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Spontaneous Vertebral Artery Dissection in a Healthy 26 Year Old Female Patient A Case Study

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CASES

ΝΟΤΕ

Abstract

Spontaneous vertebral artery dissection (SVAD) is an extremely rare, yet life-threatening, event that can potentially result in ischemic stroke or subarachnoid hemorrhage, depending on the origin and extension of the dissection. Vertebral artery dissection is more commonly associated with traumatic injury to the neck, resulting in compromised structural integrity of the vertebral artery wall. This case study discusses the clinical presentation, physical examination, diagnosis, clinical course, and outcome for a young, otherwise healthy, female patient who presented to the emergency department with a SVAD. **Key words:** arterial dissection, vertebral artery

LTHOUGH THE INCIDENCE of vertebral artery dissection (VAD) is only 1 per 100,000, spontaneous vertebral artery dissection (SVAD) is considered to be even more rare, accounting for only 2% of all ischemic strokes (Rodriguez, Berrios, & Ramos, 2016). Studies have shown that VAD may account for as many as 20% of ischemic stroke events in patients younger than 45 years (Rodriguez et al., 2016), making

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diagnosis a crucial component of decreasing ischemic stroke morbidity and mortality in this young population.

Vertebral artery dissection is generally classified as extracranial, or intracranial, and traumatic, or spontaneous. There is some debate regarding whether or not the incidence SVAD is more likely attributed to vascular or connective tissue disease, structural abnormalities, infection, or simply a subtle mechanical event that the patient has not recognized as particularly traumatic (Park et al., 2008). Vertebral artery dissection has been documented in patients who have experienced symptom onset during yoga, while undergoing chiropractic manipulation, and even sneezing. One such case of a subtle mechanism made national news in 2016 when a woman was diagnosed with VAD after having her hair shampooed in a salon chair.

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PATHOPHYSIOLOGY

The structure of an arterial wall consists of three distinct layers: the tunica intima, or the innermost layer, followed by the tunica media, and the outer layer, the tunica externa. When a tear in one of these layers occurs, blood may accumulate between these layers, resulting in a collection known as an intramural hematoma. The dissection of the arterial wall develops in different ways, depending on its location. Subintimal hematomas have the potential to expand intraluminally, resulting in arterial occlusion and ischemia, whereas subadventitial dissection may result in aneurysmal dilation of the arterial wall. The most serious complication of this vessel pathology is intraluminal thrombus formation, resulting in either stagnant occlusion of the artery, or distal propagation, resulting in ischemic stroke (Seshadri & Debette, 2017).

The vertebral artery is anatomically categorized into four distinct segments, I, II, III, and IV as seen in Figure 1. Segment I (the preforaminal segment) begins at the subclavian artery and extends to C6. Segment II (foraminal segment) spans from C6 to C2. Segment III (extradural segment) extends through the foramen magnum, reaching Segment IV (intradural segment), where it adjoins with the contralateral vertebral artery to form the basilar artery. Dissection most commonly occurs in the tortuous portion of Segment III (see Figure 2), where the artery penetrates the cranium and dura (Rodriguez et al., 2016). Despite this anatomical predisposition, dissection can occur in all three extracranial segments, where the artery passes through the intervertebral foramina. In approximately 10% of cases of VAD, extracranial dissection is found to extend intracranially (Rodriguez et al., 2016).

MORBIDITY AND MORTALITY

According to Britt and Bhimji (2017), approximately 10% of patients with VAD will not

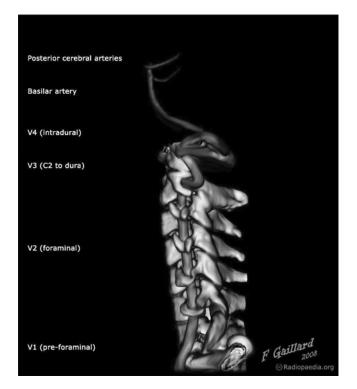


Figure 1. Segments V1-V4 of the vertebral artery. Image courtesy of Jones (2008).

