# Vertebral Artery Dissection

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## ABSTRACT

Vertebral artery dissections (VADs) following a variety of minor traumatic mechanisms have been previously reported. This article reports 2 cases of VAD with delayed recognition following motor vehicle collisions (MVCs). The first VAD patient developed major neurologic abnormalities 28 hours after an MVC. The second VAD patient presented with 3 weeks of neck and head pain beginning 8 weeks after an MVC and subsequent chiropractic manipulation. The anatomy and pathophysiology of VAD are reviewed. Early ED recognition prior to the onset of major neurologic deficits (e.g., paresis, dysarthria, ataxia, or altered mental status) is emphasized. An algorithm for the ED management of the entity is suggested.

**Key words:** vertebral artery dissection; stroke; neck trauma; case report.

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Vertebral artery dissection (VAD) following blunt trauma has been previously described as a rare occurrence.[1–3] More recently, awareness of this potentially catastrophic clinical entity has heightened.[4,5] Case reports found thus far in the emergency medicine (EM) literature have focused on making the diagnosis only after disabling neurologic deficits have occurred.[1,6,7] In 2 of these cases,[1,7] the onset of major neurologic findings was delayed and the diagnosis was made after initial release from the ED. We report 2 additional cases of delayed clinical presentation. We review the related anatomy and pathophysiology of VAD and propose a diagnostic algorithm when VAD is suspected.

## CASE 1

A 32-year-old woman was brought by paramedics to the ED after a motor vehicle collision (MVC). The patient had been the restrained driver of a vehicle that was broadsided at an intersection by a van traveling at an unknown speed. There was no loss of consciousness and the patient was ambulatory at the scene. Due to the significant passenger space intrusion on the driver's side of the vehicle, the patient was positioned with full cervical spine precautions and transported to a Level-1 trauma center.

Upon arrival at the ED, the patient complained of pain in the left neck, left lumbar area, and bilateral lower extremities. Physical examination showed the patient to be awake, alert, oriented, and obese, and to have stable vital signs. There was no evidence of head trauma, pupils were equal, round, and reactive to light, and extraocular movements were intact. On cervical examination, the patient continued to complain of vague, left-sided neck pain, but no discrete area of tenderness could be identified. Mild epigastric tenderness was noted on examination of the abdomen. There was tenderness in the left lumbar area. Ecchymoses were seen in the pretibial areas bilaterally. Sensory and motor examinations were normal. Extremity reflexes were symmetrical and toes were down-going bilaterally.

Cervical spine, chest, pelvis, lumbar spine, and lower-extremity films were negative. Initial urinalysis following catheter placement revealed microscopic hematuria with >50 red blood cells per high power field. Urine pregnancy test was negative, as was the abdominal CT. Although the patient had suffered little visible trauma, she was admitted for observation. Twenty eight hours after arrival, the patient made an attempt to get out of bed and fell to the floor. Upon arrival of her physician, the patient was able to open her eyes to verbal stimulus, follow commands, and mumble inappropriate speech [Glasgow Coma Scale score (GCS) = 12]. Within 30 minutes she had become completely unresponsive with no eye opening and no motor movement (GCS = 3).

Emergency orotracheal intubation was performed, and a head CT revealed compression of the fourth ventricle. The patient was then taken emergently to the operating room for a ventriculostomy. A subsequent angiogram of the vertebral arteries showed dissection on the left (Fig. 1), which had caused bilateral cerebellar infarcts. These became visible several days later (Fig. 2). In this case, the VAD extended intracranially, and subarachnoid blood was

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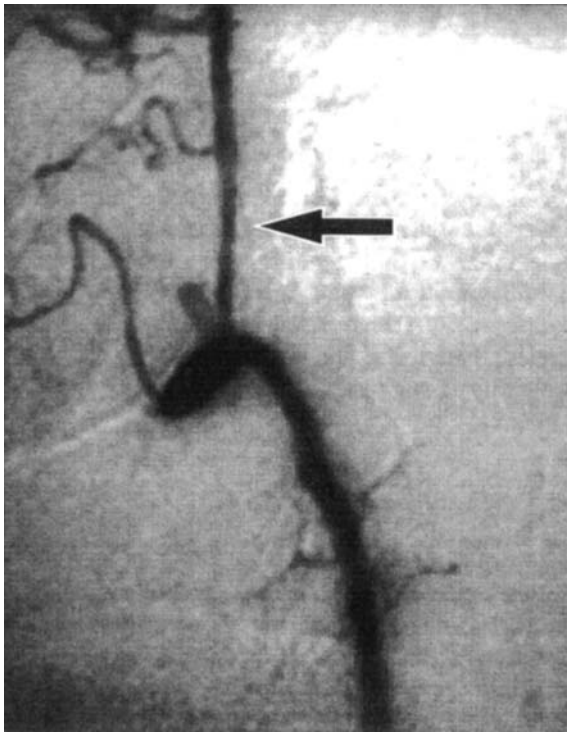

