

A Painful Stroke: Vertebral Artery Occlusion Presenting as Cervical Radiculopathy and Headache

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ABSTRACT

Background and Objectives: Vertebral artery dissection (VAD) is an increasingly recognized etiology of stroke in patients younger than 45 years. Clinical presentation of vertebral dissection can be quite variable, including cerebellar signs, neck pain, brainstem stroke, or occasionally spinal infarctions. We present a case of VAD presenting as cervical radiculopathy.

Summary of Case : A 43 year old man presented to the emergency department with a one day history of headache and nausea without appreciable neurological deficits. He was discharged with a diagnosis of gastroenteritis and treated symptomatically until he returned the next day with radiating arm pain and paresthesia. Neurologic evaluation identified a mild imprecise dermatomal primary sensory loss in his left arm. Head CT identified a left cerebellar infarction. MRI clarified the presence of a cerebellar stroke as well as a notable spinal cord infarction in the anterior spinal artery (ASA) territory. CTA showed occlusion of the left vertebral artery and non visualization of the ASA.

Conclusions: While most stroke patients manifest without pain, in the absence of trauma, acute onset radiculopathic arm pain can rarely be a manifestation of spinal cord infarction from VAD and should be considered when other explanations are elusive or a simultaneous posterior circulation stroke is identified.

Key words: vertebral artery dissection, vertebral artery stenosis, stroke, anterior spinal artery

INTRODUCTION:

Vertebral artery stenosis or aneurysmal dilatation/dissection occurs due to disruption of the arterial wall. The process may be initiated by major or minor neck trauma or without evident injury, known as spontaneous vertebral artery dissection (VAD). VAD affects people of all ages with peak incidence in the fifth decade.¹⁻⁸ VAD is not gender-linked. Annual incidence of spontaneous VAD is 2.5 – 3 per 100,000.^{1,4,5} While posterior neck pain is common with VAD, neither VAD or acute ischemic stroke typically present with radiculopathic limb pain.⁹

Case History:

A 43-year-old Swiss man, presented to the emergency department with one day

complaint of headache and nausea. He was discharged with a diagnosis of flu-like illness and gastroenteritis. The following day he returned with radicular pain in left arm. He was evaluated for acute coronary syndrome, and his electrocardiogram and bloodwork were unremarkable. Neurologic examination revealed an unimpressive general physical examination with mild decreased sensation to light touch, and sharp modalities, in the left C4, C5, and C6 distribution and incoordination of finger-to-finger-to-nose and rapid alternating movement testing. During his exam he repeatedly combed his hair with his hand and rotated his neck to the right in a tic-like pattern. His primary care physician in Europe provided a simple medical history positive for mild chronic paranoid

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successfully controlled with antipsychotic medications, which he continued to take reliably. The patient denied tobacco, drugs, or frequent alcohol use, and he only took his prescribed psychiatric medications. He denied prior episodes, herniated discs, dizziness, vertigo, hypertension, chest pain, shortness of breath, or family history of stroke or neurological disease. Noninfused head CT showed a left-sided cerebellar ischemic infarction (Figure 1) in the posterior inferior cerebellar artery (PICA) territory. Systemic anticoagulation with heparin was initiated. He was stable and transferred to the neurocritical care unit for further evaluation and management. CTA revealed occlusion of the left vertebral artery (Figures 2-4). Subsequent MRI demonstrated cervical spinal cord infarction (Figures 6 & 7). Heparin infusion continued for two days with aPTT goal of 60-80 seconds prior to conversion to warfarin therapy. Over the ensuing 48 hours, his symptoms of radiculopathy and incoordination dramatically improved to near resolution. By day eight following initial symptom onset, he had no appreciable neurological deficits and did not require rehabilitation. Weeks later as an outpatient he remained without any neurologic sequelae. Vertebral or carotid artery dissection may be precipitated by minor events such as high velocity cervical manipulation, hyperextension or rotation injury in yoga positioning, overhead painting, coughing, sneezing, recent respiratory tract infection, hyperextension during intubation, or cardiopulmonary resuscitation.^{1,2,3,5,8,11,12} Cervical radiculopathy accounts for less than 1% of clinical manifestations of vertebral artery dissection.⁹ Typical symptoms include pain in the back of the neck or head, followed by ischemia in the posterior circulation.¹ The initial manifestations of VAD are less distinct than those of carotid dissection and are often misdiagnosed as musculoskeletal.^{1,13} Two thirds of patients present with occipital headache, but rarely generalized

