Rebleeding From Intracranial Dissecting Aneurysm in the Vertebral Artery

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We describe two patients with rebleeding from intracranial vertebral artery dissecting aneurysms during the acute stage. One patient had excellent results after emergency surgery.

A review of recent reports including 60 patients with this disorder revealed a rebleeding rate of 30%, mostly during the acute stage. This suggests that a ruptured dissecting aneurysm in the vertebral artery is at risk for rebleeding during the acute stage, similar to a saccular aneurysm in the same location. (Stroke 1990;21:1628-1631)

Unlike dissecting aneurysms in the carotid artery system, those in the vertebrobasilar artery system have been reported only recently.1-4 Unsettled issues regarding natural history and treatment modalities remain, including whether ruptured dissecting aneurysms in the vertebral artery should be treated in the same manner as saccular aneurysms in the same location.

Recently in the United States, the comment was made that surgical occlusion of vertebral artery dissecting aneurysm is not necessary because no one has died of rebleeding.5 We describe the clinical courses in two patients with rebleeding from vertebral artery dissecting aneurysms.

Case Reports

Case 1

A 55-year-old woman with no remarkable history presented with the sudden onset of severe headache at 4:30 AM on February 7, 1984. On arrival at the hospital, she was alert and had no neurologic abnormalities. A computed tomography (CT) scan revealed subarachnoid hemorrhage (SAH), particularly prominent in the right ambient cistern. Retrograde brachial artery angiography on the right side disclosed a fusiform aneurysm in the right vertebral artery at the branching of the posterior inferior cerebellar artery (Figure 1). A stenotic lesion was also noted in the right vertebral artery just distal to the aneurysm. Hematologic studies revealed no abnormal findings.

The patient was managed conservatively. At 6 PM on the day of admission, she was found in a semicomatose state. CT scan performed at 10 AM on February 8 confirmed an increase in the amount of SAH. On February 9 she was transferred to another hospital, where ventricular drainage was undertaken on February 10. After surgery, she remained comatose and died on February 19. No autopsy was performed.

Case 2

A previously healthy 50-year-old housewife noticed headache at 11 PM on December 28, 1988. At 2 AM on December 29 she was brought to a nearby hospital when the severity of her headache worsened and she developed urinary incontinence and vomiting. At that time she responded to verbal commands. A CT scan disclosed SAH, prompting referral to us. Immediately after arrival at our hospital at 6 AM, the patient developed sudden respiratory arrest, necessitating ventilatory support. A CT scan revealed an increase in the volume and distribution of SAH. Left vertebral artery angiography showed a fusiform aneurysm in the left vertebral artery just distal to the posterior inferior cerebellar artery (Figure 2). A stenotic lesion in the left vertebral artery was evident proximal to the aneurysm. Results of laboratory studies were within normal limits.

Emergency suboccipital craniectomy revealed a fusiform purple aneurysm. After clipping of the vertebral artery distal to the posterior inferior cerebellar artery, massive bleeding from rupture of the aneurysm was encountered, which we controlled by additional clipping distal to the aneurysm (trapping). The patient's postoperative course was complicated by lower cranial nerve paresis, which lasted for 2 months. She was discharged from the hospital 3 months after surgery, without neurologic deficits.

Discussion

Dissecting aneurysms in the extracranial portion of the vertebral artery are known to cause vertebrobasi-
FIGURE 1. Right retrograde brachial artery angiograms (top: anteroposterior view; bottom: lateral view) in case 1 revealing fusiform aneurysm in right vertebral artery (asterisk). Narrowing of vertebral artery is also noted just distal to aneurysm (arrow).
FIGURE 2. Left vertebral artery angiograms (top: anteroposterior view; bottom: lateral view) in case 2 showing fusiform aneurysm in left vertebral artery (asterisk) and stenosis proximal to aneurysm (arrow).
lar ischemia, while those in the intracranial portion are responsible for SAH. For patients presenting with SAH, it is controversial whether to treat with surgical occlusion. In fact, there is strong objection to such surgical intervention.

The diagnosis of a dissecting aneurysm in these two patients was based on the characteristic angiographic features of fusiform dilatation associated with distal or proximal narrowing of the vertebral artery. In case 2, the diagnosis was confirmed at surgery. During the past 5 years, we have encountered three cases of ruptured vertebral artery dissecting aneurysms, of which two were observed to have rebled. The interval between the first and second hemorrhages was approximately 13 hours in case 1 and 3 hours in case 2. In case 2, a third hemorrhage developed 4 hours later. Thus, we note the early occurrence of rebleeding.

In another analysis of ruptured dissecting aneurysms of the vertebral artery, Friedman and Drake reported that two (18%) of 11 cases rebled; the interval in one was 3 hours. Of seven cases treated by Shimoji et al., two (29%) showed rebleeding 2 days later. Yamaura documented rebleeding in five (24%) of 21 cases but did not describe the interval between hemorrhages. Most recently, Kamiyama et al. reported that of nine cases, three (33%) developed rebleeding, 3 hours after the first hemorrhage in one and during the acute state in two. In other sporadic reports of nine cases, rebleeding was noted in four: 15 days, 30 days, 2 hours, and 10 hours after the initial bleeding. Among these 60 reported cases of ruptured vertebral artery dissecting aneurysms, (including our three), 18 (30%) were documented to have rebled. In eight (80%) of the 10 cases giving the interval between hemorrhages, rebleeding occurred during the acute stage, mostly within several hours. This timing does not differ from that observed in patients with intracranial saccular aneurysms. It is of interest to note that the majority of reported cases of ruptured vertebral artery dissecting aneurysms are from Japan.

Emergency surgical occlusion of the vertebral artery dissecting aneurysm was carried out in one previous case and in our case 2, with an excellent result in the latter. Most previously reported cases underwent surgical intervention during the subacute or chronic stage, precluding a precise analysis of the natural history of this entity. Nonetheless, it seems apparent that the rate of rebleeding in cases with ruptured vertebral artery dissecting aneurysms is at least 30% and that most rebleeding develops during the acute stage, similar to the rate and timing of rebleeding in patients with intracranial saccular aneurysms. Appropriate management is particularly important during the acute stage of this pathology.

Noted added in proof. Since this manuscript was accepted, we have noted two important articles that appeared in 1990 on dissecting intracranial aneurysms.

References

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