Minimally Conscious State After Ruptured Giant Basilar Aneurysm

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Objective: To report the clinical and radiologic findings in a case of transient minimally conscious state after rupture and coiling of a giant basilar aneurysm.

Design: Case report.

Setting: Neuroscience intensive care unit.

Patient: A 44-year-old man who developed a transient minimally conscious state in association with perianeurysmal edema in the rostral brainstem and thalamus after rupture and coiling of a giant basilar artery aneurysm.

Main Outcome Measure: Correlation of clinical and magnetic resonance imaging findings.

Results: A minimally conscious state and bilaterally symmetric vasogenic edema of the rostral brainstem and thalamus developed 2 days after endovascular aneurysm coiling. The clinical and radiologic abnormalities improved significantly and in parallel during the following 4 weeks.

Conclusions: Perianeurysmal vasogenic edema in the brainstem and thalamus can develop after rupture and coiling of a giant basilar artery aneurysm. This process can be transient and can produce dramatic alterations in consciousness that later resolve.

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Impaired consciousness soon after rupture of an intracranial aneurysm is usually due to global anoxic ischemic hemispheric injury, acute hydrocephalus, or intraparenchymal hematoma with mass effect.1,2 Perianeurysmal cerebral edema has been associated with thrombosis of large aneurysms, both spontaneous thrombosis and thrombosis induced by endovascular aneurysm occlusion. This phenomenon has been associated with a variety of neurologic deficits3-6 but, to our knowledge, has been described as a cause of impairment of consciousness in only 1 other case.3 We report a case of resolving minimally conscious state associated with extensive edema in the thalamus and upper brainstem in a patient with a ruptured giant basilar artery aneurysm.

A 44-year-old man was admitted to the neuroscience intensive care unit with subarachnoid hemorrhage due to the rupture of a giant basilar terminus aneurysm. On admission he followed simple commands, had a complete left third nerve palsy, exhibited severe dysarthria, and showed no evidence of hemiparesis. The next day he underwent a cerebral angiogram that demonstrated a basilar terminus aneurysm measuring 3 × 3 × 3 cm (Figure, B). This aneurysm was nearly completely embolized with 17 bare platinum helical coils. Two days later he became stuporous and anarthric and developed a complete right third nerve palsy. Magnetic resonance imaging at this time demonstrated bilaterally symmetric, extensive edema without restricted diffusion, located primarily in the gray matter and extending from the midpons rostrally to the inferior thalamus (Figure, C and D). The aneurysm had not changed in size and there was no hydrocephalus. Intravenous dexamethasone, 4 mg every 8 hours, was administered with coincident mild improvement in his level of arousal for 2 days. Thereafter, his neurologic function again declined. By hospital day 9, he was stuporous and anarthric, rarely followed commands, had bilateral ophthalmoplegia and mydriasis, and was markedly bradykinetic. Slow improvement occurred during the subse-
sequent 3 weeks. On discharge on hospital day 28, he was fully alert and oriented, had only a partial left third nerve palsy, and was mildly dysarthric and bradykinetic. Improvement of the brainstem and thalamic edema paralleled the clinical course and, at the time of discharge, only minimal edema in the tegmentum of the pons and mid-brain remained (Figure, E and F).

**COMMENT**

The source of perianeurysmal cerebral edema in our patient is uncertain. In previous cases, perianeurysmal edema has been attributed to expansion of the aneurysm at the time of rupture or coiling with resultant mass effect, occlusion of nearby penetrating vessels due to intra-aneurysmal thrombus formation, compression of venous drainage in the region surrounding the aneurysm, or a local inflammatory response due to manipulation associated with endovascular coiling. The unusually high number of platinum coils needed to occlude this patient’s aneurysm did not cause apparent expansion of the aneurysmal dome, suggesting that mechanisms other than local mass effect may have been responsible. We cannot be certain on this point, however, because even an increase in the diameter of the aneurysm that is too small to be apparent on the follow-up magnetic resonance images could lead to a significant increase in its volume.

It is remarkable that our patient developed a minimally conscious state that completely resolved, perhaps facilitated by the use of corticosteroids. Improvement of perianeurysmal edema with corticosteroid treatment has been reported in similar cases, although spontaneous improvement may occur. One case of persistent akinetic mutism has been described in a patient with edema in the midbrain and thalamus after coiling of a giant superior cerebellar artery aneurysm. Unlike our patient, the condition of the patient described in that case did not improve despite the use of corticosteroids and a decrease in surrounding edema. The minimally conscious state is typically a result of diffuse bihemispheric injury, but in our case it could be specifically attributed to a lesion in the thalamus and tegmentum of the pons and mid-brain. The presence of vasogenic and not cytotoxic edema on repeated magnetic resonance images may have predicted the reversible nature of this process.

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**Figure.** Magnetic resonance images in a 44-year-old man after the rupture and coiling of a giant basilar artery aneurysm. Computed tomography was performed before coiling (A), and an angiogram was obtained after (B). Fluid-attenuated inversion recovery (FLAIR) images demonstrated extensive edema in the pontine tegmentum (C) and thalami (D) 3 days after admission. Before discharge on day 28, FLAIR images showed resolution of the edema in the pons (E) and thalami (F).
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REFERENCES


Announcement

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For details about this new policy, and for information on how the ICMJE defines a clinical trial, see the editorial by DeAngelis et al in the January issue of *Archives of Dermatology* (2005;141:76-77). Also see the Instructions to Authors on our Web site: www.archneurol.com.