A 57-Year-Old Man With Headache, Numbness, and Weakness

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Abstract

We present the case of a 57-year-old man with headache, numbness, and weakness. We review the differential diagnosis and discuss the relevant imaging. Once the diagnosis is evident, we review the natural history and evidence in support of each possible treatment.

Keywords

stroke, vertebral artery dissection, imaging

A 57-year-old ship captain with hypertension presented with 3 days of right posterior auricular pain, abnormal sensation on the left side of his face, and weakness in the left leg following a turbulent boat ride. He had reduced sensation to light touch and pinprick over his left face and body along with mild hemiparesis affecting the left arm and leg.

Diagnosis for this subacute process with combined motor and sensory complaints begins with neuroanatomical localization. The deficits for this hemibody syndrome localize to a right thalamocapsular, brain stem, or high cervical lesion. The rapid symptom onset suggests a vascular etiology. Possibilities include hypertensive hemorrhage or infarct from small vessel disease, embolism, or arterial dissection. Other causes of painful stroke, such as reversible cerebral vasoconstriction syndrome and posterior reversible encephalopathy syndrome should also be considered. Subarachnoid hemorrhage, potentially as a consequence of dissection, could explain the headache but is less likely without decreased level of consciousness. The age, preceding trauma of a turbulent boat ride, focal retroauricular pain, and posterior circulation localization make vertebral arterial dissection (VAD) the most likely possibility though other possibilities, such as hemorrhage, need to be excluded.

Vertebral artery dissection typically causes medullary ischemia. In this patient, the left hemiparesis sparing the face implicates the right medullary pyramid, prior to the decussation. The medial lemniscus, which carries postdecussation sensory fibers to the thalamus, runs just dorsal to the pyramid. A right medullary lesion could therefore give a face-sparing left-sided hemiparesis and sensory loss. Medullary lesions can cause facial numbness on either side from involvement of the spinal trigeminal nucleus or tract. However, the spinal trigeminal nucleus and tract are somewhat removed from the

pyramids, and all but large lesions generally involve one or the other. The spinal trigeminal nucleus extends caudally into the cervical spine, and in rare cases, a rostral cervical myelopathy can produce facial numbness, though typically contralateral to the symptoms in the body. In clinical practice, one should also be aware that, in the acute setting, altered sensory complaints are frequently variable and often do not conform perfectly to expected neuroanatomical distributions.

Urgent cerebrovascular imaging is indicated in this setting to provide better diagnostic clarity.

This patient underwent a computed tomographic angiogram (CTA), which showed a tapering occlusion in the distal right vertebral artery extending intradurally, concerning for a vertebral dissection (Figure 1). Magnetic resonance imaging (MRI) revealed an area of restricted diffusion in the right medulla, involving the precrossing pyramid and the medial lemniscus, consistent with an acute ischemic infarct (Figure 1).

Interpretation of vertebral artery imaging is difficult because of the small caliber of vertebral arteries and frequent congenital asymmetry between the 2 arteries. The historical standard for diagnosing vertebral artery dissection is

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Figure 1. Radiographic findings. Axial computed tomographic angiogram (CTA) showing decreased opacification in the right vertebral artery (A). Coronal CTA showing lack of opacification in the right V4 segment (B). Magnetic resonance imaging (MRI) fluid attenuation inversion recovery (FLAIR) sequence showing hyperintensity in the right vertebral artery suggesting slow or absent flow (C). MRI T1 sequence showing absence of a flow void in the right vertebral artery (D). Axial (E) and coronal (F) diffusion-weighted imaging (DWI) sequences showing an area of restriction in the right rostral medulla.

conventional angiography, which usually shows smooth or slightly irregular tapering, stenosis, or complete occlusion. These findings, however, are nonspecific. More specific signs such as a double lumen or an intimal flap are seen in fewer than 20%.¹ The sensitivity and specificity of conventional angiography for VAD remain unknown despite its use as the “gold standard.”

