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Recognition of Spontaneous Vertebral Artery Dissection Preempting Spinal Manipulative Therapy: A Patient Presenting With Neck Pain and Headache for Chiropractic Care

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Abstract

Objective: The purpose of this case report is to describe a patient who presented to a chiropractic physician for evaluation and treatment of neck pain and headache.

Clinical features: A 45-year-old otherwise healthy female presented for evaluation and treatment of neck pain and headache. Within minutes, non-specific musculoskeletal symptoms progressed to neurological deficits, including limb ataxia and cognitive disturbances. Suspicion was raised for cerebrovascular ischemia and emergent referral was initiated.

Intervention and outcome: Paramedics were immediately summoned and the patient was transported to a local hospital with a working diagnosis of acute cerebrovascular ischemia. Multiplanar computed tomographic and magnetic resonance imaging with contrast revealed vertebral artery dissection of the V_2 segment in the right vertebral artery. Anticoagulation therapy was administered and the patient was discharged without complications after 5 days in the hospital.

Conclusion: This case highlights the potential for patients with vertebral artery dissection to present with nonspecific musculoskeletal complaints. Neurological symptoms may not manifest initially, but their sudden onset indicates the possibility of an ischemic cerebrovascular event. We suggest that early recognition and emergent referral for this patient avoided potential exacerbation of an evolving pre-existing condition and resulted in timely anticoagulation treatment.

Introduction

The development of vertebral artery dissection (VAD) arises randomly and unpredictably from neck

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movement and may occur prior to, during, or following spinal manipulative therapy (SMT) regardless of force load or neck position during manipulation.[1] Arterial dissection occurs when blood penetrates a tear in the intimal layer of the artery and a mural hematoma develops within the tunica media.[2] Dissection of the VA is a rare event, with an estimated incidence of 0.97 to 1.12 per 100 000 individuals per year.[3]

Stroke is the most clinically important complication of VAD and causes neurological symptoms that may establish an unequivocal diagnosis. One study reported that in a group of 108 patients under 45 years of age diagnosed with vertebrobasilar stroke, 8 (7.8%) had visited a chiropractic physician within 7 days of their diagnosis[4]; however, it was not clear if these strokes resulted from VAD or were due to other causes, such as thromboembolism from another vessel. The clinical diagnosis of a VAD is difficult when stroke or transient ischemic attack have not occurred, as it may be asymptomatic or present with only symptoms of neck pain.[5]

The most common clinical manifestations of VAD are neck pain and headache, which may or may not be followed by posterior circulation ischemia.[3,6] Neck pain and headache in such a case could easily be mistaken as musculoskeletal in origin, such as the myofascial pain syndrome.[7] It is possible that patients with VAD seek medical or chiropractic attention due to the associated neck pain and headache.[4] There is a possibility for SMT to be performed on a patient with a VAD in progress, thus potentially exacerbating the condition.[1,8]

The purpose of this case report is to describe the presentation of a patient with a VAD in progress presenting to a chiropractic physician for evaluation and treatment of a new episode of neck pain and headache.

Case Report

A 45-year-old white female, well-nourished and employed as a school administrator, presented to a chiropractic clinic complaining of upper back/neck pain and stiffness as well as headache and pain in the posterior portion of the right arm down to the elbow of 3 days duration. Her level of discomfort progressed in severity in the 24 hours prior to presentation, which is what prompted her appointment. Because this was a new complaint, an updated history and examination were performed. No history of trauma was disclosed. Physical examination revealed painful and limited active range of motion in the cervical region. Palpation was provocative for tenderness. After the initial examination, a working diagnosis of myofascial pain syndrome was established. Therapeutic ultrasound (Chattanooga Medical Supply, TN) was applied (4 minutes, 1 W/cm^2 at 1 MHz) in the seated position over the suboccipital and posterior cervical musculature. While still in the seated position, soft tissue treatment was performed by a licensed massage therapist on the suboccipital and posterior cervical musculature. The patient was shown to a treatment room and was supine when the clinician entered and asked how she felt. The patient responded that her neck pain was much better, but she was more aware of her headache. The patient was assisted to the seated posture, became dizzy, reported visual and cognitive disturbances, and had difficulty speaking. She proceeded to lose control of her right leg, which spontaneously assumed a flexion contracture. The clinician suspected a vascular etiology at this time and SMT was not performed. Paramedics were immediately summoned and the patient was transported to a local hospital with a working diagnosis of acute cerebrovascular ischemia.