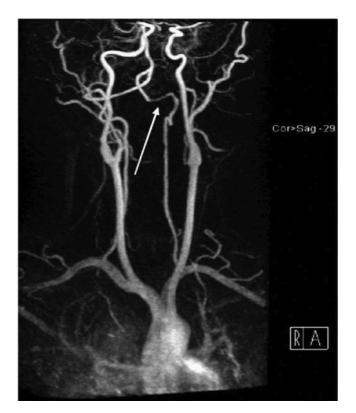


Figure 2. Left vertebral artery narrowing characteristic of dissection at the V3 segment. Image courtesy of Miller, Kotachchi, and Miller (2009).

survive due to extensive intracranial dissection, subarachnoid hemorrhage (SAH), or brainstem infarction. Those who do survive the acute phase of dissection, generally have a good prognosis (Britt & Bhimji, 2017). Patients with intracranial dissection are generally considered to be at a significantly higher risk of complication. Complications of VAD include ischemic stroke, cerebellar stroke, SAH, and cranial neuropathy as a result of a compressive vertebral artery pseudoaneurysm (Lang et al., 2017).

CLINICAL PRESENTATION

Patients with VAD may typically present with a range of symptoms, including, but not limited to, intense headache, severe neck pain, dizziness, nausea, vomiting, localized weakness, paresthesias, and visual disturbances (DeSouza, Crocker, Haliasos, Rennie, & Saxena, 2011). Physical examination findings also vary extensively depending on the location at risk for ischemia and may include visual field deficits, gait ataxia, dysarthria, dysphagia, dysesthesia, Horner's syndrome (ptosis, miosis, and anhidrosis), and Wallenberg's syndrome (sensory deficits over the ipsilateral portion of the face and contralateral body) (DeSouza et al., 2011).

IMAGING

Conventional angiography has previously been considered the gold standard of imaging for radiographic confirmation of VAD. However, in recent years, magnetic resonance imaging (MRI)/magnetic resonance angiography (MRA) and computed tomography (CT) angiography (CTA) have evolved as feasible

alternatives (Rodriguez et al., 2016). Computed tomography angiography has been found to have 100% sensitivity and 98% specificity in the diagnosis of VAD (Rodriguez et al., 2016). It is less invasive than traditional angiography, less time-consuming, and more cost-effective for initial diagnosis than MRI/MRA, with comparable sensitivity and specificity. In patients without any appreciable neurological deficit, low suspicion for acute ischemia, and normal renal function, obtaining a CTA scan is an excellent choice for initial imaging. In the presence of positive findings on a CTA scan, MRI/MRA is often done to further evaluate the dissection and any evolving ischemia, which cannot be appreciated on a CT/CTA scan. Most emergency departments have selective MRI protocols and will utilize MRI/MRA only in the presence of acute neurological deficits concerning for cerebrovascular accident or in patients presenting with suspected cauda equina syndrome.

MANAGEMENT

Treatment of VAD is aimed at preventing thromboembolic complications. In extracranial VAD without acute ischemia, antithrombotic therapy, generally by heparin drip, is initiated. In extracranial VAD with evidence of ischemia, intravenous recombinant tissue plasminogen activator (r-TPA) and endovascular interventions are common treatment options. However, in the presence of VAD that extends intracranially, thrombolysis is contraindicated, as it significantly increases the risk of SAH. Management of SVAD in the outpatient setting is generally dependent upon the location and extension of the dissection and the presence or absence of any ischemia. In the otherwise healthy adult, without ischemia, antiplatelet agents are generally preferable as outpatient treatment. In patients with ischemic changes as a result of the dissection, generally outpatient management includes oral anticoagulation acutely, with a switch to oral antiplatelet agents at 3 months after discharge (Rodriguez et al., 2016).

CASE PRESENTATION

History

A 26 year old woman with a past medical history significant only for unexplained unilateral hearing loss since childhood and unspecified chronic headaches presented for evaluation of transient episodes of visual disturbances in the form of black floaters in both visual fields and mild global headache, with simultaneous unilateral paresthesias to the left side of the face and the left arm. The episodes were described as intermittent and fleeting, lasting only 30 s to 2 min, with spontaneous resolution. The patient first noticed the episodes upon waking, 3 days prior.

The patient denied any history of similar episodes, neck pain, nausea, vomiting, fever, chills, recent trauma or physical exertion, weakness, recent falls, or gait instability. Her general review of systems was otherwise negative. She denied any current medications, illicit drug or alcohol use, and any family history of connective tissue disorders, neurological disorders, malignancies, or thrombophilia.

