

Vertebral Artery Dissection after Chiropractor Exercise: A Case Report

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Abstract

Vertebral artery dissection is an important cause of brain stem stroke, especially in the young. Dissections of carotid and vertebral arteries in neck account for about 20% of strokes in young compared with 2.5% in the elderly. One patient of vertebral artery dissection after one class of chiropractor exercise is described in this case report. She didn't have a direct severe neck trauma or concomitant risk factor like hypertension, connective tissue disease or migraine. Clinical symptomatology included occipito-nuchal pain, headache and brain stem dysfunction chiefly in the posterior inferior cerebellar artery (PICA) territory. MRA confirmed dissection with a predominant steno-occlusive picture. Cases of so called trivial neck movement/torsion related dissection have been described previously but have not received any major importance. Usually classified as 'spontaneous' or 'traumatic', there is a possible ambiguity in literature about appropriate terminology. We emphasize that a history of such subtle precipitating events be taken while diagnosing young patients with brain stem strokes, to recognize this clinical entity. Although mechanisms are not absolutely clear, yet there seems to be an important relationship between arterial dissection and neck movements or minor trauma.

INTRODUCTION

Dissection of cervical or cerebral arteries is important, but very rare cause of stroke. Recently, this entity has received increased attention, possibly due to better diagnostic aids, as well as characterization of typical symptoms and signs. Vertebral artery dissection is an important cause of brain stem ischemia in adults. Usually classified as 'spontaneous' or 'traumatic', cases related to trivial trauma have been frequently reported, although they have not received any major attention or classification. Here we describe one such case is described, where a definite temporal correlation was present between neck movement, trivial trauma and acute arterial dissection. It is important to recognize and treat this syndrome because of a relatively benign course and potentially excellent outcome in most patients.

CASE REPORT

65 year old Caucasian lady with chronic back pain from last 20 years used to do chiropractor exercises for her back pain. On the day of presentation in ER, she was performing her usual chiropractor exercise when she started feeling dizziness. This dizziness was sudden in onset and she felt like spinning of the room around her. Dizziness resolved in 15 min but it developed again after several minutes. Dizziness was associated with difficulty in eating and patient

developed the tendency to fall on left side while walking. She also developed blurriness of vision. The patient never lost her consciousness or she never developed weakness in any part of her body. She had no complaint of chest pain, palpitations and shortness of breath. There was also no complaint of nausea or vomiting. Her past medical history was significant only for chronic back pain for which she used to do chiropractor exercise from last 20 years. Her social habits were significant for social drinking and smoking. Physical examination was not significant for any finding.

Her laboratory investigations showed HB: 12.5, WBC: 8.9, platelet count: 157 BUN: 14, Creatinine: 0.96, PT: 14.7, PTT: 24.5 and INR: 1.17. Her fasting lipid profile showed cholesterol: 168, triglyceride: 63, HDL: 42, LDL: 142. Urinalysis didn't show any evidence of blood or infection. CT angiogram of the neck showed left vertebral artery dissection extending from the level of C5-6 superiorly to the intracranial portion of the left vertebral artery. US Doppler of the carotids showed no hemodynamically significant stenosis bilaterally. MR MRA of the neck showed normal flow within the left and right common and internal carotid arteries. MRI of the brain showed tiny left inferior cerebellar acute infarct while the rest of the brain appeared with in the

normal limits.

On basis of history, physical examination and investigations, diagnosis of left vertebral artery dissection was made. The patient was started on intravenous heparin to prevent thromboembolic complications. The patient's symptoms improved markedly over the course of six days in the hospital and patient was discharged on warfarin with weekly follow up at the clinic.