Computed tomography (CT) of the head and neck (with and without contrast) and a chest film were performed. The chest exam was negative. The CT demonstrated 50% stenosis of the right vertebral artery (VA) extending from C5 cephalad to C3 without irregularity either proximal or distal to those levels (Fig 1). Also noted were subtle findings in both distal

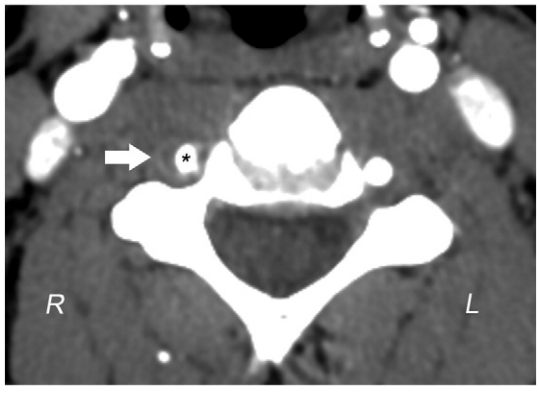

Fig 1. CTA with intravenous contrast at the level of C5 demonstrates a crescent-shaped mural thickening with annular enhancement (arrow) around a narrowed lumen of the right vertebral artery (asterisk).

extracranial internal carotid arteries attributed to underlying fibromuscular dysplasia. No intracranial abnormalities were noted on that or on any subsequent exam. Time of flight magnetic resonance angiography (TOF-MRA) images the next day also revealed 50% stenosis involving a small portion of theV_2 segment in the right VA (Fig 2). Axial images from magnetic resonance imaging (MRI) of the cervical spine on the day after admission revealed a halo surrounding the right VA with reduction of the luminal caliber (Fig 3). Three-dimensional reformat of CT angiography (CTA) demonstrated narrowing of the right V_2 segment of the VA compared to the left (Fig 4). Hospital records described transient ischemic attack, but imaging showed no evidence of stroke.

The patient was treated with anticoagulant therapy using low molecular weight heparin (enoxaparin) every 12 hours during her hospitalization. She was discharged from the hospital 5 days after presentation on oral warfarin. The patient gave written consent for educational usage of images and clinical data.

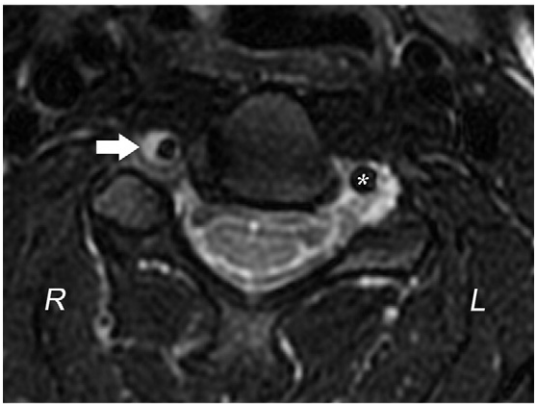

Fig 3. Axial gradient echo MRI demonstrates a narrowed lumen of the right vertebral artery compared to the left (asterisk) with a crescent-shaped mural thickening of high intensity (arrow) compared to the flow void within the lumen.

