Repair of a vertebral artery dissection

Case report

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The case is presented of a 38-year-old woman who suffered multiple cerebellar infarctions as a result of emboli from a vertebral artery dissection. Surgical therapy led to a satisfactory recovery. This case emphasizes the importance of an aggressive approach to such lesions.

**Key Words** • vertebrobasilar insufficiency • arterial dissection • shear stress • thromboembolism • vertebral artery • dissecting aneurysm

More than 50 cases of cerebral, cerebellar, or spinal cord infarction have been reported in association with an abrupt change in head position. While most of these cases have been related to chiropractic cervical manipulation, other causes have included vigorous calisthenics, yoga, overhead work, diving, and trauma leading to fracture-dislocation of the cervical spine. Clinical manifestations include signs of posterior fossa ischemia, such as dizziness, ataxia, vertigo, syncope, or visual disturbance. In addition, cases of ipsilateral cranial nerve defects, Horner’s syndrome, quadriplegia, and sudden death have been described.

This report documents a case of vertebral artery dissection resulting from a traumatic endotracheal intubation. Successful arterial reconstruction of this unusual lesion has, to our knowledge, not been reported previously.

**Case Report**

This 38-year-old nurse had a diagnosis of Samter’s syndrome, steroid-dependent asthma, nasal polyposis, and acetylsalicylic acid sensitivity. Her prior medical history included multiple nasal polypectomies and a prolonged course of steroids and bronchodilators for the treatment of asthma. In March, 1984, her inadvertent use of aspirin at home led to severe respiratory distress, requiring rapid orotracheal intubation and fluid resuscitation at a local hospital. Approximately 24 hours later, following successful extubation, she complained of persistent global headaches with dizziness and photophobia.

Physical examination showed her to be alert and oriented. There was no neck stiffness. Cranial nerves were intact, and no sensory or motor deficit was appreciated. Deep-tendon reflexes were brisk and bilaterally symmetrical. Babinski and Kernig signs were absent.

Although her gait was normal, the patient was found to have a left finger-to-nose dysmetria and a mild left heel-to-shin ataxia. There were no carotid or vertebral bruits, and auscultation of the heart revealed a regular sinus rhythm.

Cranial computerized tomography (CT) at this time demonstrated a hypodense lesion of the left cerebellum with a shift of the fourth ventricle to the right. This lesion was thought to be due to a hypertensive episode and the patient was treated with dexamethasone, mannitol, and phenytoin. One week later, worsening headaches and an ataxic gait led to repeat CT of the brain with intravenous contrast enhancement. This study revealed multiple asymmetrical cerebellar infarcts, suggesting an embolic etiology. The patient was then transferred to the University of Chicago for further evaluation.

**Examination.** An electrocardiogram and echocardiogram showed no evidence of a cardiac source of
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emboli. An intravenous digital subtraction angiogram demonstrated normal carotid arteries bilaterally. The right vertebral artery was small but otherwise normal. The left vertebral artery was very irregular with what was interpreted as a plaque located approximately 3 to 4 cm from its origin (Fig. 1). Due to the recurrent nature of the embolic episodes, anticoagulation therapy alone was thought to be inadequate.

Operation. The patient underwent surgical exploration of the left vertebral artery, which revealed a subintimal dissection of the cervical portion of the artery extending from T-1 to the transverse foramen of C-6. Serial sections of the affected vessel later showed an organizing hematoma involving the media and extending to the adventitia, with oblitative compression of the arterial lumen (Fig. 2). There was no evidence of an underlying abnormality of the arterial wall.

A common carotid to vertebral artery bypass procedure was performed using a reversed saphenous vein graft. The proximal vertebral artery was ligated and an end-to-end anastomosis was performed distally. Partial unroofing of the vessel within the transverse process of C-6 was required to expose sufficient normal vessel to complete the distal anastomosis. There was severe intimal fragmentation which precluded anastomosis proximal to the interosseous portion of the artery. An intraoperative angiogram with injection into the proximal graft showed a patent distal anastomosis with no residual vertebral artery lesions (Fig. 3).

Postoperative Course. The patient recovered without incident and was discharged on the 5th postoperative day. Regular follow-up visits have revealed full resolution of her initial deficits, and no subsequent neurological symptoms have been noted.

Discussion

The pathogenesis of vertebral artery injury is related to its anatomical course, with potential compression

Fig. 1. Intravenous digital subtraction angiogram, right posterior oblique view, showing an abnormal irregular left vertebral artery (arrow).

Fig. 2. Interval sections through the excised segment of the left vertebral artery. Sections are arranged in order from left to right in both rows from the proximal to the distal ends. The media is occupied by the hematoma which compresses the lumen. The hemorrhage extends into both the intima and the adventitia.
areas at the transverse foramina of the C-1 through C-6 vertebrae, at the atlanto-occipital joint, and at the atlanto-axial joint, which is the major site of cervical rotation. Furthermore, compression by skeletal muscle and fascial bands can occur at the first portion of the vessel. A reduction in vertebral artery blood flow due to hyperextension, tilting, or rotation of the cervical spine has been well described. In addition, these maneuvers can subject the artery to stretching and shear stress, which can cause intimal injury and result in arterial thrombosis, dissection, or pseudoaneurysm formation.

In a recent review of the literature, Goldstein reported nine cases of vertebral artery dissection which had been clearly documented by angiography or demonstrated by examination of the operative specimen. Five of these involved the intracranial portion of the vessel, while two were localized to C-2, one to C-2-4, and one involved the entire cervical portion of the artery from T-2 to C-2. The clinical presentations in that series included subarachnoid hemorrhage in five patients, global ischemia in one, and posterior fossa ischemia in two; one patient was asymptomatic. All patients were in the fourth to sixth decades of life, and there was an equal sex distribution. Since that review, a single additional report has been published of an intracranial vertebral artery dissection occurring in a 45-year-old man; this presented as a stroke with subarachnoid hemorrhage.

Dissection of the vertebral artery due to mechanical injury can occur in either a subintimal or subadventitial plane. Both lead to luminal narrowing, flow turbulence, and possible thromboembolism. With dissection between the internal elastic lamina and the media, intraluminal thrombosis often results in posterior fossa infarction without intracranial bleeding. This disorder has generally been treated with anticoagulation therapy, supportive care, and rehabilitation. However, progressive basilar occlusion has often led to a poor outcome. With dissection between the media and adventitia resulting from medial degeneration, the patient more often presents with subarachnoid hemorrhage, and has an equally poor prognosis.

A successful therapeutic approach to vertebral artery dissection has been severely limited by a failure of recognition, the frequent intradural location of this lesion, and the rapid and progressive neurological injury associated with it. The present report describes a case of vertebral artery dissection, likely related to manipulation of the cervical spine during rapid orotracheal intubation, in which the resulting neurological injury was minimal due to effective evaluation and surgical treatment. As suggested by others, we believe that this favorable outcome further supports aggressive surgical management of vertebral artery lesions, especially when flow in the contralateral vessel is reduced by hypoplasia or stenosis.

References

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Manuscript received March 28, 1985.
Accepted in final form September 17, 1985.
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