De novo dissecting aneurysm in a patient with a ruptured saccular lesion

Case report

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SEVERAL reports have documented in detail the development of new saccular aneurysms in patients known to have experienced aneurysmal SAH, defining the need for long-term neuroradiological follow-up; however, a de novo dissecting aneurysm is considered extremely rare. In the present study we report on the development and rupture of a dissecting aneurysm of the right vertebral artery, which arose from a previously angiographically documented normal artery. This rare association sheds light on the causes and growth of two distinct types of aneurysms, both clinically and pathologically.

KEY WORDS • de novo aneurysm • dissecting aneurysm • saccular aneurysm • subarachnoid hemorrhage • vertebral artery

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Case Report

This 42-year-old man suffered sudden, severe headache associated with vomiting in August 1998. He had been in good health despite smoking 20 to 40 cigarettes per day, and no family history of cerebrovascular or connective tissue disease was reported.

Examination revealed severe nuchal rigidity but no neurological deficit. Computerized tomography scanning revealed diffuse SAH predominantly around the right sylvian fissure (Fig. 1). Four-vessel cerebral angiography demonstrated a saccular aneurysm of the bifurcation of the right MCA (Fig. 2A). The left carotid (not shown) and vertebrobasilar circulations were normal (Fig. 2B and C). The ruptured aneurysm was surgically clipped on the same day via a right perional approach, with no untoward results. Postoperative angiography was performed to confirm complete obliteration of the aneurysm.

The patient remained well and normotensive until No-

Abbreviations used in this paper: CT = computerized tomography; ICA = internal carotid artery; MCA = middle cerebral artery; SAH = subarachnoid hemorrhage; VA = vertebral artery.

Fig. 1. First admission CT scans demonstrating SAH in the basal cisterns (A) and right sylvian fissure (B).
vember 2000, when he suddenly collapsed and experienced cardiac arrest while driving a car. Resuscitation was performed immediately and he regained systole during transfer. The patient was comatose and decerebrate with fixed pupils on arrival at the hospital. His blood pressure was 180/80 mm Hg and his pulse was 140 beats/minute; he was dyspneic. Intubation was performed. A CT scan revealed thick SAH predominantly in the posterior fossa, with ventricular reflux. Acute hydrocephalus was evident (Fig. 3). Bifrontal ventriculostomy catheters were placed and cerebrospinal fluid was drained to control intracranial pressure. With the patient under controlled respiration, cerebral angiography was performed and revealed no filling of the previously clipped right MCA aneurysm (Fig. 4A); however, it did demonstrate a newly developed aneurysm of the right VA in the region of the posterior inferior cerebellar artery with mild luminal narrowing proximal to the dilation (Fig. 4B–D). Contrast medium was seen to be retained in the aneurysm until the capillary phase (Fig. 4E). The ruptured aneurysm and right VA were occluded on the same day with Guglielmi Detachable Coils and fibered coils to prevent early rebleeding. Considering the poor neurological condition of the patient and well-developed ipsilateral anterior inferior cerebellar artery, parent artery occlusion was the treatment of choice despite the potential for ischemic complications (Fig. 5). The patient subsequently regained spontaneous respiration and a ventriculoperitoneal shunt was placed; however, he has remained comatose and unresponsive.

Discussion

Authors of recent reports have demonstrated that patients with completely obliterated ruptured aneurysms have a significant risk for recurrent SAH. Notably, the risk of de novo aneurysm formation in such cases is estimated to be much higher than the risk of regrowth for clipped aneurysms. During the past two decades, development of saccular aneurysms at locations previously demonstrated to be angiographically normal have been widely reported. Young patients, those with a history of hypertension, smoking, and hemodynamic alterations have a higher risk of this dual aneurysm formation. Authors of another cohort study have demonstrated that women and cigarette smokers are at increased risk of aneurysm formation during adulthood. In patients with a familial history of aneurysm formation, a genetic factor has been proposed. These studies support the hypothesis that a combination of congenital defects and acquired degeneration of the internal elastic lamina increases susceptibility to the formation of saccular aneurysms. Conversely, de novo formation of a dissecting aneurysm seems to be an extremely rare occurrence. Only five specific cases have been reported in the literature: those include two patients in whom a de novo dissecting aneurysm developed in the contralateral VA shortly after therapeutic occlusion of the VA for a primary dissecting aneurysm. The other three were children with fusiform vertebrobasilar aneurysms that formed in association with a giant fusiform ICA aneurysm. These reports elucidated hemodynamic stress and congenital structural predisposition as causative factors in selected patients.

In line with the increase in recognition of this entity, authors of recent publications have explained the clinical and histological features of dissecting aneurysms in some detail. The primary cause of this lesion is considered to be acute disruption of the internal elastic lamina, lead-
De novo dissecting aneurysm

Fig. 4. Second admission cerebral angiography. A: Right ICA angiogram revealing no filling of the clipped right MCA aneurysm. B–D: Right and left VA angiograms (arterial phase) demonstrating a newly developed aneurysm in the right VA. Equivocal stenosis was observed just proximal to the aneurysm. E: Right VA angiogram (capillary phase) revealing the contrast medium that remains in the aneurysm.

Fig. 5. Right (A) and left (B) VA angiograms demonstrating complete occlusion of aneurysm after coil embolization.

ing to intramural hemorrhage with or without luminal connection. Trauma, fibromuscular dysplasia, and arteritis have been proposed as triggering factors, but the cause remains unclear. The present report is the first to document de novo formation and rupture of a dissecting aneurysm in a patient with a history of ruptured saccular aneurysm, suggesting both aneurysms may have progressed through common contributing factors, such as inherent weakness of the vessel wall and cigarette smoking. Accumulation of similar cases will help to define the clinical and pathological pathogenesis of two distinct types of aneurysms.

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an adult developing apparently de novo. 

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