Discussion

In this case, the clinician suspected vascular compromise and avoided SMT, opting instead for

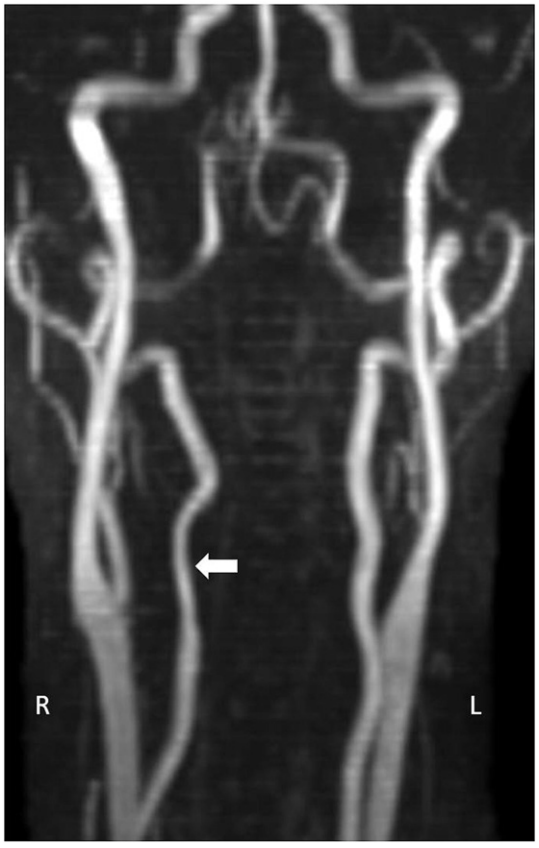

Fig 2. TOF-MRA with intravenous contrast demonstrates a narrowed V_2 segment of the right vertebral artery (arrow), with normal caliber vessel both cranial and caudal to the short stenotic region.

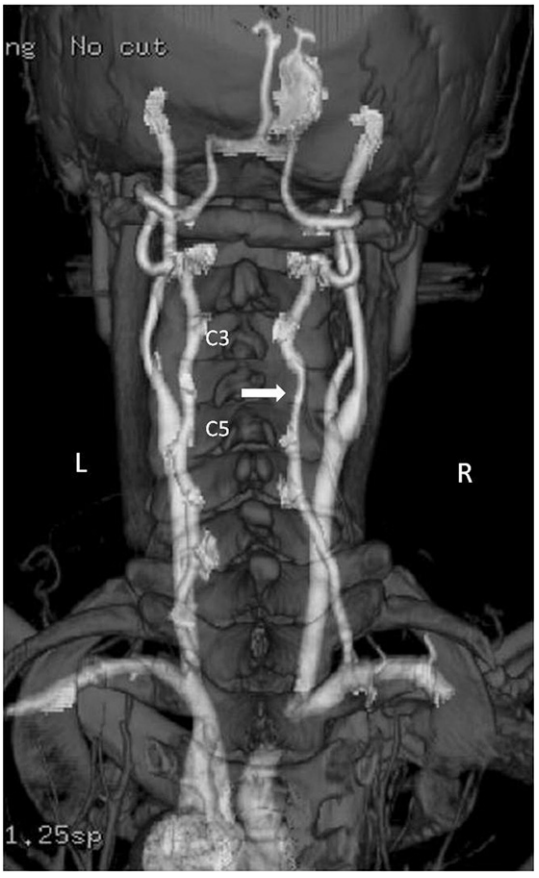

Fig 4. 3D reformat CTA viewed from posterior demonstrates narrowing of the V_2 segment of the right vertebral artery (arrow) extending from C5 to C3, with normal caliber vessel both cranially and caudally.

emergent referral for further diagnostic workup. This report highlights how important it is for clinicians to be aware of VAD and its non-specific clinical presentation in order to avoid potentially complicating the condition by applying SMT.[8,9]