**FIGURE 1.** Angiogram showing irregular stenosis of the left vertebral artery extending into the basilar artery representing the internal lumen of the dissecting artery.

detected. Due to the subarachnoid hemorrhage (SAH) and the fear of further hemorrhage, the patient was given aspirin therapy rather than heparin. Over the course of the next few weeks, despite an improvement in mental status, the patient continued to have significant weakness and loss of motor control in all 4 extremities as well as a left gaze palsy. Four weeks following admission, she was transferred to a regional rehabilitation center to undergo therapy for her persistent neurologic deficits.

## CASE 2

A 36-year-old man presented to a community hospital ED complaining of a severe right-sided neck, occipital, frontal, and retro-orbital pain of 3 weeks' duration. The patient had been seen by several physicians prior to this and had been told he had a migraine headache. Although he had been taking the medications prescribed and had also been to see a chiropractor several times, he was unable to obtain any relief of his symptoms. His only other complaint was of intermittent blurred vision in the right eye when the pain was most severe. He had no other neurologic complaints.

Physical examination revealed only a slight decrease in light touch and pinprick on the right face and body. During the examination the patient stated that he had been in an MVC 8 weeks prior to presentation. He had been seen in an ED at that time complaining of neck pain, had normal cervical spine films, and had been released home with the diagnosis of cervical strain.

Based on this information, magnetic resonance angiography (MRA) of the head and neck was obtained and the patient was found to have a dissection of the right vertebral artery (Fig. 3). Emergency neurologic consultation was then obtained and the patient was subsequently anticoagulated. Eight weeks after the diagnosis, he was doing well taking warfarin sodium.

## DISCUSSION

**Epidemiology:** The exact incidence of VAD is unknown. In a search of the literature between 1978 and 1988, >100 reported cases of VAD were identified.[4] Mas et al. cited an audience poll conducted at a meeting of the Stroke Council of the American Heart Association in 1988, which found 250 additional unreported cases.[8]

Generally, VAD occurs in middle-aged adults with an average age of 38 years and a range of 3–63 years.[1,9] Most often patients are healthy and without predisposing risk factors for stroke.[2,5–8] Failure to make the diagnosis and institute appropriate therapy can result in serious long-term neurologic sequelae or death.[5] Clinically, VAD results in posterior circulation transient ischemic attack, stroke, and less frequently, SAH.[4] The initiating factor is

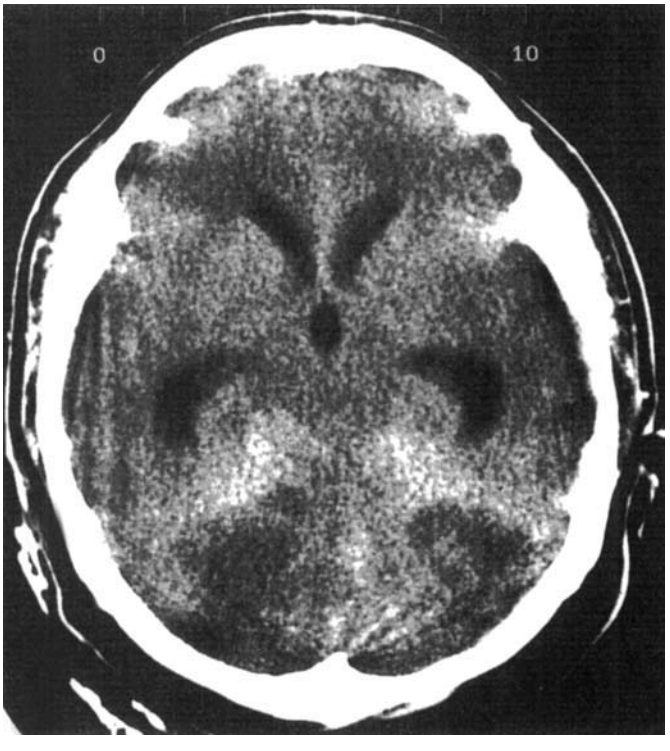


**FIGURE 2.** Head CT scan with bilateral cerebellar infarcts and hydrocephalus.

a tear in the arterial intima with subsequent hemorrhage into the wall of the blood vessel and the formation of a clot. The injury may involve only the arterial intima, but most often extends into the tunica media. Occasionally, the adventitial layer is perforated and bleeding occurs outside of the wall of the vessel.[8]

