Delayed sequelae of vertebral artery dissection and occult cervical fractures

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Article abstract—We report three patients who developed delayed (ie, more than a week after the injury) symptoms of vertebrobasilar ischemia following motor vehicle accidents. The patients all had angiographic evidence of vertebral artery dissection and, upon further evaluation, occult fractures of the second cervical vertebra that were not detected by simple cervical spine radiography and required polytomography or CT for diagnosis. Vertebral artery dissection can result from occult cervical spine fractures and may present with delayed symptoms of brain ischemia.

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Trauma to the extracranial portion of the vertebral artery may produce intimal tears, often accompanied by thromboembolism and distal ischemia.[1,2] Sometimes the degree of trauma capable of causing dissection is surprisingly mild and does not prompt suspicion of dissection.[3,4] The immediate sequelae of vertebral artery dissection include infarction of the brainstem, cerebellum, and posterior portions of the cerebral hemisphere.[4-9] Delayed neurologic sequelae, on the other hand, do not seem to be as frequently recognized. Consequently, we report three patients who suffered symptoms consistent with vertebrobasilar ischemia over weeks or months following motor vehicle accidents. Each had occult cervical spine fractures and vertebral artery dissections.

Case reports. Patient 1. In 1975, a 47-year-old mailman suffered a brief concussion and rotational head injury in an automobile accident. He complained of pain and tenderness in the right side of the neck. The cervical pain decreased, and he remained asymptomatic until 3 months later, when he suffered a drop attack. Shortly afterward, mild hoarseness and dysphagia were noted, with subsequent improvement over a period of days. At 8 months, double vision and gait imbalance occurred, followed by worsening of his hoarseness, dysphagia, and vertigo. He developed decreased sensation of the right side of the face and the left side of the body. Repeat cervical spine x-rays were normal, but polytomography showed a fracture of the lateral mass of the second cervical vertebra (C-2), and angiography revealed an intraluminal clot and occlusion of the right vertebral artery immediately above C-2 (figure 1).

Patient 2. In 1975, a 45-year-old man sustained a rotational injury to his neck and a mild concussion during an automobile accident. Afterward, he experienced severe neck pain for 3 weeks and then became asymptomatic. One morning 2 months later, he awoke with moderate dysarthria, dysphagia, left facial numbness, and dizziness, which lasted 3 days. Plain x-rays of the cervical spine were normal. Polytomograms showed a fracture at the junction of the lateral mass and lamina of C-2. Angiography disclosed occlusion of the right vertebral artery at the level of C-2 (figure 2).

Patient 3. In 1991, a 63-year-old man was the unrestrained driver of a pick-up truck that skidded and fell into a ditch. He appeared normal at the scene, but was admitted to a local hospital for observation for 24 hours without any complications. Two weeks after the accident, he developed the first of seven episodes of transient vertigo, blurred vision, right facial numbness, and right-hand weakness. The episodes recurred over a 2-month period until he presented for evaluation. Neurologic examination was normal. MRI showed nonspecific white matter changes in the left hemisphere, and magnetic resonance angiography (MRA) showed irregular attenuation of the left vertebral artery. Digital subtraction angiography demonstrated focal narrowing of the left vertebral artery at the level of the fourth cervical vertebra (C-4), consistent with dissection. Routine cervical spine radiographs were normal, but CT showed a linear fracture of C-2 (figure 3).