may develop over the subsequent two weeks, but typically occur within 15 hours of headache onset.¹³ Unilateral arm pain or weakness as a result of C5 or C6 root involvement, usually at the level of C5-C6 or spinal epidural hematomas are unusual occasional manifestations of VAD.^{1,15} Ischemic symptoms of the lateral medulla (Wallenberg's syndrome), thalamus, and cerebral or cerebellar hemisphere occur in more than 90% of cases.^{1-3,8,13} Isolated ischemia of the cervical spinal cord is an uncommon but possibly under-recognized complication of VAD.¹⁶ MRA has reduced the demand for conventional angiography as a gold standard in the diagnosis of VAD, because MRA can show intramural hematoma and does not require ionizing radiation or risk of catheterization.^{1,17-18} CTA also reduces the necessity of catheter angiography, as it is rapidly performed at the time of admission without embolic risks associated with catheterization. Anticoagulation is often acutely administered in the absence of relative contraindications, such as intracranial extension of the VAD or hemorrhage. While its indication may be most compelling in the setting of dissection associated artery to artery embolism, antiplatelet therapy is also an accepted initial mode of management.^{1,9} Most VAD heal spontaneously. However, surgical or endovascular treatment is reserved for patients suffering persistent symptoms of ischemia despite medical therapy.^{1,19} Prognosis following VAD is generally good except in patients with large territorial infarctions or lack of adequate collateral blood supply. Headache typically resolves within 3-7 days in more than 90% of patients.^{1,13} Reported mortality rate associated with dissection of both carotid and vertebral arteries is less than 5%, and about 75% of patients have excellent functional recovery.¹⁻⁴ Following VAD, the risk of recurrence is 2% at one month, and 1% per year thereafter for a decade or longer. However the risk of recurrent dissection in young patients with hereditary

PATIENTS AND METHODS:

Spontaneous dissection of the vertebral artery is increasingly recognized as an etiology of stroke in younger people, often without classic risk factors. Also, most often VAD causes no permanent deficits if diagnosed and treated in a timely fashion. However, ischemia to the inferior territory supplied by the vertebral arteries, including the anterior spinal artery, represents one of few possible etiologies where stroke can

elicit pain. Further, like in this case, neck pain with radiculopathy may easily be mistaken for cervical disc disease, but spontaneous non-provoked onset and, in particular, simultaneous posterior circulation infarction(s) should raise suspicion of a dialation vascular posterior artery complicated by spinal cord infarction. Since such complications can lead to more serious, life-threatening disability, prompt diagnosis, monitoring and treatment can profoundly impact

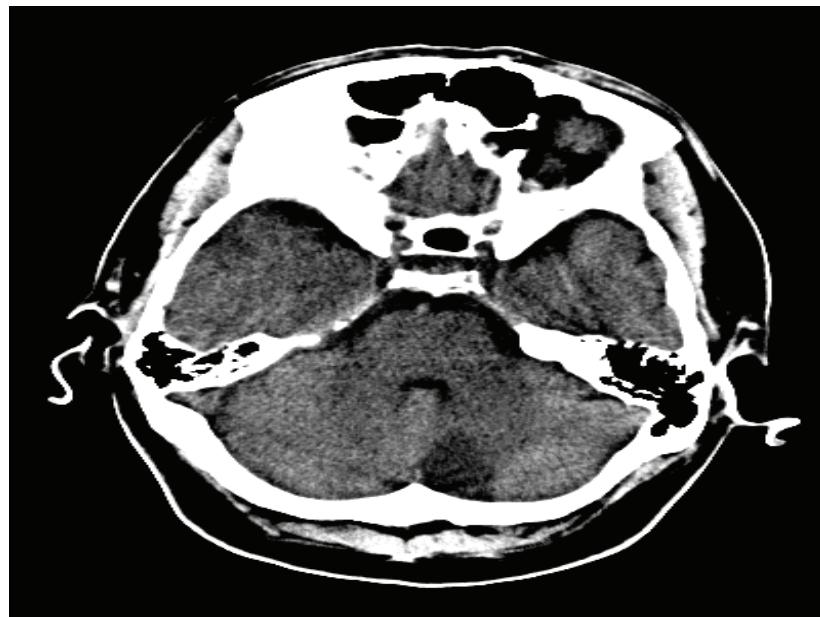


Figure 1. Noninfused CT head showing left cerebellar infarction (PICA territory).

