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Case Report

Atypical Presentation of Vertebral Artery Dissection

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Abstract

Vertebral artery dissections can cause ischemic damage to the brain. It is more common in young individuals and can be caused by minor trauma. The presentation of the disease varies and an atypical presentation can easily be missed and lead to long-term brain injury. Dissection is commonly missed because focal neurological symptoms such as Horner's syndrome are only present in 20%-58% of carotid artery dissections and occasionally in vertebral artery dissections. The diagnosis can get further clouded if the patient has a history of other neurological problems such as migraines [1]. This case study explores a case of vertebral artery dissection that presented atypically and was missed in the hospital due to absence of any significant findings on the CT imaging and other comorbidities that clouded the differential diagnosis. The goal of this study is to emphasize the importance of neurological physical exam and a thorough history taking in a young patient who presents with non-specific symptoms.

Introduction

Arterial dissection occurs in 0.3% of individuals across all ages but comprises of 20% of the ischemic strokes in young patients. Dissection occurs when the structural integrity of the arterial blood vessels allows blood to pool between two layers of the artery. Creation of the false lumen and the subsequent thromboembolism or hypo-perfusion can cause ischemic damage to the brain [1]. The most frequent cause of vertebral artery dissection is minor trauma caused by sports related injuries, roller coaster rides, vigorous exercise, childbirth, sexual intercourse, or sneezing/coughing. Connective tissue or vascular disorders can also play a role in dissection. Most individuals with vertebral artery dissection present with head or neck pain but many times can be asymptomatic [2]. Horner syndrome can occur in 25% of the patients. Other clinical manifestations include tinnitus, audible bruit, cranial neuropathies, or scalp tenderness.

It can also cause a lateral medullary infarction (Wallenberg syndrome) and also lead to subarachnoid hemorrhage. Angiography is the most sensitive mode of diagnosis but due to its invasive nature, it has been replaced by brain MRI or cranial CT with tomographic angiography [3].

Case Presentation

A 31 year old female with a 2 week history of cough and nasal congestion woke up with generalized headache, nausea, light sensitivity, and tingling in the left side of her body. Her past medical history was significant for migraines. Physical exam revealed no focal deficits and a blood pressure of 152/98 mmHg. A CT of head w/o contrast was ordered which showed no acute intracranial abnormality. Patient was given Promethazine, Ketorolac, and Morphine, and was put under observation for 2 hours. Patient fell asleep in these two hours and looked com-

fortable when sleeping but when awakened, revealed that her headache persisted. The doctors in the ED concluded that she was having a complex migraine with left sided paresthesias. She was discharged to home with Promethazine/Acetaminophen as needed for recurrent symptoms. Repeat CT the next day showed no acute intracranial abnormalities.

She followed up with her primary care doctor 3 days later. She continued to have occipital headaches and nausea. The patient reported new onset ataxia. She also reported numbness in her left cheek area, weakness of the left forearm and hands, with additional paresthesias. She also had some blurred vision and difficulty focusing on objects. Physical exam was significant for ataxia, decreased strength in left biceps/triceps, decreased lumbrical strength in left hand, and decreased grasp of left hand. The patient also appeared very anxious and was crying intermittently. The doctor then decided to set up a MRI for the same day. MRI scanning revealed acute infarctions of the left cerebellum, left posterior medulla, and small acute infarction of the right cerebellum. The MRI also revealed the distal left vertebral artery surrounded by some hyper intensity. The radiologist informed her primary care doctors, who sent her to a different hospital for further evaluation. Patient presented to the ER the next day with decreased focal symptoms. She still complained about the headache but with decreased intensity of 3/10. The pain was non-radiating and the blurry vision was now resolved. Patient still had left arm numbness but no associated weakness. The doctor concluded that her clinical symptoms were most likely secondary to left and right cerebellar and posterior medullary stroke, related to the bilateral vertebral dissection also confirmed by the MRI report. The patient was admitted to ICU for close monitoring and neurology was consulted. She was started on Plavix 300mg daily and aspirin 325 mg daily. A CT angiography was ordered to confirm the diagnosis. The CT angiography report revealed left cerebellar infarcts without associated hemorrhage. The acute right cerebellar and brainstem infarcts seen on the MRI were not revealed on the CT angiography. Diffusely irregular extra-cranial and intracranial left vertebral artery defects were seen compatible with dissection. There were also areas of severe flow limiting stenosis most significant at the levels C1 and C2. The patient was treated in the hospital and followed up 2 weeks later with her primary care doctor. She still reported some headache symptoms, difficulty balancing, and walking. Overall, she was much improved and was instructed to follow up again in 2 weeks.

Discussion

This case highlights the atypical presentation of vertebral artery dissection in a young patient. The presentation was further clouded by the fact that patient had history of migraines. The patient had a two-week history of cold and cough, which could be one of the potential causes of her condition. Minor trauma associated with coughing or sneezing can lead to ver-

tebral artery dissection. The absence of focal neurological deficits on presentation, history of migraines, and absence of any significant findings on the Head CT scan added to the atypical presentation of this patient. The focal deficits did not show up until 3 days later when she followed up with her primary care doctor who noticed left sided weakness due to a thorough physical exam. The MRI was more sensitive in revealing the culprit and CT angiography further confirmed the diagnosis. Duplex Sonography of carotid and vertebral artery can reveal VAD in 68-95% of patients but not duplex scan was ordered on initial admission [2]. Although the duplex Sonography is not the best choice to assess majority of VAD, it can be a useful non-invasive test to assess the mid-cervical region. It can also show some indirect signs of VAD such as elevated intracranial blood flow velocity. The most specific ultrasonography finding of arterial dissection is a double lumen that is separated by echogenic intima [4]. To avoid missing a vertebral artery dissection, it is important to perform a thorough physical exam to look for focal deficits. Additionally, a thorough history can reveal important clues to trigger a clinician to suspect and evaluate for VAD. The differential diagnosis for VAD is very broad as it can manifest in many different varieties. Some conditions to consider in the differential diagnosis are different types of unilateral headaches such as cluster and migraines. Trigeminal neuralgia, trigeminal cephalgia, and thunderclap headaches may also be considered. Conditions such as cardiogenic embolus, small vessel disease, atherosclerosis, and secular aneurysm rupture could be considered due to stroke type of presentation. The goal for any young patient presenting with headaches and paresthesias, particularly with any history of trauma, should be careful evaluation to rule out a cerebrovascular event.

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