Discussion. The frequency of delayed (ie, presenting later than 1 week after the injury) cerebrovascular sequelae of traumatic vertebral artery injury is unknown, as the precipitating event is considered trivial when it does not cause immediate symptomatology. Trauma to the carotid or vertebral artery wall may produce tearing of the intima and subsequent mural dissection.[1,10,11] This process

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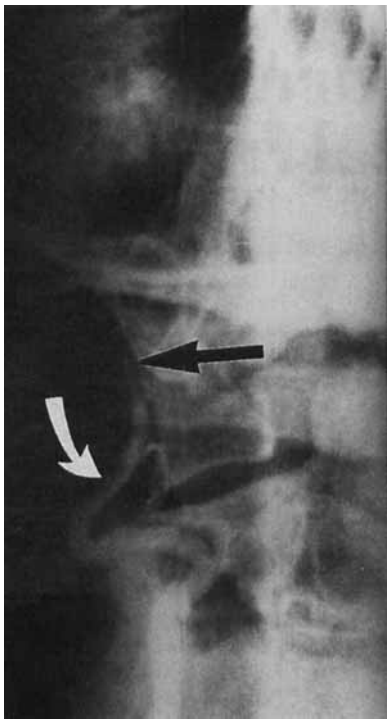

Figure 1. Anteroposterior view of vertebral angiogram of patient 1 demonstrating an intraluminal clot (white arrow) and tapered occlusion of the right vertebral artery (black arrow) above C-2.

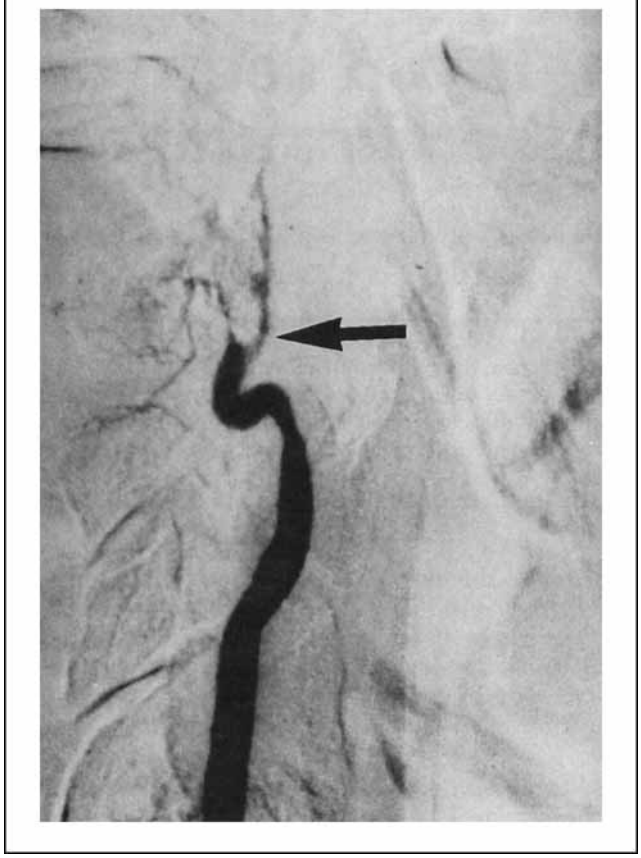

Figure 2. Lateral view of vertebral angiogram of patient 2 demonstrating tapered occlusion of the right vertebral artery at the C-2 level (arrow).

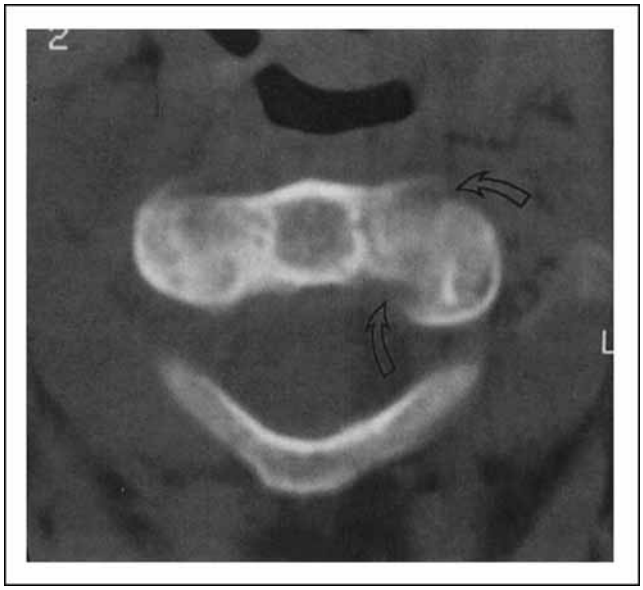

Figure 3. CT of the spine of patient 3 showing linear fracture of C-2 (arrows).