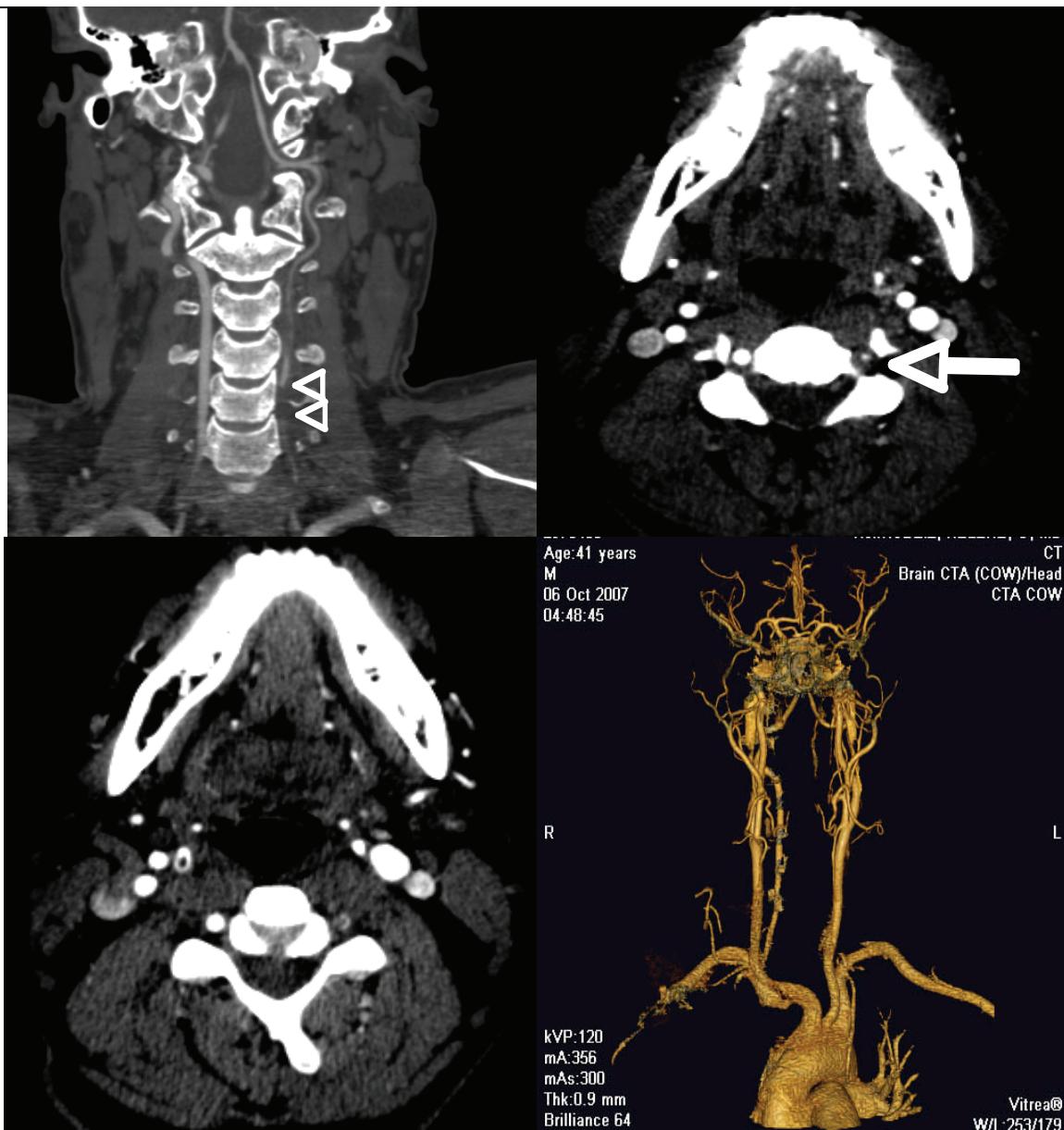


Figure 2. CTA head and neck shows left vertebral artery occlusion (Panels A-D).