Spontaneous dissections are rare. Most are anecdotal cases related to underlying pathologic conditions such as cystic medial necrosis,[10] Behçet's disease,[11] rheumatoid arthritis,[12] giant cell arteritis,[13] osteogenesis imperfecta,[14] and fibromuscular dysplasia.[2] Cases of VAD secondary to trauma are much more commonly reported, often in the presence of major cervical trauma with accompanying cervical spine fracture or dislocation.[15,16] However, most recognized cases of VAD occur following a seemingly trivial traumatic event such as sneezing, coughing, or doing yoga exercises. Perhaps the most frequently reported cases are those observed following chiropractic manipulation.[5,8,17]

**Anatomy and Pathophysiology:** Anatomically, the vertebral arteries are divided into 4 segments: the first part originates from the subclavian artery and traverses to the transverse foramen of C6; the second part passes through the transverse foramina of C6 to C2; the third part exits the transverse foramen of C2, winds around C1 posteriorly in a tortuous manner, and enters the dura at the foramen magnum; the fourth part begins at the foramen magnum and ends where it joins the opposite vertebral artery at the basilar artery.[18]

In the majority of cases of traumatic VAD, the initial injury begins in the third part of the vertebral artery. Most authors concur that the most likely reason for this is that, with neck movement, 50% of rotation occurs at the atlantoaxial joint before any rotation begins in the cervical spine.[5,8] Rotation of the head stretches and compresses the vertebral artery at this most mobile cervical joint, an action that has been demonstrated angiographically in normal volunteers.[19]

Several mechanisms have been proposed for the resultant neurologic sequelae. Severe stenosis or occlusion may result in brain stem or spinal cord ischemia if there is a concomitant dissection of the contralateral vertebral artery that limits collateral flow. In some cases one vertebral artery may end distally in the posterior inferior cerebellar artery (PICA), resulting in minimal flow to the basilar artery distally. In such cases, a lesion of the opposite vertebral artery may result in brain stem and pontine ischemia. A second mechanism involves the formation of an intraluminal clot, which can break off, embolize, and occlude branches anywhere in the posterior circulation distal to the source. Finally, the dissection itself may ascend in a cephalic direction extending intracranially into the basilar artery, occluding branches that supply the upper spinal cord, brain stem, pons, or cerebellum. There is also the added danger of dissection through the entire wall of the blood vessel, resulting in SAH. The intracranial portion of the vertebral artery is more susceptible to rupture than the extracranial section because the artery walls thin as they cross the dura.

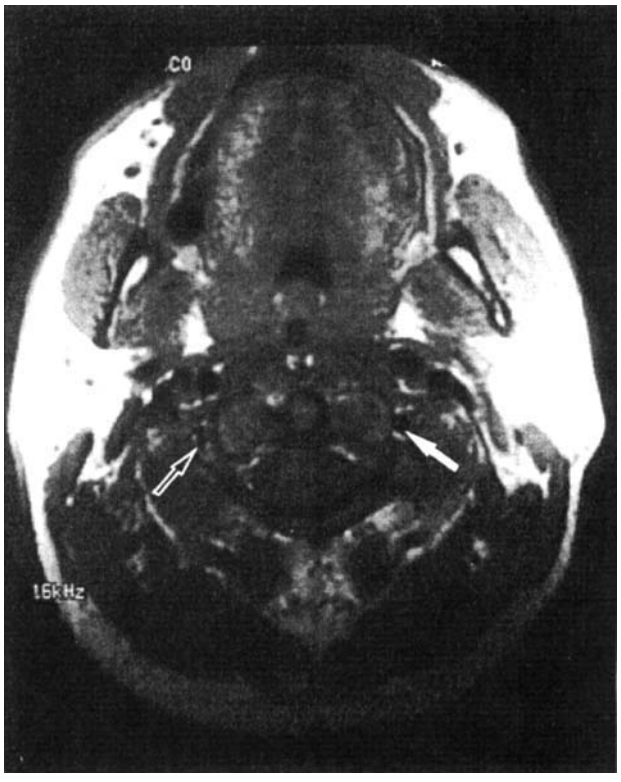


**FIGURE 3.** Angiogram showing dissection of the right vertebral artery (*open arrow*). Normal round lumen of left vertebral artery identified for comparison (*closed arrow*).