Noninvasive modalities such as CTA and magnetic resonance angiography (MRA) are increasingly used to diagnose VAD. Computed tomographic angiogram demonstrates the same findings as conventional angiography and is faster than MRA but requires intravenous contrast and exposes the patient to radiation and nephrotoxicity. Magnetic resonance imaging can highlight thrombus in the vessel wall with T1 “fat-saturation” sequences, and MRA can be performed with or without gadolinium contrast. When compared to conventional angiography, the reported sensitivity and specificity of CTA vary widely but appear to have sensitivity of at least 60% and specificity greater than 80%.² Sensitivity of MRA is at least 70% but specificity is lower (estimated to be more

than 65%).² The added diagnostic value of contrast-enhanced MRA to prove lumen patency has not been compared in large series with time-of-flight MRA. T1 fat-suppression sequences can show mural thrombus in the vessel wall and potentially add diagnostic accuracy, but hyperacute mural thrombus may not be detected, due to the presence of intracellular oxy- and deoxyhemoglobin that may be isointense on T1-weighted sequences.³ Furthermore, normal venous plexus within the transverse foramina can be T1 hyperintense and mistaken for mural thrombus.³ In comparing CTA and MRA, a small retrospective study showed that radiologists preferred CTA 80% of the time to diagnose vertebral artery dissection.³ In no case did MRA demonstrate the dissection more clearly, though MR imaging added complementary information such as the presence of an infarct.

If imaging done in the acute setting is inconclusive, the diagnosis of arterial dissection can be made retroactively by serial imaging. One series estimated that 64% of all vertebral dissections, 75% of stenotic dissections, and 57% of

occlusive dissections will recanalize by 1 year with standard medical therapy.⁴

Our patient had prolonged duration of minor symptoms, which precluded any acute thrombolytic therapy. Due to intracranial extension of his nonaneurysmal dissection, he was treated with aspirin 81 mg daily to reduce his risk of future strokes.

Arterial dissections generally fall into 2 categories—dissections from the intima into the media and those confined to the media and adventitia. Intimal dissections can cause stenosis and embolism from platelet thrombi aggregating along damaged endothelial surface. Medial to adventitial dissections tend to form pseudoaneurysms. Dissections extending intracranially pose risk of rupture because vertebral arteries lose medial elastic fibers and adventitial thickness after piercing the dura.⁵

Data on the natural history of VADs are limited to treated patients. The largest historical prospective study followed 72 vertebral dissections⁶; the recent randomized controlled Cervical Artery Dissection in Stroke Study (CADISS) trial enrolled 132 vertebral dissections.⁷ Headache and neck pain are the most common symptoms, but stroke or Transient Ischemic Attack (TIA) are also quite common, present in more than 70% of patients.⁷⁻⁹ The ischemia almost always occurs in the posterior circulation and most frequently in the distribution of the posterior inferior cerebellar artery. If the dissection extends intracranially, which happens in 10% to 33%^{8,10} of dissections, subarachnoid hemorrhage is relatively common, occurring in 15% to 24%^{8,10} of this subgroup.

The management of a patient with VAD focuses around 3 questions: (1) Is there associated pseudoaneurysm formation or hemorrhage? (2) Is there infarction? and (3) Does the dissection extend past the dura?

Patients with subarachnoid hemorrhage should be referred emergently to neurosurgery for conventional angiography to identify small, dissecting pseudoaneurysms. Patients with ischemic symptoms, as this patient, should have their candidacy for thrombolysis evaluated. Limited data suggest that intravenous tissue plasminogen activator can be given safely in dissection, and theoretical concerns about wall rupture and expansion of the mural hematoma with thrombolysis have not been borne out.¹¹ In patients presenting acutely with posterior circulation dissection with proximal occlusion, there are no randomized data to guide consideration for thrombectomy such as in the anterior circulation.

Patients with nonaneurysmal vertebral dissection are generally given antiplatelets or anticoagulants for a period of time, usually for 3 to 6 months, with the goal of preventing initial or recurrent stroke. Once dissection is diagnosed and treatment is initiated, recurrent stroke risk is low, with estimates ranging from 0% over 2.5 years⁹ to 1.3% in 6 months.⁶ Prognosis is generally good with long-term modified Rankin scores less than 3 in 70% to 85% of patients.⁶ Antiplatelet and anticoagulant therapies were compared in the recent CADISS trial, which demonstrated no significant difference between

