Acute deterioration from thrombosis and rerupture of a giant intracranial aneurysm

Article abstract—The authors describe a patient with an unusual clinicopathologic picture of giant aneurysmal hemorrhage followed by sudden deterioration due to acute intra-aneurysmal thrombosis and fatal rebleeding. This patient underscores the poor natural history associated with this devastating disease and serves to highlight the dangers inherent in the delayed treatment of these life-threatening lesions.

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Clinical deterioration in patients harboring intracranial aneurysms is a well-recognized phenomenon.[1-3] Several reports suggest that the risks of initial rupture, thrombosis, and rebleeding of giant intracranial aneurysms are comparable with if not greater than those associated with smaller aneurysms, and the poorer overall outcome associated with giant aneurysms is widely recognized.[4-8] We report a woman with a subarachnoid hemorrhage (SAH) from a ruptured giant intracranial aneurysm who suddenly deteriorated from acute intra-aneurysmal thrombosis followed by fatal aneurysmal rebleeding documented by CT and pathologic examination. This pattern of deterioration represents a previously undescribed clinical course among patients presenting with ruptured intracranial aneurysms.

Case report. A 53-year-old right-handed woman developed an acute-onset, severe headache that was not associated with altered level of consciousness or focal neurologic deficit. She had no history of previous SAH or "warning headache." Her medical history was unremarkable except for borderline hypertension, mild hypercholesterolemia, and a 25-pack-year history of cigarette smoking.

Initial clinical evaluation. The patient was taken immediately to the local emergency room where she was found to be alert and stable hemodynamically. Her Glasgow Coma Scale score was 15 points, and despite her ongoing, severe left temporal headache, there was no neck stiffness or focal sensorimotor deficit (World Federation of Neurologic Surgeons [WFNS] grade I). CT demonstrated a giant left middle cerebral artery (MCA) aneurysm (figure 1). She was transferred via air ambulance within 24 hours of her hemorrhage (posthemorrhage day 0 [PHD 0]) to our hospital for further evaluation and treatment.

Clinical course. At the time of admission to our hospital (PHD 1), she was mildly aphasic and obtunded, with an occipital headache and mild neck stiffness but otherwise was without focal neurologic deficit (WFNS grade III). On the morning of PHD 2, the patient was alert and orientated and reported no headache; her neurologic examination was normal (WFNS grade I). By the afternoon of PHD 4, the patient had reported a steadily worsening occipital headache over a period of a few hours; she was, however, alert and there was no associated nausea, vomiting, fever, or neurologic deficit (WFNS grade I). On the morning of PHD 5, the patient suddenly deteriorated, becoming globally aphasic with a dense, right flaccid hemiplegia and Babinski sign (WFNS grade IV). Repeat brain CT demonstrated acute thrombus formation within the aneurysm but no evidence of rebleeding. Within 10 hours of her initial deterioration on PHD 5, the patient acutely manifested further deterioration, becoming hypertensive and bradycardic, withdrawing only to pain, and was found to have dilatation of her left pupil (WFNS grade V). Repeat CT (figure 2) demonstrated aneurysmal rebleeding. She rapidly developed uncal herniation and died.

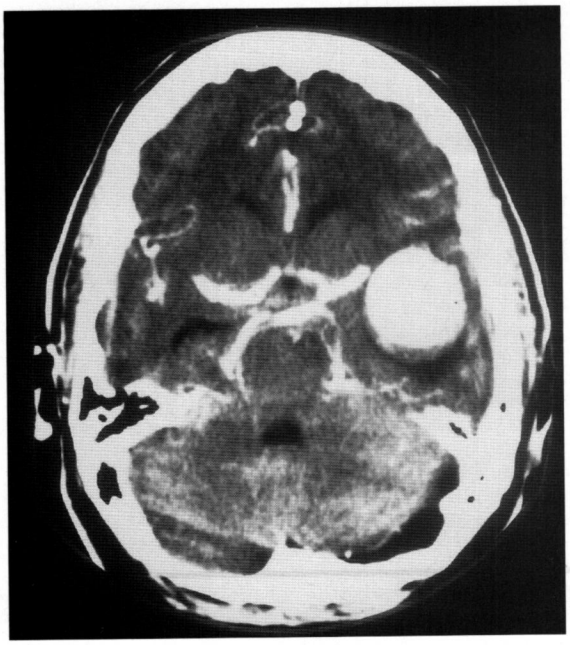

Figure 1. Contrast CT performed on the day of initial hemorrhage demonstrates Fisher grade I subarachnoid hemorrhage from a giant left middle cerebral artery aneurysm containing a thin rim of thrombus along the dorsal aspect of its dome. There was no evidence of hydrocephalus.

