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 aggressive evaluation of posterior circulation ischemic events by angiography, particularly in young adults. In some patients the vertebral artery dissection has been discovered during angiographic evaluation of subarachnoid hemorrhage. Subarachnoid hemorrhage occurs because the thinner muscular layer of the intracranial vertebral artery allows dissection into and through the adventitia.

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.

Case 1

On January 4, 1990, a 21-year-old right-handed college student suddenly noted clumsiness of his right hand when reaching for a glass, followed by a loss of sensation on the right side of his face and his right arm. He went to bed, but was awakened about 2 AM by a telephone call. He was told that his speech was slurred. In the morning he noted right hemihypesthesia, difficulty when writing, and "thumping" of his right leg when walking. He went to the student health service, from which he was referred to a local hospital.

The patient had no history of head trauma, headache, neck pain, past migraine or family history of migraine, heart disease or palpitations, rheumatological disease, past thrombotic events, diabetes, or chiropractic manipulation. He was a soccer player and had last played soccer about 2 weeks before the onset of his symptoms, but he did not recall any head or neck pain or injury.

At the hospital he was found to have a right hemiparesis involving the face, arm, and leg equally; right hemihypesthesia; and slurred speech. Complete blood count, sedimentation rate, antinuclear antibodies, and electrocardiogram were normal. An echocardiogram was normal. A noncontrast MRI scan of the brain revealed an area of increased signal intensity in the left half of the pons. After a 4-day hospitalization he was sent home and advised to take 325 mg q.d. aspirin.

Within 1 month after discharge the patient felt that he had a complete neurological recovery. On March 8, 1990, he saw a neurologist who noted right-sided hyperreflexia but no other neurological abnormalities. A cerebrospinal fluid examination was done. The findings were normal, including a normal immunoglobulin G level, absent oligoclonal bands, and a nonreactive VDRL.

On June 19 a repeat MRI scan without gadolinium revealed the same area of increased signal intensity on...
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 infar-
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 bruits. 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
showing a flow abnormality at the site of the left vertebral outpouching.

The patient had no further transient ischemic attacks while continuing to take coumadin.

Discussion

Vertebral dissection may cause subarachnoid hemorrhage, infarction, and as these cases illustrate, repeated ischemic events. The wide distribution of strokes and transient ischemic attacks in the first patient suggested that he had recurrent embolic events. The nearly identical events in the second patient may have been due to emboli or intermittent occlusion in the severely stenotic left vertebral artery. In both patients the disruption of the endothelial surface by the dissection most certainly created a site for platelet aggregation. Surprisingly, only a few patients have been reported to have recurrent ischemic events.11,17,23

The cause of vertebral dissection in these patients was unclear. The first patient had played soccer about 2 weeks before the occurrence of his stroke. However, there had been no injury or cervical pain. The second patient had long-standing hypertension. Speculation exists that hypertension may increase the risk of dissection. The incidence of hypertension among patients with vertebral dissection is 23–53%.11,17,18,23 The vertebral dissections in these two patients appeared to have been “spontaneous.” The dissections could not be ascribed to specific traumatic events. There was no evidence of fibromuscular dysplasia, syphilis, Marfan’s syndrome, vasculitis, or other underlying vascular disease. Also, unlike in most patients, the dissection was painless. Even without overt trauma or subarachnoid hemorrhage, headache or neck pain have been reported to occur in more than 75% of patients with vertebral dissection.18

The recent availability and safety of MRI and MRA may lead to a further increase in the frequency of diagnosis. However, the results in our patients indicate that MRI is not yet sensitive enough to always visualize the dissection. MRI and MRA failed to detect the dissection in both of our patients. In our second patient, MRI indicated the likely presence of a right vertebral dissection. In their report of patients with cervical internal carotid artery dissection, Bogousslavsky et al22 described varying degrees of recovery in 20 patients with recurrent transient ischemic attacks or stroke treated with anticoagulants. However, the results in our patients indicate that MRI is not yet sensitive enough to always visualize the dissection. MRI and MRA failed to detect the dissection in both of our patients. In our second patient, MRI indicated the likely presence of a right vertebral dissection but failed to reveal evidence of the left vertebral dissection.

Most patients with vertebral artery dissection have a single ischemic event followed by resolution of the dissection within 3 months.28 Spontaneous recovery did not occur in our patients. Moreover, these patients continued to have ischemic events despite their use of aspirin. The first patient was an active, young man for whom chronic coumadin treatment was unappealing. Furthermore, although coumadin anticoagulation might have prevented embolism, it seems doubtful that anticoagulation would have allowed healing of the dissection. The technique of balloon occlusion seemed an elegant method of preventing further embolization. Because retrograde flow could occur through the right 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 al22 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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Stroke. 1993;24:598-602
doi: 10.1161/01.STR.24.4.598

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