Vertebral Artery Dissection (VAD) refers to a non-hemorrhagic cerebrovascular event caused by a flap-like tear in the vertebral arteries of the cervical vasculature. VAD events along with carotid artery dissection (CAD) are generally spontaneous and are among the more common causes of ischemic stroke in the younger patient population. The clinical sequelae of VADs follow that of posterior circulation infarctions. Signs and symptoms can range from vision loss to dysphagia. We present the case of a patient who underwent a lumbar laminectomy at an outside hospital, and five days later presented to our institution with hoarseness. He was later found to have a left VAD.

Case Report
The patient is a 64-year old man who initially presented to our emergency room (ER) from his neurologist’s office with a chief complaint of worsening hoarseness. He has a past medical history of hypertension, hypercholesterolemia, and restless legs syndrome. The patient denied any other complaint—no headaches, diplopia, weakness, dizziness, dysphagia, odynophagia, nausea, vomiting, slurred speech, or focal weakness. His vitals and lab results were unremarkable. Physical exam was only significant for voice hoarseness and a slight right pronator drift. The rest of his exam was otherwise normal.

Prior to his presentation, the patient had undergone a lumbar laminectomy approximately 45 days prior. Five days after the procedure, the patient noticed his voice was changing; it was slowly becoming more hoarse. The patient had undergone outpatient otolaryngology evaluation from two different surgeons for his hoarseness. After laryngoscopic evaluation, the patient was found to have vocal cord paralysis. Electromyography later confirmed that he had 20 percent activity of his left cricothyroid muscle (innervated by the superior laryngeal nerve) and 20 percent activity of his left posterior cricothyroid muscle (innervated by the recurrent laryngeal nerve). The patient’s right side was normal. Subsequent neuroimaging via magnetic resonance imaging and angiography (MRI/MRA) revealed a dissection of the intracranial left vertebral artery, as well as multiple small...
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acute/subacute embolic ischemic strokes of the posterior circulation—right inferior cerebellar peduncle, left midbrain, and left thalamic regions (Figure 1). The suggestion of brain stem infarctions by MRI led to the recommendation of emergent CTA imaging for further characterization of the cervical and intracranial vasculature with possible intervention.

The patient was admitted to a telemetry unit for neuroendovascular treatment. A CTA of the head and neck confirmed the dissection of the intracranial left vertebral artery without other neurovascular pathology. The patient was aspirin and plavix loaded the night before his procedure. The next day he had undergone femoral cerebral angiography and intracranial stenting of the left vertebral artery (Figure 2a & 2b). The procedure and recovery were uneventful. A follow up MRI/A two months later showed flow within the stent. NOVA blood flow analysis demonstrated antegrade flow in the left vertebral artery and the basilar artery with no residual pseudoaneurysm (Figure 3). A follow-up EMG performed two months from the initial exam showed 60 percent function of his left-sided cricothyroid and posterior cricothyroid muscles. Clinically, the patient has near 100 percent recovery from his hoarseness.

Discussion

The vertebral arteries are major arteries of the cervical chain. These arteries originate as branches off the bilateral subclavian arteries and merge into a single basilar artery that feeds the posterior part of the Circle of Willis. The vertebral artery is divided into four segments: V1 (pre-foraminal), V2 (foraminal), V3 (C2 to dura), and V4 (intradural). The vertebral arteries are the primary arteries of the medulla; each supplies the lower three-fourths of the pyramid, the medial lemniscus, all or nearly all of the lateral medullary region, the restiform body, and the posteroinferior part of the cerebellar hemisphere through the posterior inferior cerebellar arteries (Figure 4).

Although there are two vertebral arteries, the left vertebral artery typically dominates with regards to blood flow delivery to the brain. The relative sizes of the vertebral arteries vary considerably, and in approximately 10 percent of cases, one vessel is so small that the other is essentially the only artery of supply to the brainstem. In the latter cases, if there is no collateral flow from the carotid system via the circle of Willis, occlusion of the one functional vertebral artery is equivalent to occlusion of the basilar artery.
Vertebral artery dissections primarily affect middle-aged people with men and women at seemingly equal risk. Precipitating causes of VADs include trauma (e.g., sports injuries or motor vehicle accidents) or spinal manipulation therapy. The V2 and V3 vertebral segments are reported to be the most common areas for spontaneous dissection. Characteristics of at-risk patients include active smokers, hypertension, diabetes, hyperlipidemia, family history of stroke, oral contraceptive use, history of migraines, and history of neck trauma.