treatment groups in the 3-month primary outcome of ipsilateral stroke or death.⁷ This trial was clearly underpowered, since only 1.6% of all treated patients had a recurrent stroke, and power calculations suggested a need for at least 40 times as many total patients to detect a difference.⁷ Although the CADISS trial demonstrated the relative equivalency of antiplatelet and anticoagulant treatment, conventional wisdom suggests that anticoagulation should be avoided in select patients. Patients with a large cerebellar or posterior cerebral artery territory infarction should probably not receive anticoagulation for the first few weeks, given the risk of hemorrhagic transformation. Dissections that extend intracranially may also have increased risk of arterial rupture with anticoagulation due to the lack of an adventitial sheath covering this arterial segment. This conventional idea was questioned by an observational study of 81 patients with intracranial vertebral and carotid dissections without subarachnoid hemorrhage, who did not have adverse outcomes despite treatment with anticoagulation.¹⁰ This study, however, included only a small number of vertebral dissections. Most of the patients who had hemorrhage on presentation had dissections within the posterior circulation, indicating a theoretical predisposition to hemorrhage. Indeed, the recent CADISS trial included a patient with intracranial vertebral dissection who had a subarachnoid hemorrhage after starting warfarin.⁷

Optimal duration of treatment is unknown. The risk of stroke seems to be the highest in the first weeks and is remote after several months.^{6,7} Following patients angiographically to look for recanalization is done commonly but does not seem to have any bearing on risk of stroke recurrence.⁹

In the rare case that stroke or TIA recurs after treatment is initiated, the first step is to repeat imaging and determine whether the symptoms are likely caused by the ongoing embolism or flow limitation from the stenosed segment. If embolism is suspected, the standard approach is to escalate medical therapy—adding anticoagulation if an antiplatelet was used or adding an antiplatelet if anticoagulation was being used. Although the indications for endovascular or surgical intervention on a dissected vertebral artery are not firmly established, recurrent symptoms on combined antiplatelet and anticoagulant therapy are generally considered an indication for endovascular stenting or occlusion of the affected artery.¹² Intractable flow limitation to the posterior circulation from extension of the dissected, stenotic segment, is an additional indication for endovascular intervention. Surgical bypass is now only performed in cases in which endovascular intervention is not feasible or has failed.¹²

Discussion

Vertebral artery dissection is a common cause of posterior circulation stroke in the young patients. Minor or major neck trauma precedes approximately 35% of all VADs.⁶ Fibromuscular dysplasia is identified in 8% to 11% of patients with VAD without major preceding trauma.⁸ Smoking, diabetes,

and hypertension may also confer increased risk.⁸ There is no convincing data to suggest a difference in the risk of stroke between spontaneous and traumatic dissections. The overall stroke rate in modern series of traumatic cervical arterial dissections ranges from 16% to 34%.¹³⁻¹⁵ This rate is lower than that seen in the CADISS trial, in which 80% of patients had a stroke as the presenting symptom.⁷ The discrepancy may be because most traumatic dissections are identified by screening while many spontaneous dissections do not come to clinical attention unless stroke symptoms develop. Once antiplatelet or anticoagulant treatment has been initiated, traumatic dissections have stroke rates ranging from 0% to 9%,¹³⁻¹⁵ comparable to the 1.6% rate identified in the CADISS trial.⁷

Historically, vertebral dissections have only been detected after a stroke. With improved noninvasive imaging, an unresolved question is whether a subset of patients with VAD and headache can be identified and treated early enough to prevent an initial stroke. Evidence from patients with trauma, in whom screening routinely identifies asymptomatic dissections, supports this conclusion.¹³⁻¹⁵ An estimated 13% of patients who present with stroke from spontaneous arterial dissection have transient neurologic symptoms that precede the stroke by at least 1 day.⁸ In a post hoc analysis in the CADISS trial, 19 of 194 patients identified a TIA or minor stroke in the days preceding their presenting stroke.⁷ Paradigms to determine when to image patients for apparent minor symptoms and correct interpretation of these results may decrease the morbidity from dissection through earlier treatment.

What is known with certainty is that recurrent stroke on medical therapy is rare, occurring in only 1.6% of 250 patients in the recent CADISS trial.⁷ The decision to use antiplatelets or anticoagulation should be individualized, but the optimal approach is still unknown. One can easily argue for the relative safety or the relative inefficacy of anticoagulation, but practically, the difference between the 2 therapies is likely minor.

Declaration of Conflicting Interests

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