Physical Examination

Physical examination of the patient revealed a well-developed, well-nourished woman. She was hemodynamically stable, afebrile with normal vital signs, and well appearing, with a normal physical examination. On neurologic examination, the patient was awake, alert, and oriented and with clear, fluid, articulate speech with excellent comprehension and recall. Her face was symmetric, tongue was midline with normal eye closure, and her smile was symmetric. Extraocular movements were normal with conjugate gaze. Her pupils were round, briskly reactive at 3 mm, with normal consensual reaction and convergence. Fundoscopic examination was grossly normal, with sharp discs, and no vascular changes appreciable. Visual fields were full to gross confrontation. Visual acuity testing using a Snellen chart revealed 20/20 vision left eye (OS), right eye (OD), and both eyes (OU). Facial sensation was intact in all three distributions to sharp

touch. Right hearing was normal to finger rub; left hearing was markedly diminished, as is consistent with her reported baseline. Head turning and shoulder shrug were symmetric with normal strength. There was no pronator drift or lag, and no dysmetria noted on finger-to-nose testing. Rapid alternating movements were normal. Romberg testing was negative, and the patient's gait was noted to be smooth, steady, and nondisrupted. Tandem walking and heel-toe walking were normal. The patient exhibited symmetric, 5/5 upper and lower extremity strength bilaterally, with normal sensation throughout to sharp and dull touch. Reflexes were +2 and symmetric at the biceps, patellas, and achilles.

Clinical Course

During the patient's clinical course while the nursing staff performed venipuncture, the patient experienced a return of her symptoms, reporting left-sided paresthesias, a global throbbing headache, with floaters in her visual fields. She was also noted to become tachycardic on the cardiac monitor at 110 beats/min, with no other significant change in vital signs. A full neurologic examination was repeated during this episode, and the patient was noted to remain awake, alert, and articulate with only decreased discrimination to dull and sharp touch to the left side of the face and left upper and lower extremity. No other focal deficits were appreciable, and her symptoms resolved spontaneously within 3 min of their onset. A CTA of the head and neck, as well as a complete blood cell count, basic metabolic panel, erythrocyte sedimentation rate, C-reactive protein, human chorionic gonadotropin, electrocardiogram, troponin, urinalysis, urine drug screen, and coagulation studies, were ordered.

The differential diagnosis included conditions with the recognized potential for transient symptoms, including adult-onset complex partial seizure, complex hemiplegic migraine, transient ischemic attack, aneurysm, and anxiety. The stroke service was consulted pending CTA results. Laboratory results were significant only for mild pyuria, of which the patient was asymptomatic. The CTA scan of the head and neck revealed mild segmental narrowing of the right vertebral artery from the level of C3-C5, suggestive of VAD with intramural hematoma. The inpatient stroke service was consulted for admission; the patient was given 325 mg of aspirin, and inpatient MRI of the brain was ordered for further evaluation.

Outcome

The patient was admitted to the stroke service in stable condition and remained neurologically intact and hemodynamically stable throughout her hospital stay. An inpatient magnetic resonance image of the brain revealed no evidence of any acute infarct. The MRA scan of the patient's neck reiterated the findings on the CT scan, thus confirming a diagnosis of SVAD. The patient was discharged 2 days later on 81 mg of aspirin daily and prescribed magnesium oxide and Zofran (ondansetron) for headaches. Outpatient followup was arranged for primary care physician establishment 2 days after discharge and neurology follow-up in 1 month, with repeat MRI scheduled at 3 months after discharge.

CONCLUSIONS

While SVAD remains a rare cause of ischemic stroke and SAH in the general population, it remains a substantial provocation of stroke in children and young adults that is often difficult to recognize. The culmination of varying potential symptoms and presentations, with a lack of recognizable traumatic precipitating injury, in younger patients that may or may not be able to effectively articulate subjective symptoms, could have potentially disastrous patient outcomes if overlooked by clinicians.

In this case, the patient presented only 3 days after symptom onset. However, oftentimes, because of the presence of only transient symptoms, patients will present days or even weeks after the initial dissection occurs, often prompted to seek care when they develop stroke-like symptoms.

The clinical provider should always remain cognizant and hypervigilant when treating patients of any age with a chief complaint of headache in the presence or absence of recent trauma. In this case, the patient denied any neck pain, was neurologically intact on arrival, and asymptomatic at the time of her initial presentation. A missed diagnosis of VAD may result in death or significant resultant disability.

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