The clinical presentation of vertebral artery dissection typically includes headache or neck pain with or without deficits in brain stem function. Given its anatomical role to the posterior circulation of the brain, signs and symptoms may also often reveal those of basilar artery insufficiency—including vertigo, unilateral facial paresthesias, and cerebellar signs. VADs patients have also been reported to succumb to lateral medullary syndrome, medial medullary syndrome, Horner’s syndrome. Ophthalmic manifestations have also been reported, and may comprise of diplopia, blurred vision, or visual field defects.

What makes our patient unique is that after his lumbar laminectomy he presented with the isolated symptom of hoarseness, which suggests that the vascular territory affected was in the issuing fibers of cranial nerves nine and ten. The proposed mechanism of injury is likely a spontaneous dissection of his V4 segment, as there was no direct alteration to the cervical spine either via manipulation therapy or trauma.

V4, the intracranial portion of the VA, extends from the foramen magnum to the basilar artery. The intracranial portion of the VA gives rise to three major branches: the posterior inferior cerebellar artery (PICA), the anterior spinal artery, and the posterior spinal artery. Dissections of the V4 segment can be classified as either traumatic or spontaneous. In the majority of cases, intracranial VA dissection occurs spontaneously. Several diseases have been associated with spontaneous cervical artery dissection. These diseases include hereditary connective tissue disorders, Marfan syndrome, Autosomal dominant polycystic kidney disease, and Osteogenesis imperfecta type I. Hyperhomocysteinemia, alpha-1 antitrypsin deficiency, and abnormalities of neural crest cells have also been associated with an increased risk of spontaneous cervical dissections.

Optimal therapy for vertebral artery dissections are based upon expert opinion. As of now there are no randomized control trials comparing medical versus surgical treatment modalities. To avoid the dreaded thromboembolic complications of vertebral artery dissections, we recommend anticoagulation with intravenous heparin with bridging to Coumadin with a goal INR of 2-3 for a duration of three to six months. To our knowledge, there is at least one published small clinical trial examining the outcomes of novel anticoagulants (NOACs) in 68 patients showing no difference in safety outcomes with vitamin K antagonist therapy. These recommendations are irrespective of the symptomatology of the patient. However, there are incidences when anticoagulation is contraindicated. These incidences include the presence of mass effect due to a large infarct, the presence of hemorrhagic conversion of the infarcted area, and the presence of an intracranial

Figure 3. Left VA 2 months later (A). Non-invasive Optimal Vessel analysis (NOVA) demonstrates antegrade flow in the left vertebral artery status post stenting (B).

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Aneurysm as well as in cases with extension of an aneurysm intracranially.9

Many vertebral artery dissections may heal without intervention, however, surgical intervention is indicated with the presence of a subarachnoid hemorrhage. Additionally highly symptomatic or Vertebral artery dissections that have not improved or healed after six months of anticoagulation also need consideration for surgical repair.7

When surgical intervention is pursued, it is preferably done so via endovascular methodology for several reasons. As compared to surgical ligation or bypass of the vertebral artery, endovascular technique is associated with lower complication rates. Additionally endovascular methods allow prompt retrieval of blood flow through the vessel lumen and enhance the ability to evaluate for potential pseudoaneurysms.8

In our patient, who presented with a posterior ischemic circulation symptom, the goal of therapy was to prevent further thromboembolic events. Typical management as mentioned before would include anticoagulation. Patients with ischemic symptoms and a “pearl and string” appearance on angiography should also be considered for endovascular coil occlusion therapy, because a VA dissection with this aneurysmal morphology has the potential to grow and eventually rupture.16 Stent-assisted angioplasty may also be considered in conjunction with anticoagulation, as stents allow for the dissected segment to unionize with the vessel wall and thereby eliminate the false lumen, decreasing the occurrence of further embolic events.17

Conclusion

Vertebral artery dissections commonly affect the younger to middle-aged patient population. This vascular injury commonly occurs secondary to trauma or spinal manipulation, and these patients present with symptomatology reflective of posterior circulation ischemia. Treatment of VADs generally is amenable to antiplatelet or anticoagulation therapy. Endovascular recanalization therapy may also be considered. General follow-up with MRI or angiography is recommended three months after initiation of therapy.

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