Nontraumatic dissecting aneurysm of the vertebral artery

Case report

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A case is described of a nontraumatic dissecting aneurysm of the vertebral artery, which presented as a subarachnoid hemorrhage (SAH). Differentiation from vasospasm and from atherosclerosis is critical. Dissection between the adventitia and media should be noted in the differential diagnosis of SAH when an aneurysm or vascular malformation is not demonstrated angiographically.

KEY WORDS • vertebral artery • dissecting aneurysm • subarachnoid hemorrhage • stroke

The sparsity of reported cases of dissecting aneurysm of the intracranial carotid and vertebrobasilar system probably does not reflect its true incidence. It is possible that some cases of dissecting intracranial aneurysms are not correctly diagnosed clinically or angiographically.

The purpose of the present report is to describe a case of nontraumatic dissecting aneurysm of the vertebral artery, which presented as a stroke with subarachnoid hemorrhage (SAH). The operative and angiographic findings are discussed.

Case Report

This 45-year-old nonhypertensive white man came to the emergency room with complaints of headache, neck pain, and falling to the left. He had a sudden onset of these symptoms 24 hours before admission, while he was backing his car out of a driveway. For several days before this event, he had noted left suboccipital pain, left ear pain, and vertigo.

Examination. The patient had a blood pressure of 120/70 mm Hg and normal vital signs. Neurologically, he was awake and alert but had dysarthric speech. There was a coarse nystagmus, more to the left than to the right, and ocular bobbing while at rest. He had a depressed gag reflex, loss of sensation in the left oropharynx, and deviation of the uvula to the right. No motor or sensory deficit was found, but there was dysmetria and past pointing in the left upper extremity. He had a mildly stiff neck, but no photophobia. The remainder of the examination was normal.

Initial complete blood count, sedimentation rate, antinuclear antibodies, and electrolytes were normal. A complete skull series and computerized tomography (CT) scan were also normal. Spinal tap revealed an opening pressure of 170 mm H$_2$O, 168 red blood cells (RBC)/ml, and faint xanthochromia. A clinical diagnosis was made of cerebellar and lateral medullary infarction, possibly secondary to a ruptured aneurysm. A vertebral arteriogram 3 days later (Fig. 1) revealed segmental narrowing of the intracranial left vertebral artery and a possible aneurysm at the origin of the left posterior inferior cerebellar artery (PICA). A second lumbar puncture, performed on the 4th day for a metrizamide CT scan, revealed 400 RBC/ml and definite xanthochromia. The metrizamide CT scan (Fig. 2) showed left cerebellar swelling and a possible left PICA-vertebral artery aneurysm. After 3 weeks of bed rest, left lateral gaze nystagmus was the only abnormal finding on neurological examination. A second arteriogram revealed persistent narrowing of the vertebral artery, interpreted as vasospasm, and a possible aneurysm at the origin of the left PICA. One week later, the patient was taken to surgery.
for clipping of the presumed left vertebral-PICA aneurysm.

**Operation.** A paramedian suboccipital craniectomy was performed with the patient in the lateral position, and the arch of C-1 was removed. The left cerebellar tonsil was discolored from the previous SAH; it was partially resected to expose the vertebral artery from its dural entry to its junction with the right vertebral artery. Beginning approximately 1 cm intracranially, the left vertebral artery became markedly dilated and discolored. At the origin of the left PICA, no aneurysm was seen; but the point of takeoff of the PICA was stenosed, presumably by dissection between the media and adventitia of the parent vertebral artery. The ninth, 10th, 11th, and 12th cranial nerves were displaced by the dilated aneurysmal fusiform enlargement of the vertebral artery. Just before its junction with the right vertebral artery, the left vertebral artery again appeared normal. The point of SAH, as evidenced by the large subadventitial hematoma, was proximal to the PICA origin (Fig. 3). Consideration was given to trapping the vertebral artery with an occipital artery-to-PICA bypass procedure. We elected, however, to doubly clip the intracranial vertebral artery proximally and wrap the dissecting aneurysm circumferentially with cotton. It was thought that coating with acrylic or glue would mechanically and thermally injure the adherent cranial nerves.

The postoperative course was benign, and the patient was discharged on the 12th postoperative day without neurological deficit.

**Discussion**

Primary intracranial dissecting aneurysms usually occur in young individuals without evidence of atherosclerotic disease and with no or only incidental trauma. Cases with survival from intracranial arterial dissections have been reported, but the usual course is that of progressive vascular occlusion, brain swelling, and death. Accurate clinical diagnosis is almost never made preoperatively or premortem.

Yonas, *et al.*, separated these aneurysms into two groups. One group (the more common type) presents as stroke without SAH, and is characterized by dis-
sections between the elastica and media. This type can result from trauma and is ascribed to a defect in the internal elastic lamina. The second group is even more unusual; only five cases have been described previously. In this group, the dissection occurs between the media and adventitia and is presumably due to rupture of a vas vasorum. These patients present with a "pilot" SAH. This second group seems to occur most frequently in the vertebrobasilar circulation and has been of either traumatic or unexplained etiology. In this group, ours is the first patient reported to be diagnosed by metrizamide cisternography and to be treated surgically, who survived. We have no pathological confirmation except for the angiograms and intraoperative photographs. 

There is a wide spectrum of angiographic findings of arterial dissecting aneurysms, including the following: 1) irregular tapering or narrowing of the arterial lumen; 2) intimal flap; 3) double lumen; 4) false aneurysm; and 5) irregularity of the arterial wall, with a wave-like pattern but no appreciable luminal narrowing. In our case, luminal narrowing of the intradural portion of the left vertebral artery was misconstrued as spasm from rupture of an aneurysm at the origin of the PICA, which in fact was a loop of this artery at its origin, as revealed at surgery. The difficulty arises in differentiating dissection from vasospasm and from atherosclerotic lesions. If an aneurysm cannot be unequivocally seen on the angiogram, then the presence of SAH and arterial luminal narrowing should suggest dissection between the adventitia and the media. Rupture of vasa vasorum may in fact be responsible for a number of patients with proven SAH but negative four-vessel angiographic findings. Isolated unusual locations of arterial stenosis as well as the presence of smooth rather than irregular luminal narrowing should help differentiate dissection from atherosclerosis.

Intracranial dissecting aneurysms are probably more common than previously thought. Those that present as a vascular occlusion without SAH, especially in the anterior circulation, seem to have a fulminant, rapidly progressive course. In the posterior circulation, dissection is associated with a pilot SAH and is subadventitial in location. These dissections are better tolerated, probably due to collateral supply from the opposite vertebral artery and the more benign course of the lateral medullary infarction syndrome in general. If the diagnosis of dissecting aneurysm can be made preoperatively, then trapping and microvascular bypass may become the surgical treatment of choice.

References

Manuscript received May 12, 1981. Accepted in final form July 30, 1981.
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