**Clinical Findings:** These multiple mechanisms, along with the complexity of the neuronal structures supplied by the posterior circulation and the variability of the posterior circulation itself, account for the wide variety of neurologic deficits seen in patients with VAD. The most classic finding is the lateral medullary or Wallenberg's syndrome characterized by dysarthria, dysmetria, ataxia, dysphagia, vertigo, nystagmus, ipsilateral Horner's syndrome, and crossed sensory deficits, i.e., loss of pain and temperature sensation on the ipsilateral face and contralateral extremities. When present, Wallenberg's syndrome is due to occlusion of the PICA that supplies the lateral medulla. However, as this area is most commonly supplied by branches from the vertebral artery, the lateral medullary syndrome is not commonly seen in its entirety.

The most common initial symptoms in VAD are neck pain and occipital headache, which may precede the onset of neurologic symptoms from seconds to weeks.[2,3,7,8] It is thought that this pain is due to the initial injury to the

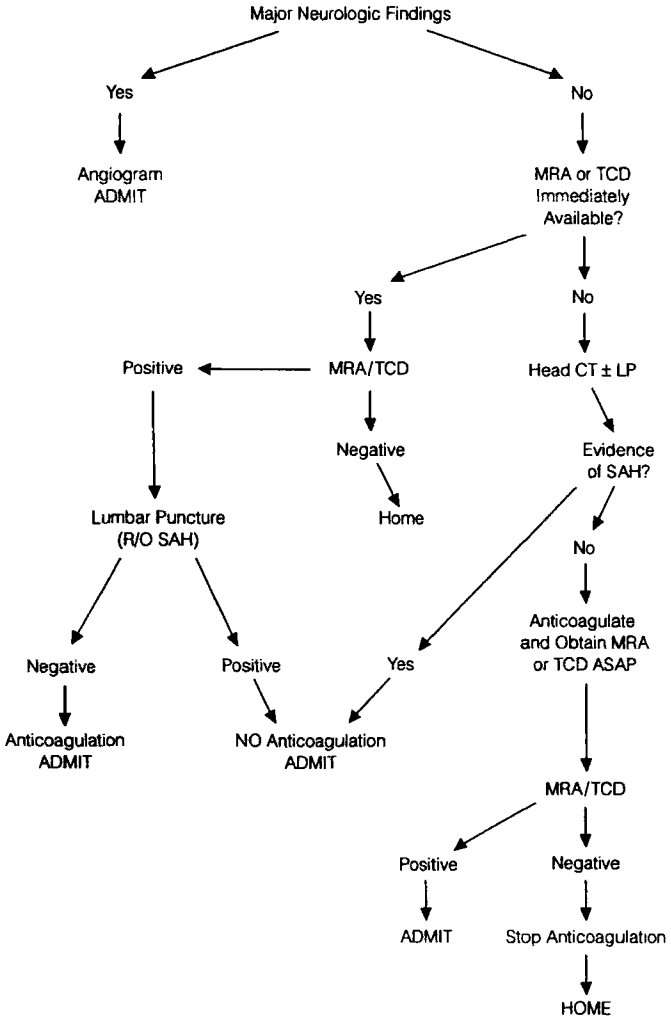


**FIGURE 4.** Suggested workup of patients presenting with neck pain that is suggestive of vertebral artery dissection after trauma. Major neurologic findings = paresis, ataxia, dysarthria, altered mental status, or similar major dysfunction. MRA = magnetic resonance angiogram; TCD = transcutaneous Doppler study; R/O = to rule out; SAH = subarachnoid hemorrhage; CT = computed tomography of head; LP = lumbar puncture; ASAP = as soon as possible.

blood vessel because most often the cervical or occipital pain is lateralized to the side overlying the injured vessel[5] and the vessels of the neck are known to be pain-sensitive.[20]

In 1993, Sturzenegger studied the characteristics of the pain associated with VAD. In 10 of 14 patients, the cervical pain was ipsilateral and posterior. Eleven of 14 patients experienced occipital headache. In most patients the pain was of acute onset, sharp or dull, and moderate to severe. The pain occasionally radiated to the ipsilateral temporal area, frontal area, eye, or ear. In 11 of 14 patients, similar pain had not been previously experienced. It was also found that none of the 14 patients had cervical tenderness or restriction of neck movement, but most (9/14) had exacerbation of pain with head movement.[5] For most of the patients (9/14), there was a history of some sort of minor trauma that heralded the onset of the pain.

