

Vertebral Artery Anomaly With Atraumatic Dissection Causing Thromboembolic Ischemia

A Case Report

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Study Design. A case report is presented.

Objectives. To illustrate a rare cause of atraumatic vertebral artery dissection resulting from anomalous entry of the vessel at the C3 transverse foramen induced by normal physiologic head and neck motion, and to review vertebral artery anatomy and mechanisms whereby it is vulnerable to pathologic compression.

Summary of Background Data. The vertebral artery usually enters the transverse foramen at C6. Rarely, the artery enters at C5 or C4. Only one prior case with entry at C3 has been reported. That patient experienced recurrent quadriplegia and locked-in syndrome caused by vertebral artery obstruction. A 27-year-old woman with a history of classic migraine experienced neurologic symptoms on three occasions related to physiologic neck and arm movements. Magnetic resonance angiogram was not diagnostic, but standard arteriography demonstrated anomalous vertebral artery entry into the C3 transverse foramen and focal dissection.

Methods. Pertinent literature and the patient's history, physical examination, and radiologic studies were reviewed.

Results. Standard cervico-cerebral arteriogram demonstrated focal dissection at C4 and thromboembolic complications in distal vertebral and basilar arteries. Initially, diagnosis by magnetic resonance angiogram was illusive. However, arteriography allowed prompt diagnosis followed by anticoagulation with resolution of neurologic symptoms.

Conclusions. Vertebral artery dissection without trauma is rare, but should be considered when neurologic symptoms accompany physiologic cervical movements. For cases in which vertebrobasilar thromboembolic ischemia is suspected, magnetic resonance angiogram may prove inadequate for demonstrating the causative vascular pathology. Therefore, standard cervico-cerebral arteriography should be performed. [Key words: anomaly, cerebellar infarct, dissection, ischemia, obstruction, transverse foramen, vertebral artery] *Spine* 2000;25;1989–1992

Vertebral artery injury by various mechanisms has been reported. This problem, which can occur after chiropractic manipulation and other types of trauma, has been described extensively in the literature.^{5,7,8,11,12,14} Spontaneous dissection is a far less common disorder. Although termed “spontaneous,” in the absence of arteriopathy, unrecognized or forgotten trauma may be the cause despite the patient's inability to recall an etiologic

event.³ Several authors have reported vertebrobasilar infarction or ischemia as a result of physiologic head and upper extremity motions, most commonly hyperextension and lateral rotation.^{2,3,5,15} Arteriographic abnormalities at the atlantoaxial level were described in all cases. The following case report describes a young woman with recurrent vertebrobasilar ischemia from vertebral artery injury caused by anomalous entry of the artery into the transverse foramen at C3 on the left, in contrast to the usual entry at C6.

■ Case Report

A 27-year-old, right-handed female mail carrier experienced dizziness without vertigo while turning her head to the right and reaching backward with her right hand to pick up a bundle of mail for delivery. She also experienced simultaneous onset of left upper extremity paresthesia and a pressure-like discomfort in the upper cervical and occipital region. She next noticed onset of headache with nausea and photophobia as well as left upper extremity paresis. She sought emergency room evaluation and was observed overnight. Her left arm weakness resolved over 5 hours. Workup included a normal echocardiogram and electrocardiogram. She continued to experience right occipital and intermittent bilateral pressure-like occipital headaches.

Four weeks after the first episode, the woman experienced onset of occipital headache and orthostatic-type dizziness, with transient weakness and paresthesia of the left arm while performing the identical work activity. Neurologic symptoms resolved over 1 hour. However, the next day she sought neurologic consultation after recurrence of left-hand paresthesia and blurred vision.

Physical examination showed normal blood pressure without orthostasis. No bruits were noted in the head or neck. Neurologic examination was normal. The woman's history was significant for typical migraine-type headaches with an aura of scintillating scotoma and associated nausea, vomiting, photophobia, and phonophobia. Brain magnetic resonance imaging (MRI) showed a small left cerebellar hemisphere lacunar infarction without explanatory vascular abnormalities. Cervical spine MRI was normal. She was placed on sustained-release verapamil 180 mg daily and aspirin twice daily as prophylactic treatment for presumed complicated migraine. The workup also included a normal electroencephalogram. Her hemoglobin A1-C, thyroid panel, complete blood count, erythrocyte sedimentation rate, anti-nuclear antibodies, and standard test for syphilis were normal.

One year later (October 1998) while performing the same head and arm movement as a mail carrier, the woman experienced left upper extremity paresthesia, weakness, and clumsiness associated with headache, nausea, and lightheadedness with true vertigo. She was admitted to the hospital. Her brain

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Figure 1. Spin-echo T2-weighted axial magnetic resonance image of the brain showing an acute left inferior cerebellar hemisphere infarction.

computed tomography (CT) scan was normal, but her brain MRI revealed a new “acute left inferior cerebellar hemispheric infarction without mass effect or hemorrhage” (Figure 1). The magnetic resonance angiogram (MRA) showed fetal origin of posterior cerebellar arteries bilaterally and a mild narrowing in the distal left vertebral artery (Figure 2). The radiologist considered this of doubtful significance and partly related to artifact.



Figure 2. Two-dimensional time-of-flight sagittal magnetic resonance angiogram of the cervical spine showing narrowing of the distal left vertebral artery initially considered to be of doubtful significance and partly related to artifact.



Figure 3. Lateral view of a conventional left vertebral arteriogram showing left vertebral artery compression at the C4 vertebral as the artery courses posteriorly to enter the transverse foramen of C3.

