Case Reports

Recurrent Ischemic Events in Two Patients With Painless Vertebral Artery Dissection

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Background and Purpose: Vertebral artery dissection causes endothelial changes and stenosis that may lead to recurrent ischemic neurological events. The diagnosis may not be obvious because the dissection may be painless and "spontaneous" (no obvious trauma). Magnetic resonance angiography has increasingly been used to screen patients for this disorder, but its accuracy has not yet been established.

Case Description: Two patients were admitted with repeated transient ischemic attacks and strokes over 11 months and 1 month, respectively. Neither had a history of trauma, cervical pain, or headache. Magnetic resonance angiography failed to visualize vertebral artery dissections that were later revealed by conventional angiography. One patient's events were stopped by balloon occlusion of the vertebral artery proximal to the posterior inferior cerebellar artery branch.

Conclusions: Magnetic resonance angiography is not yet sensitive enough to always visualize vertebral artery dissection. Vertebral artery dissection is a life-threatening condition that requires aggressive evaluation and treatment. (Stroke 1993;24:598-602)

Key Words: angiography • dissection • vertebral artery • young adults

Contusion and dissection of the vertebral artery at the C1-C2 level with neck rotation may occur with frightening ease. The many reports of vertebral dissection after chiropractic manipulation provide evidence that abrupt neck movement is the cause of vertebral injury. "Spontaneous" vertebral dissection has been reported with swimming, playing tennis, looking over the shoulder to back up a car, and a variety of seemingly mundane activities.

In the past decade vertebral artery dissection has been reported with increasing frequency. This increasing frequency of reports probably reflects the more widespread, the true incidence of vertebral artery dissection. Vertebral artery dissection is a life-threatening condition that requires aggressive evaluation and treatment.

Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) offer the possibility of easy, noninvasive diagnosis of vertebral artery dissection. When these technologies become affordable and widespread, the true incidence of vertebral artery dissection may become known.

This report describes two patients in whom spontaneous, painless vertebral artery dissection was associated with recurrent ischemic neurological events, MRA did not reveal the dissection.

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FIGURE 1. Cerebral angiogram (left vertebral artery injection, lateral projection) demonstrates large intraluminal thrombus (arrows) attached to dissection site at C1 level.

the left side of the pons on the T2-weighted image. There were no new lesions.

On August 21 he had an episode of several hours of tingling paresthesia on the right side of his face and his right arm. This same paresthesia returned for several hours on September 23. On October 6 he had several hours of tingling involving the left side of his face and his left arm. There was no loss of sensation or paresis during any of these episodes.

On October 9 he sought further neurological evaluation. His only neurological abnormality was right-sided hyperreflexia. Sedimentation rate, antiphospholipid antibody, anticardiolipin antibody, serum complement levels, prothrombin time, partial thromboplastin time, fibrinogen level, protein C level, protein S level, antithrombin III level, and factor XIII level were normal. A saline contrast echocardiogram and transthoracic echocardiogram were normal, as was a carotid ultrasound.

A repeat MRI revealed a new small area of increased signal intensity in the left anterior thalamus and an unchanged left pontine lesion. These lesions were not enhanced with gadolinium. The patient was instructed to take 325 mg q.d. aspirin.

In November he had two episodes of right homonymous hemianopia, one lasting 5 minutes and the other 10–15 minutes. On November 26 the patient underwent cerebral angiography, which showed an intraluminal thrombus of the left vertebral artery with intimal flap. No false lumen was seen. There was a left vertebral artery dissection that began at C1 and ended proximal to the posterior inferior cerebellar artery (Figure 1). In addition, angiography revealed a striking lack of penetrating left pontine arteries, segmental narrowing of the right superior cerebellar artery, and a cut-off sign of a right calcarine branch of the left posterior cerebral artery (Figure 2). MRA failed to reveal any abnormality. Treatment with coumadin was begun.

After a week of coumadin therapy, the patient obtained a neurosurgical opinion. A vertebral artery ligation was recommended, and the coumadin was discontinued.

Surgical ligation was not performed because the patient awoke on December 8 with marked vomiting, vertigo, and gait imbalance. He did not have visual loss, diplopia, facial paresis, paresthesia, sensory loss, or paresis. After computed tomographic scan excluded hemorrhage, he was started on heparin. His symptoms and neurological abnormalities resolved entirely within 24 hours. An MRI scan revealed a new area of increased signal intensity in the left cerebellar hemisphere, consistent with a new infarct.

The patient was referred to the University of California, San Francisco, for a balloon occlusion of the left vertebral artery proximal to the dissection. On December 12 the left vertebral artery was occluded without complications. The patient has since remained free of symptoms.

Case 2

A 56-year-old man with a 30-year history of hypertension who began to have episodes of vertigo of less than 5 minutes’ duration 1 month before initial evaluation. The vertigo was a visual hallucination of environment rotation. During several episodes he also had horizontal diplopia. The diplopia disappeared when his right eye was covered.