Previous studies suggested an association between SMT and VAD or stroke in young adults under 45 years of age,[10,11] while others assert that the connection is likely a consequence of patients with non-specific presenting symptoms of VAD in progress seeking SMT.[1,4] Furthermore, the perceived risk of VAD post SMT may be different amongst various healthcare providers. Haldeman et al have presented data showing that for each case of VAD seen by a chiropractor approximately 3 neurologists will see that same patient. Therefore, this may falsely inflate the perception of risk for VAD post SMT amongst neurologists.[12] Our case highlights the potential for a patient with VAD in progress to present with non-specific symptoms that are seemingly musculoskeletal in origin. To our knowledge, only 1 other case has been published of undiagnosed VAD presenting to a chiropractic physician for treatment of neck pain and headache. As in our case, the clinician was suspicious of vascular etiology and did not perform SMT, instead opting for emergent referral.[13] Our case is unique in that the patient presented with non-specific musculoskeletal symptoms that quickly progressed to neurologic deficits. The onset of acute neurological symptoms and signs will alert the clinician to consider a more threatening diagnosis, however, it is important to note that diagnostic imaging showed no evidence of stoke in our patient.

The VA is described as having 4 anatomical segments (V_1-V_4). The V_1 segment arises from its origin at the subclavian artery to its entry into the transverse foramen of C6; V_2 describes its course from the transverse foramen of C6 cranially to where it exits the C1 transverse foramen to become V_3, which wraps around C1 and ascends into the foramen magnum. The intracranial portion (V_4) extends from the foramen magnum where it pierces the dura and joins the contralateral VA to form the basilar artery, which perfuses the brainstem, cerebellum, and occipital lobes.[2,14] Extracranial dissection is more common than intracranial, but the V_2 segment (dissected in this case) is the least common of the extracranial segments to dissect because it remains relatively fixed within the transverse foramina along its course.[15] The first and third segments are highly mobile and, thus, are more frequently involved in dissection.[15]

Most dissections of the VA are spontaneous, while others can be considered traumatic in etiology.[16] In the present case, the dissection was considered spontaneous. Even though a VAD may be spontaneous, there are underlying risk factors that are believed to contribute to its occurrence. Risk factors for arterial dissection include connective tissue disorders, hypertension, diabetes mellitus, smoking, hyperlipidemia, oral contraception, and others.[5,17] One study found fibromuscular dysplasia (FMD) in 15% of cases of spontaneous dissection of the carotid or vertebral arteries.[18] Our patient had imaging findings suggestive of FMD, but not associated with either of the vertebral arteries.

Dissection occurs when blood enters and accumulates within the layers of the arterial wall, causing luminal narrowing and sometimes total occlusion. Ischemia or infarction may occur distal to the territory of the occluded vasculature.[5] A complication of VAD includes ischemia in the vertebro-basilar artery (VBA) distribution, but stroke in young adults secondary to VAD is far less common than those secondary to dissection of the internal carotid artery.[2,19] A literature review that combined carotid and vertebral artery (cervical artery) dissections indicated that a minority of dissections (6%) were associated with SMT.[20] Lateral medullary infarction (Wallenberg syndrome) is a common ischemic stroke in the VBA distribution.[8] The Wallenberg syndrome includes ipsilateral Horner's syndrome (ptosis, anhydrosis, and miosis), ipsilateral facial sensory disturbance, nystagmus, ipsilateral limb ataxia, and contralateral pain and thermal sensory impairment.[6] Although our patient did not have a stroke, a population-based case-series showed that 75% of patients with a VBA stroke had at least 1 cardio- or cerebrovascular comorbidity, most of which overlapped with the risk factors for arterial dissection.[21]

In the absence of stroke, VAD may present with non-specific symptoms such as neck pain or suboccipital headache,[3,22] which likely relates to intramural nociceptors in the VA receiving efferent innervation from upper cervical nerves.[23] Presentation less commonly includes symptoms of upper limb radiculopathy, which could be explained by neural ischemia secondary to impaired perfusion by the dissection.[23] Initial presenting symptoms may be mistaken for musculoskeletal disorders.[18] Some patients with these symptoms seek chiropractic evaluation and care and thus facilitate an implied link between SMT and VAD.[4] It has been suggested that cervical SMT may exacerbate a dissection that has already occurred.[8,9] Those who were diagnosed with VAD after SMT were likely already experiencing a VAD before presentation, or

had a predisposition to dissection.[24,25] Cassidy et al concluded that there was a similar association in patients who sought clinical care for symptoms of VAD (neck pain and headache) and proceeded to have a stroke, regardless of whether management was by chiropractic or medical care.[4]