After 24 hours, all symptoms resolved. However, on the subsequent day the woman experienced right-side neck pain, right upper extremity weakness, gait ataxia, speech dysrhythmia, and transient headache. On the third day, she again was evaluated neurologically. She was normotensive with mild cervical paraspinal muscular tenderness without meningismus. Her speech was mildly hesitant, but she had normal volume and no dysarthria. Her cranial nerves were intact without a Horner’s syndrome, and her reflexes were increased slightly in the right arm. She demonstrated mildly clumsy, irregular, rapid alternating movements in both upper extremities, more on the right, and had difficulty with tandem walk. An arteriogram showed that the left vertebral artery coursed anteriorly along the spine to the C4 vertebral body, where the vessel then passed posteriorly to enter the C3 transverse foramen (Figure 3). At C4, there was a focal area of internal dissection with intimal irregularity and less than 50% stenosis. Provocative neck maneuvers, including flexion, extension, rotation, and arm positioning, did not increase stenosis or alter the caliber of the left

vertebral artery. The distal left vertebral artery appeared to be smaller than observed on the magnetic resonance angiogram, and the flow in the basilar artery was sluggish, suggesting thrombosis. A hypercoagulability workup was negative. She was heparinized, then switched to warfarin sodium. She has experienced some recurrent dizziness, but her blood pressure and neurologic status have remained stable. Subsequently, she has been restricted from the characteristic head and upper extremity movement presumed to be the cause of the ischemic events, and has not experienced further neurologic symptoms. At this writing, the woman's typical migraine-type headaches are controlled with the calcium channel blocker, verapamil.

■ Discussion

"Spontaneous" dissection of the vertebral artery is an uncommon disorder. Unrecognized trauma or vigorous neck motion is thought to be the cause in most cases. Haldeman et al⁸ recently published a detailed literature review of vertebral artery dissection to determine whether the incidence of this disorder can be reduced by assessment of reported precipitating neck movements and risk factors. Specific neck movements or trauma and potential risk factors including age, gender, migraine, hypertension, diabetes, smoking, oral contraceptives, and vasculopathy were examined in an attempt to identify factors that potentiated the occurrence of this disorder. However, the authors were unable to support any specific neck movement or risk factors that correlate with a higher likelihood of vertebrobasilar artery dissection.

The woman described in the case report experienced left vertebral artery injury during physiologically normal neck and arm movements that she performed repetitively as part of her normal work activities. Her only known potential risk factor was a history of migraine headaches. Vascular injury probably resulted from the anomalous left vertebral artery anatomy. The vertebral artery followed a course from its origin, traveling anteriorly to the spine as it ascended. The artery then passed posteriorly at C4 to enter the C3 foramen transversarium. Intimal irregularity and focal dissection were noted at this level. This abrupt change in course and compromise by the bony anatomy most likely caused focal injury. Local compression coupled with motion resulted in intimal injury, thrombosis, and cephalad thromboembolic involvement of the basilar artery. This vascular lesion was thought to be the cause of the woman's intermittent neurologic symptoms and left cerebellar ischemic stroke.

Most commonly, the vertebral arteries are bound by fascia,¹¹ thereafter traversing the neck to enter the transverse foramen of C6 by passing between the tendinous interdigitations of the scalenus anterior, longus colli, and scalenus medius at their vertebral attachments.¹⁰ The second portion of the vertebral artery then proceeds through the bony rings formed by the transverse foramina of the upper six cervical vertebrae.

Adachi¹ investigated the site of vertebral artery entry into the foramina of the cervical vertebrae in 500 autopsy cases. In 95% of the cases, the vertebral artery entered the transverse foramen at C6. Only 3.6% entered at C5, and 1% at C4. Adachi found no case in which the entry site was at C3. Daseler and Anson⁴ published similar results in their study of the subclavian artery and its branches.

Anomalous vertebral artery entry into the transverse foramen of C3 was reported in a 35-year-old Japanese man who experienced recurrent quadriplegia and locked-in syndrome.⁶ Arteriography demonstrated obstruction of the right vertebral artery at the upper edge of C4, with displacement of the artery and surrounding soft tissues provoked laterally by rotation of the head and neck to the right. Head rotation to the right was presumed to cause compression of the vertebral artery at its anomalous entry site, resulting in vascular obstruction and brain stem ischemia.

Other studies have demonstrated three clinical situations and sites wherein obstruction of the extraluminal vertebral artery is likely to occur as a result of head turning.¹¹ Sites reported liable to compression include the junction of the first and second portions of the vertebral artery where it enters the foramen, with compromise by adjacent muscular and fascial insertions. Compression in the second portion is caused by juxtaposed osteophytes, usually at C4–C5 and C5–C6, and compression or elongation of the artery is caused by sliding structures of the atlantoaxial joint.¹¹

The predilection for compression at the junctions of these vertebral artery segments is related most likely to the anatomic fact that the first and third segments are mobile, whereas the second and fourth segments are relatively fixed. Investigators also have reported occlusion of the vertebral artery during head rotation, which was thought to be caused by extrinsic constriction by the deep cervical fascia.⁹ Cervical spondylotic changes adjacent to the artery as it courses within the transverse foramen also have been demonstrated to displace the artery, most often without compromising its flow.¹³

The reported case demonstrates the importance of clinical suspicion for vertebral artery dissection when even vague neurologic symptoms accompany normal physiologic cervical movements. Magnetic resonance angiogram did not clearly demonstrate the anatomic variant or the extent of arterial injury and thromboembolic consequence. Therefore, standard cervicocerebral arteriography was necessary to verify diagnosis and institute appropriate medical therapy.

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