Ten days before seeking evaluation, he abruptly developed rotational vertigo while seated in the car with his wife, who noted that his right eye was deviated medially. On getting out of the car, he had an unsteady gait. All his symptoms resolved within 3 minutes.

The patient had no history of visual loss, tinnitus, dysarthria, arm incoordination, paresthesias, sensory loss, paresis, headache, transient monocular blindness, past stroke, cardiac arrhythmia, past myocardial infarc-
tion, or head trauma. He had recently been found to have a slight high-frequency hearing loss in his right ear. He was known to have left ventricular hypertrophy. He did not have diabetes mellitus or hypercholesterolemia. He had smoked from age 14 to age 28, up to a maximum of one pack per day. He had no cervical spine pain, manipulation, or injury.

The patient's past medical history was unremarkable. He had undergone septoplasty in 1988. He took probenecid for hyperuricemia. His hypertension was treated with 50 mg b.i.d. captopril and 50 mg q.d. chlorthalidone.

His paternal grandmother had diabetes, and a paternal uncle had diabetes, myocardial infarction, and stroke. Three paternal uncles had hypertension. His father had hypertension and died of a myocardial infarction.

The patient was a mildly overweight, right-handed man. There were no cranial bruises. There was no tenderness of the cervical paraspinal muscles and no pain with neck movement. Carotid pulses were normal, without bruit. His neurological exam was normal. Laying the patient down with his head 30° over the examination table did not cause nystagmus or vertigo. After being rotated clockwise five times, he had unsustained nystagmus on extreme left gaze.

The patient was instructed to take aspirin. MRI revealed multiple small areas of increased signal intensity in the periventricular white matter and basal ganglia and increased signal intensity in the right vertebral artery, suspicious for slow flow (Figure 3). The left vertebral artery had a normal flow void.

The patient had no further episodes of vertigo for about 2 weeks, but then several episodes recurred. Treatment with coumadin was started. An MRA revealed occlusion of the right vertebral artery and a normal left vertebral artery. Cerebral angiography demonstrated occlusion of the right vertebral artery at the C1 level and intracranial dissection of the left vertebral artery with a 90–95% stenosis and “telltale” pouch. A combined posterior inferior cerebellar artery–anterior inferior cerebellar artery complex branched from the basilar artery above the dissection. He had bilateral hypoplastic P1 segments of the posterior cerebral arteries. Retrospectively, review of the MRA was read as

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**FIGURE 3.** Axial spin-echo magnetic resonance imaging (repetition time, 2,500 msec; echo time, 60 msec) demonstrates normal signal void in left vertebral artery and abnormal increased signal in right vertebral artery consistent with slowly flowing blood or occlusion.

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**FIGURE 4.** Cerebral angiograms (left vertebral injection, anteroposterior [left panel] and right anterior oblique [right panel] projections) demonstrate focal dissection of intracranial vertebral artery (arrow). Dissecting pouch produces high-grade stenosis of adjacent vertebral artery. Right vertebral artery was occluded, resulting in retrograde filling of posterior inferior cerebellar artery from the left.
Vertebral artery to the level of the left posterior inferior cerebellar artery, it was felt that balloon occlusion could be done safely. This technique avoided the greater risks of anesthesia and surgical ligation. To date, coumadin has been effective in preventing further neurological events in our second patient. Nevertheless, the extremely marginal posterior circulation blood flow and the potential risk of further dissection intracranially causing subarachnoid hemorrhage has led us to consider the possible value of posterior circulation extracranial–intracranial bypass in this patient.

Our review of the treatment of the reported patients with vertebral artery dissection does not provide us with clear guidelines of the optimal treatment. Although aspirin appeared to be effective in some patients, the failure of aspirin in our two patients now tilts our opinion toward the use of heparin, followed by coumadin (except, of course, in cases in which there is contraindication). In their report of patients with cervical internal carotid artery dissection, Bogousslavsky et al26 described varying degrees of recovery in 20 patients treated with anticoagulants, but all seven patients not given anticoagulants died. There has been no convincing evidence that anticoagulation has worsened an extracranial arterial dissection. When repeat angiograms reveal resolution of the dissection, it seems safe to switch patients to antiplatelet medication.24 The optimal time to repeat angiography has not been defined, although recurrent events in our patients suggest it should be repeated before anticoagulation is stopped. Patients in whom angiography shows persistent irregularity of the vertebral artery intimal surface and patients who have recurrent transient ischemic attacks or stroke will need continued anticoagulation and, later, repeat angiography. The recurrent ischemic events in our patients highlight the need to consider definitive treatment by surgery or balloon occlusion distal to the dissection.22,26 It is our opinion that treatment by the interventional radiologist is preferable because it involves less risk, is less invasive, and does not exclude later surgery if necessary. Long-term evaluation of patients treated by balloon occlusion will be necessary before this technique can truly be considered a definitive treatment.

Addendum

Although coumadin had suppressed transient ischemic attacks in our second patient, he died in his sleep 12 months after his initial presentation.

References

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