Digital subtraction angiography has been the gold standard in imaging of arterial dissection.[15] Findings include luminal stenosis or occlusion, aneurysmal dilation, intimal flap, and double lumen.[2] MRI and MRA are replacing conventional catheter angiography as they are non-invasive, lack ionizing radiation, and offer multiplanar imaging of all major arteries (and veins) of the head and neck.[26] A key MRI finding for VAD is an intramural rim of variable signal intensity (depending on the age of the blood products) surrounding a narrowed lumen.[26] Hyperintensity of the intramural hematoma on MRI represents the presence of methemoglobin, and may not be present until sufficient metabolism of the blood products has occurred, which takes approximately 48 hours.[23] TOF-MRA allows for visualization of a long segment of an artery with a short acquisition time and may demonstrate luminal narrowing.[2] CTA may demonstrate a narrowed lumen surrounded by mural thickening, which may enhance at its outermost aspect as seen in our patient.[5]

Most clinicians, whether medical or practitioners using SMT, when faced with VAD in progress aren't aware that non-specific symptoms such as neck pain and headache may be the only symptoms.[1] Examination findings may be non-contributory and the history may be more helpful, as was true in our case.[27] Neck pain and headache associated with VAD are usually described as sudden in onset, sharp, severe, and different from previous episodes of pain.[6] Since VAD may present with non-specific pain that is difficult to differentiate from musculoskeletal pain, it is important for the clinician to be wary of pain that is described as new, unexplained, or different from previous episodes, as was the case in our patient.[1,6] These common symptoms may represent VAD in progress that could be aggravated by cervical SMT.[1] Consequences such as stroke may be avoided when an early diagnosis is established.[28] Less commonly, patients with frank symptoms of stroke (visual disturbance, slurred speech) may present to a chiropractic physician for evaluation and treatment.[29]

Treatment of VAD is directed at preventing thromboembolic complications that may lead to stroke.[30] This is achieved with anticoagulant or antiplatelet medication, providing the patient has no contraindication to antithrombotic therapy.[22] Most extracranial VADs carry a good prognosis, heal spontaneously with time, and rarely require surgical intervention unless complicated by other factors, such as intracranial extension.[30]

Further research is necessary to elucidate the relationship between SMT and VAD. Diagnostic imaging provides the definitive diagnosis of VAD, but prior to imaging the clinician is left with only clinical suspicion. Given the variable clinical presentation of VAD, it would be virtually impossible to determine an association between it and SMT given its low prevalence and need for confirmation by contrast MRI, CT, or MRA. However, the assessment of every patient presenting with musculoskeletal complaints of neck pain and headache with non-invasive diagnostic imaging modalities prior to SMT is clearly not a cost-effective strategy.

Limitations

There are limitations of this case report that should be noted. Although this case of VAD is classified as spontaneous, the underlying mechanism and etiology of dissection cannot be determined with certainty. Also, it is impossible to determine the time interval related to the onset of the VAD and her presentation. She only presented when her neck pain and headache worsened. It is also not possible to distinguish her musculoskeletal symptoms from those of the VAD.

Conclusion

Our case report emphasizes that undiagnosed VAD may present with neck pain and headache, a common presentation for patients undergoing cervical SMT. Although the presentation evolved rapidly to include neurological symptoms, those symptoms were not present initially and may not be present at all in a given case of VAD. Awareness of the non-specific symptoms of VAD is important because SMT could exacerbate the condition and lead to complications such as stroke. We suggest that emergent referral for diagnostic imaging, in the setting of suspected VAD, optimizes the likelihood of an accurate diagnosis and appropriate treatment.

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No funding sources or conflicts of interest were reported for this study.

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