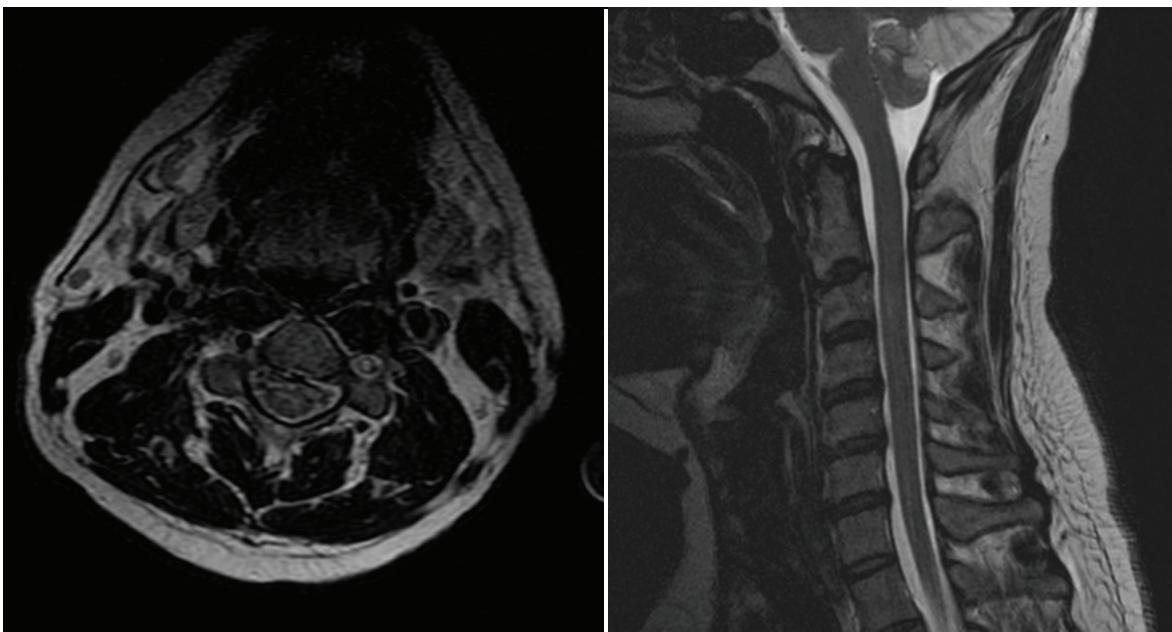


Figure 3. MRI neck identifies the cervical cord infarction with clot in the left vertebral artery and hyperintensity in the anterior spinal artery territory on MRA. The MRA reveals ASA occlusion and left VAD

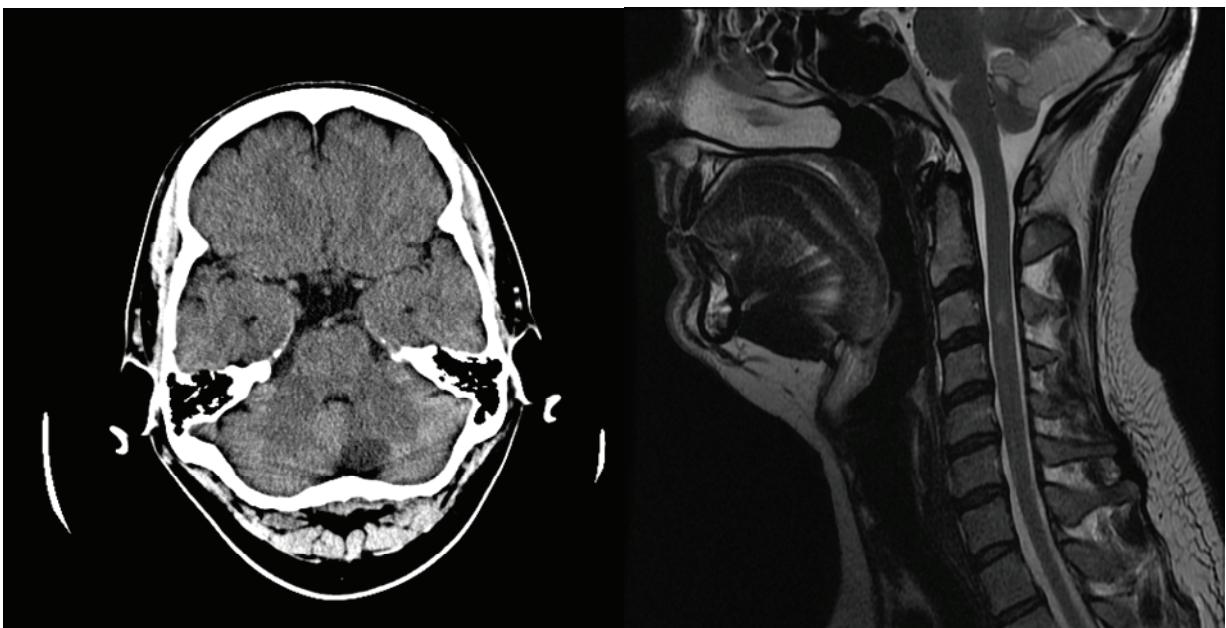


Figure 4. Follow-up CT and MRI identifies the evolved left PICA stroke as well as the area of ischemia in the C2 cord.

CONCLUSION

Spontaneous dissection of the vertebral artery is increasingly recognized as an etiology of stroke in younger people, often without classic risk factors. Also, most often VAD causes no permanent deficits if diagnosed and treated in a timely fashion. However, ischemia to the inferior territory supplied by the vertebral arteries, including the anterior spinal artery, represents one of few possible etiologies where stroke can elicit pain. Further, like in this case, neck pain with radiculopathy may easily be mistaken for cervical disc disease, but spontaneous non-provoked onset and, in particular, simultaneous posterior circulation infarction(s) should raise suspicion of a dialation vascular posterior artery complicated by spinal cord infarction. Since such complications can lead to more serious, life-threatening disability, prompt diagnosis, monitoring and treatment can profoundly impact

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