Posttraumatic bilateral vertebral artery occlusion

Case report

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A case of bilateral vertebral artery occlusion following trauma in a 25-year-old woman is presented. The patient had minimal subluxation of C-2 on C-3 without neurological deficit. Her neck was immobilized for 16 days, and then a posterior fixation of C-1 through C-4 was performed with Kirschner wires and methyl methacrylate. Occlusion of the vertebral arteries has persisted, but collateral vessels are adequate and the patient has remained neurologically normal.

KEY WORDS: vertebral artery occlusion, cervical trauma, cervical subluxation, cervical spine

Vertebral artery injury has resulted from chiropractic manipulation; penetrating injuries of the neck; football, wrestling, and diving injuries; prolonged abnormal positioning of the neck; birth trauma; and closed head and neck trauma. The constant feature of the nonpenetrating mechanisms that injure the vertebral artery is hyperextension of the neck, with or without rotation or lateral flexion. A variety of lesions result: pseudoaneurysms, arteriovenous fistulas, arterial emboli, and frank occlusion of the vertebral artery.

Unilateral, extracranial occlusion of the vertebral artery may cause a serious, even fatal neurological deficit with brain-stem infarction, cerebellar infarction, or infarction of both areas. More often, however, unilateral occlusion of the vertebral artery is well tolerated unless the contralateral vertebral artery is hypoplastic or ends in a posterior inferior cerebellar artery. Bilateral occlusion of the vertebral arteries, when caused by atherosclerosis or chronic instability of the cervical spine, is usually well tolerated, because it develops slowly enough to allow the growth of collateral vessels and tends to occur in older, less active patients. That type of bilateral vertebral artery occlusion should not be confused with the acute occlusion resulting from trauma, which is usually manifested by immediate neurological deficit.

Nowhere have we found a report of asymptomatic posttraumatic bilateral artery occlusion such as is presented here.

Case Report

This 25-year-old woman was a passenger in a vehicle that was involved in a traffic accident. She was not wearing a seat belt. Her face struck the windshield of the car, but she was not rendered unconscious. Her only complaint initially was severe neck pain. However, over the next 4 to 5 hours, she became increasingly hoarse and had difficulty in swallowing.

Examination revealed superficial facial abrasions and marked swelling of the neck and posterior pharynx. The neurological examination showed no abnormalities, nor did examination of other systems. She had no bruits in her neck and both carotid arteries had normal pulsation.

Roentgenograms of the cervical spine in the anteroposterior and lateral (Fig. 1) projections showed a huge, prevertebral soft-tissue mass; no fractures were noted, but a 2-mm subluxation of C-2 on C-3 associated with a 12° anterior angulation was present.

A carotid arterial tear was suspected to be the cause of the soft-tissue mass, but bilateral carotid arteriography demonstrated no lesions. The right common carotid artery and the lower portion of the right internal
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carotid artery were displaced laterally by the mass. On the left carotid artery injection, the superficial occipital artery could be seen reconstituting the vertebral artery at the level of C-2. Left subclavian (Fig. 2) and innominate (Fig. 3) arteriography demonstrated occlusion of both vertebral arteries at the level of C-3. Each vertebral artery was reconstituted by intramuscular collateral vessels of the thyrocervical trunk and by collaterals from the superficial occipital artery. The intracranial vascular anatomy was normal, except that dye entered the entire distal half of the basilar artery and the posterior cerebral tree through a right posterior communicating artery.

The patient was treated with cervical traction by Gardner-Wells tongs and heparin anticoagulation. Over 10 days, the swelling of her neck subsided and her swallowing and speech returned to normal. Sixteen days after the accident, a posterior cervical fixation of C-1, C-2, C-3, and C-4 was carried out by placing Kirschner wires through the spinous processes and strengthening with methyl methacrylate. Postoperatively, the patient still had a normal neurological examination; subclavian arteriography (Fig. 4) showed persistence of the vertebral artery occlusion and reconstitution by collateral vessels.

**Fig. 1.** Lateral cervical spine film showing minimal subluxation and angulation at C2–3. Arrows indicate the anterior margin of the prevertebral hematoma.

**Fig. 2.** Left subclavian arteriogram, anteroposterior (left) and lateral (right) views, showing the left vertebral artery occluded (lower arrowhead) and reconstituted (upper arrowhead) by the thyrocervical trunk. The lateral film also shows the extent of the prevertebral hematoma (arrows).
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FIG. 3. Innominate arteriogram, anteroposterior (left) and lateral (right) views, showing occlusion and reconstitution of the right vertebral artery (large arrowheads). The anteroposterior view also shows displacement of the carotid artery by the hematoma (small arrowheads).

She was discharged from the hospital, and anticoagulation therapy was continued for 1 month. Six months after the injury she remains well.

Discussion

The lack of neurological deficit in this patient leads us to theorize that intramuscular collateral vessels and an extensive posterior communicating artery system immediately reconstituted the vertebral arteries above the occlusion. Her difficulty with breathing and her hoarseness were almost certainly due to local pressure from the massive prevertebral hematoma, and were not manifestations of brain-stem ischemia.

Many of the reported cases of posttraumatic vertebral artery occlusion had an asymptomatic interlude of a few hours between the trauma and the onset of signs of brain-stem or cerebellar dysfunction. Marks and Freed suggested that this interlude was due to the slow progression of the thrombosis or, alternatively, to the gradual swelling of the vessel wall secondary to trauma. Another cause might be the slow swelling of the infarcted brain stem and cerebellum, which are only partially supplied by collaterals.

The mechanism of injury of the vertebral artery following nonpenetrating trauma is stretching and tearing of the intima and media in a vessel tethered to bone. A similar mechanism was proposed by Stringer and Kelly as the cause of carotid artery dissection during hyperextension of the neck. The vertebral ar-
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tery is susceptible to this type of injury at multiple levels, most commonly at C-5 and C-6 just after it enters the foramen transversarium, at the atlanto-axial level just after it leaves the foramen transversarium, and at the atlanto-occipital level where it is tethered by the dura. It has been shown that the last two sites can also be "physiologically" occluded by a change in head position.26,27

It is possible that vertebral artery injuries are more common than is realized, but remain unrecognized because arteriography is not performed. As computerized tomography (CT) replaces arteriography in the neurological evaluation of cervical trauma, major vascular injuries in the neck may be overlooked. Therefore, for such cases, arteriography must be considered if the CT scan is normal and a focal neurological deficit develops, either immediately or within a few hours. In assessing cervical trauma, the vertebral artery must not be forgotten.

The management of our patient was directed toward stabilizing the cervical spine dislocation and decreasing the risks of embolization from the occluded vertebral arteries. The spine was stabilized initially by skeletal traction and later by a fixation procedure placing Kirschner wires through the spinous processes and strengthening with methyl methacrylate. Anticoagulation therapy was initiated as soon as angiography had shown occlusion, and had indicated no active bleeding into the hematoma site.

References
17. Pratt-Thomas HR, Berger KE: Cerebellar and spinal injuries after chiropractic manipulation. JAMA 133:600-603, 1947
28. Yates PO: Birth trauma to the vertebral arteries. Arch Dis Child 34:436-441, 1959

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