms to the temporal lobe [1]. The likelihood of an aneurysm's causing seizures appears to increase with the size of the aneurysm. In one series of patients with unruptured intracranial aneurysms, 5% of patients presented with seizures and all had aneurysms larger than 1 cm [4]. Seizures can precede discovery of the aneurysm by many years, especially giant (>2.5 cm) aneurysms that often come to clinical attention because of mass effect rather than aneurysmal rupture [1].

Perianeurysmal hyperintense signal on T2weighted images has previously been reported and attributed to edema [5]. The findings at surgery in our patients indicate that structural changes within the mesial temporal lobe may be responsible for the signal abnormality. The cause of the temporal lobe seizures in our second patient can clearly and reasonably be attributed to right mesial temporal lobe gliosis and, perhaps, the irritative effects of subarachnoid blood from previous subclinical rupture of an aneurysm. Also, the seizure in our first patient is most likely related to temporal lobe gliosis found at surgery, with the tonic-clonic seizure probably having begun as a complex partial seizure that secondarily generalized, a relatively common event. Various findings at surgery have been reported in patients with aneurysms that presented as seizures, including temporal lobe compression; occasional perianeurysmal blood products indicative of subclinical hemorrhage [1]; and, in one case in which amygdalohippocampectomy was performed, severe neuronal loss and gliosis [2].

To further assess the importance of abnormal perianeurysmal MR signal, we reviewed the MR imaging examinations of 32 other patients with aneurysms (who had a total of 43 aneurysms) seen over a 5-year period at our institution. We found abnormal signal within brain parenchyma in two patients, both of whom had clinical and MR imaging findings of aneurysmal rupture. In one patient, hyperintense signal on T2-weighted images was seen within the medial temporal lobe adjacent to a superior cerebellar artery aneurysm in a pattern similar to our two other cases. Another patient, who had a temporal lobe hemorrhage that was caused by rupture of an aneurysm at the bifurcation of the middle cerebral artery, had abnormal signal adjacent to the hemorrhage but none around the portion of the aneurysm that was not surrounded by hemorrhage. Neither patient had seizures. None of the 41 unruptured aneurysms in this group of patients had adjacent abnormal MR signal. Therefore, of a total of 43 unruptured aneurysms seen on MR imaging studies (including the two illustrated earlier), only two had abnormal peri-

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aneurysmal MR signal, and both were associated with seizures.

We suggest that the MR imaging examinations of patients with seizures and a cerebral aneurysm should be carefully inspected for abnormal perianeurysmal MR signal, which may indicate that the aneurysm is not an incidental finding but related to the cause of the seizures. However, the true significance of abnormal perianeurysmal MR signal will become apparent only when a larger number of cases have been analyzed. For instance, it is possible that patients with abnormal perianeurysmal MR signal have a risk of aneurysmal rupture different from that of patients with

aneurysms that lack such abnormality. The frequency with which abnormal perianeurysmal MR signal is correlated with an earlier subclinical aneurysmal rupture will be an important link. Patients with abnormal perianeurysmal MR signal may also suffer seizures at a different frequency than in patients lacking such abnormality, in which case this MR finding might be an indication for earlier, or more aggressive, anticonvulsant therapy.

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