**Treatment:** A VAD is a dynamic lesion, with neurologic symptoms most often progressing in a stuttering fashion over the course of hours to days. The progression is thought to result from propagation of the thrombus or from distal embolization.[4] This is the rationale for the use of anticoagulation in the treatment of extracranial VADs. Tramo et al.[21] and Mas et al.[17] report the use of antiplatelet agents (aspirin or dipyridamole) in patients with VAD. In their cases the patients' symptoms had not changed in the 24 hours prior to admission. Although no randomized clinical trial has been done to date studying the benefits of anticoagulation for VAD, anecdotal experience using heparin for the acute treatment of extracranial VAD has resulted in the arrest of the progression of the neurologic deficits in most cases.[1,4–7,17]

The treatment of intracranial VAD remains controversial.[4] Anticoagulant therapy is contraindicated in those patients at risk for hemorrhagic infarcts or SAH. In general, if the VAD is intracranial and/or there is a suspicion of SAH, the patient should be given only antiplatelet agents such as aspirin or dipyridamole.

**Early Recognition:** Since it is possible to stop the progression of further neurologic sequelae in patients already presenting with major neurologic deficits (e.g., paresis, ataxia, dysarthria, altered mental status), it would be preferred to diagnose VAD before major neurologic findings develop. The characteristic pain of VAD is a unilateral headache and/or neck pain, abrupt in onset and different from prior episodes of discomfort. There should be a history of flexion, extension, or rotation of the neck. Together these should lead to a suspicion of VAD even in the face of trivial trauma.

In such cases, noninvasive diagnostic techniques such as transcutaneous Doppler (TCD) studies or MRA may be used to establish the diagnosis. Patients found to have VADs may then be given anticoagulant therapy with the hope of preventing major neurologic sequelae.[5]

It is interesting to note that in at least 2 of the 3 cases of VAD reported in the EM literature, the patients had neck pain prior to the onset of neurologic symptoms. In the third case, the patient initially presented to the ED following an MVC, and although neck pain was not specifically mentioned, the patient presumably also was complaining of neck pain since she was referred to an orthopedist and prescribed a soft cervical collar. Seven days after the MVC, she presented to the ED again, this time with cranial nerve palsies and truncal ataxia.[7] Both our patients also complained of headache and neck pain. In these scenarios, MRA, as used in the second case, or TCD may have helped make the diagnosis prior to the onset of major neurologic deficits.

Rother et al.[22] reported that MRA has a sensitivity of 97% and a specificity of 99% for diagnosing VAD. The more invasive vertebral angiography should be reserved for cases where MRA or ultrasonographic (duplex and TCD) imaging is nondiagnostic. Ultrasonography can be used to measure flow velocity, direction, and pulse contour in middle, anterior, and posterior cerebral arteries as well as the vertebral and basilar arteries. Unfortunately, this modality is less specific and less sensitive than MRA and demands a highly skilled operator.[20]

Hoffman et al.[20] propose the use of MRA/ultrasonography in place of the invasive angiography as the modality of choice for VAD. Despite the recommendations of Hoffman et al., vertebral angiography remains the current standard for the diagnosis of VAD although its findings are not always diagnostic. When specific radiographic signs such as pseudoaneurysm or intraluminal clot are found, the diagnosis is usually assured. More often, irregular stenoses or incomplete occlusions are found, which are less specific for VAD and may be seen in atherosclerotic or partially recanalized embolic occlusions of the vertebral artery. These findings must be interpreted in conjunction with the clinical presentation.[4,17,20]

We suggest a workup approach as shown in Fig. 4. Patients with an appropriate history and major neurologic findings should have a head CT scan to rule out a mass lesion, and a vertebral angiography. For the patient without major neurologic findings, a head CT scan and lumbar puncture are warranted to rule out SAH when MRA or TCD is not immediately available. If there is no evidence of SAH, anticoagulation can be initiated pending MRA or TCD.

## CONCLUSIONS

These cases illustrate the importance of maintaining a high index of suspicion for vascular injury in any young to middle-aged person presenting with persistent unilateral head or neck pain after even minor trauma. In cases of posterior circulation symptoms with antecedent trauma, one's index of suspicion should be even higher. Evaluation of such cases with urgent MRA, TCD, or vertebral angiography should be undertaken. Anticoagulation represents the primary therapy for this condition.

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