Pathologic examination. A giant atherosclerotic aneurysm, measuring 3.6 × 3.5 × 3.0 cm, originated from the left MCA bifurcation and was filled with premortem thrombotic material (coagulation thrombus; figure 3), except for a thin peripheral crescent of radiopaque barium gelatin mixture injected via a vertebral artery for the purpose of postmortem cerebral angiography. Microscopic examination of the aneurysm revealed a variably thin wall comprised mainly of collagen, with sparse remnants of internal elastic membrane and medial smooth muscle. Focal distortion of the wall by deposits of atheromatous material was also noted. Based on gross and microscopic findings, it was determined that the aneurysm contained relatively recent thrombotic material, with some fibrinous lamination but no evidence of frank organization.

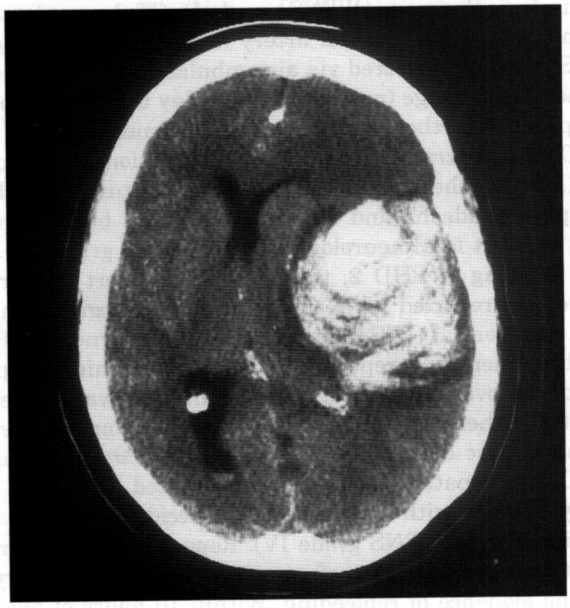

Figure 2. Noncontrast CT performed after the second deterioration on posthemorrhage day 5 shows a massive intralobar hematoma surrounding the known aneurysm. Evidence of mass effect and changes consistent with ischemia in the left middle cerebral artery territory and brainstem are also seen.

Figure 3. Gross section of cut, giant middle cerebral artery aneurysm distended with recent thrombotic material consistent with early coagulation thrombus. Note only a thin peripheral crescent of contrast material from postmortem angiography.

Discussion. The pathologic sequence of initial aneurysmal rupture followed by acute intra-aneurysmal thrombosis and rerupture, as in the case of our patient, is unique in our experience and, despite the widely studied natural history of intracranial aneurysms, we were not able to find a similar report in the literature.

SAH from giant aneurysm. Approximately 25% of giant aneurysms present clinically with SAH. It has been suggested that the development of giant saccular aneurysms from small aneurysms at the same arterial bifurcation occurs through a cycle of repeated injury and attempted healing of the defective aneurysmal wall.[9] Despite organization of the wall of a growing aneurysm, the threat of rupture arises from progressive distention of the sac due to persistent hemodynamic forces and aneurysmal wall ischemia—processes that are likely to be exaggerated in larger aneurysms. In this context, the size of our patient's aneurysm, almost 4 cm in diameter, predisposed her to an increased risk of SAH—the eventual mode of her clinical presentation.

Deterioration from giant aneurysmal thrombosis. Spontaneous intra-aneurysmal thrombosis is a well-recognized phenomenon that occurs in as many as 50% of giant intracranial aneurysms and has been implicated as a cause of acute clinical deterioration

in patients presenting with these lesions before rupture.[8,9] There is no evidence that aneurysmal thrombosis, even if extensive, confers any protection against SAH or improves the natural history of this disease.[6,8-10] It is possible that the relatively rapid development of global aphasia and dense right hemiplegia in our patient could be explained by direct extension of the thrombus into the lumen of the parent left MCA, with its subsequent occlusion. Although pathologic examination of the aneurysm, including postmortem angiography, revealed patent M1 and M2 segments, it does not exclude this possibility as the mechanism for ischemia because spontaneous thrombolysis may have occurred.

Giant aneurysmal rebleeding. Findings from the cooperative study relating to rebleeding from aneurysms of all sizes indicate that the cumulative incidence of rebleeding is approximately 5% in the first 24 hours after the initial ictus, and approximately 15% by the end of the first 2 weeks.[7] A similar pattern has been reported recently for ruptured giant aneurysms.[6] The dynamic nature of the giant aneurysm, particularly after rupture when organization, recanalization, and ongoing ischemia of the already weakened aneurysmal wall are noted to occur frequently, undoubtedly predisposed our patient to rebleeding, whereas the resultant, large intracerebral hematoma—a possible predictor of her poor prognosis—heralded the onset of her rapid clinical demise.

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