results from either shearing forces secondary to rotational injuries[10,12,13] or direct trauma to the vessel wall upon bony prominences,[14] especially the transverse processes of the cervical vertebrae.[15]

The classic symptoms of vertebral artery dissection include occipital and neck pain accompanied by signs of transient or persistent brain ischemia. The key to diagnosis is a high degree of suspicion, especially in patients who have a history of head and neck injury, who present with headache, neck pain, and clinical evidence of brainstem ischemia.[16-19] It must be kept in mind that ischemia of the basilar artery territory may lead to homolateral face and body symptoms (such as in our patient 3) owing to bilateral brainstem involvement or unilateral thalamic/midbrain ischemia. Although definitive diagnosis still requires angiography, recent advances in noninvasive diagnostic procedures may facilitate the early evaluation and follow-up of patients with vertebral artery dissection. Real-time B-mode ultrasound can show intimal flaps in patients with carotid artery dissection, while color Doppler imaging may also demonstrate the presence of a dual (true and false) lumen.[20-22] Unfortunately, ultrasonic imaging of the vertebral artery within the vertebral canal (V2 segment) is more difficult and less sensitive.[23] In addition to ultrasound, MRI and MRA are gaining importance as noninvasive methods for studying the cerebral blood vessels and have allowed others to diagnose vertebral artery dissection.[2,24]

Prior to the present report, there were only six other cases of delayed symptoms from vertebral artery dissection in the literature (table).[2,9,10,17,23,25] In addition, we found three additional patients in whom symptoms did not occur until 6 or 7 days following the injury (table).[1,4,10] None of the patients previously reported underwent specific radiographic investigation for the presence of occult spinal frac-

Table. Cases previously reported in the literature of patients suffering delayed neurologic sequelae of vertebral artery dissection

Reference	Age	Trauma	Interval	Symptoms and signs	Angiographic findings
1	12	Fall from car	7 d	Headache, vomiting, coma, decerebration	VA and BA occlusion
2	31	MVA	1 yr	Diplopia/ vertigo, HA	Irregular VA narrowing
4	22	Minor head trauma	7 d	Ataxia, facial and tongue weakness, abducens paresis	Traumatic aneurysm of VA
9	31	MVA	2 wk	Locked-in syndrome	Bilateral VA occlusion
10	27	Neck manipulation	23 d	Vertigo, HP	VA occlusion, VA stenosis
10	32	Neck manipulation	6 d	Nystagmus, dysarthria	Traumatic aneurysm and stenosis of VA
17	39	MVA	6 wk	Vertigo, dysarthria, dysphagia, facial weakness, hypalgesia	Irregular VA narrowing
23	33	MVA	5 mo	Dysarthria, vertigo, HA	Stenotic VA at C1-2; PCA occlusion
25	32	MVA	1 yr	HA, impaired memory	Stenotic VA at C-1; PCA occlusion

MVA Motor vehicle accident.
HA Hemianopia.
HP Hemiparesis.
VA Vertebral artery.
BA Basilar artery.
PCA Posterior cerebral artery.

tures. In our patients, cervical spine radiography was inadequate, and either polytomography or CT were necessary to demonstrate the fractures. It is of interest that the levels of the fractures and those of the angiographic abnormalities were not exactly the same, suggesting that the former were markers for the significant adjacent soft tissue injuries sustained by the patients. Thus, in patients with symptoms of vertebrobasilar ischemia, it is important to inquire about preceding head or neck trauma in recent weeks or months. This is especially true in younger patients or in patients in whom other risk factors for stroke are not present. The patients described also illustrate the need to suspect occult cervical spine fractures in patients found to have vertebral artery dissection with delayed symptomatology.

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