DISCUSSION

Arterial dissection is one of the many non-atherosclerotic vasculopathies that can cause brain ischemia. Once considered rare and recognized only post-mortem, the credit for clarifying the symptomatology and delineating the physiology of this condition goes to Fisher and Mokri¹. Vertebral artery dissection is an uncommon disorder with a variable reported frequency, and estimated annual incidence of approximately 1-1.5 cases per 10,000². However, it is an important cause of posterior circulation ischemia in young and middle aged patients and accounts for nearly one-fifth of such cases, compared to 2.5% in older patients³, although, the estimation of prevalence in elderly may be limited by co-existence of atherosclerosis as a confounding factor. Dissection of an artery occurs, when blood under pressure finds its way into the vessel wall, along a line of cleavage which is usually near the endothelial surface. It either leads to luminal narrowing and/or occlusion, if the tear is sub-intimal, or formation of a pseudoaneurysm with potential risk of bleeding if the dissection is subadventitial. Most cases of vertebral artery dissection are in the age group of 25-55 years, with a slight female preponderance⁴. Headache and neck pain are important warning symptoms of dissection with reported incidence of neck pain in half and headache in two thirds of patients. Pain often precedes neurological features by hours, days or rarely weeks^{2, 5}. It usually starts suddenly, is sharp in quality and majority of time on the side of arterial dissection. Pain is possibly caused by excitation of noci-receptors in the vessel wall as extra-cranial cerebral arteries are known to be pain sensitive. However, exact mechanisms are still elusive. Lateral medullary ischemia has been seen in most of the patients, although involvement of thalamus and cerebral/cerebellar hemisphere is seen frequently². Pathophysiological mechanisms underlying neural damage following an arterial dissection may be diverse⁶. Stenosis or occlusion of the vertebral artery results in direct ischemia of the brainstem or spinal cord. It may result from an intra-luminal thrombus or by compression of the true lumen due to blood in the vessel wall⁷. Progressive

thrombosis or artery to artery embolization can cause distal ischemia and branch artery occlusions^{1, 6, 7}. The process can obstruct distal basilar artery flow, compress cranial nerves or cause a subarachnoid hemorrhage⁶. Spinal cord infarction is possibly related to hypoperfusion and watershed infarction, embolisation or anterior spinal artery occlusion^{8, 9}. Vertebral artery dissection is routinely labeled 'spontaneous' or 'traumatic', with cases related to minor, non-penetrating neck trauma or torsion placed in between the two. This classification is possibly ambiguous. A review of the literature on arterial dissections showed, a history of abnormal neck movement, exercise or subtle trauma was present^{4, 6}. In a Canadian survey¹⁰, 81% of dissections were associated with sudden neck movement ranging from the therapeutic neck manipulation to a rigorous volleyball game, but some occurred during mild exertion such as lifting a pet dog or during a bout of coughing. Such causal potential trauma such as violent coughing, trampoline exercises, neck turning during a parade, basket ball game, dancing, swimming and minor falls have immediately anticipated the initial symptoms of dissection⁶. Names like 'bottoms up' and 'beauty parlour' stroke have also been applied to such cases. Chiropractic neck maneuvers have been strongly associated with arterial dissection, with many cases reported in literature. The distinction between spontaneous and movement related dissections seems more arbitrary and blurred when a post-resuscitation case is referred as spontaneous and those occurring during backing-up of an automobile or swinging a baseball bat, as traumatic. The possible explanation of this causation is still not well defined⁷. Different authors have suggested the association of vertebral artery anomalies, tortuosity, atherosclerosis as well as duration and force of the movement. It is also likely that inherent vessel wall abnormalities predispose to dissection upon subtle trauma. Anatomically, the vertebral artery is divided into four arbitrary parts, designated V1 to V4. V1 extends from its origin to its entry into the foramen transversarium of the cervical vertebra. The entire length within the vertebral column is labeled as V2. V3 extends between its exit from the atlas up to its entry into the foramen magnum and the entire intracranial part is termed as V4. Most of the traumatic dissections involve the atlanto occipital segment. It is likely that increased mobility, poor anchoring into the neighboring tissue and increased mechanical torsion and stretch at C1-C2 region predispose to mechanical injury^{6, 11, 12}. This also explains the increased incidence of dissection along this segment during chiropractic maneuvers. Reduction in vertebral artery blood

flow during neck movements has been shown angiographically. Hyperextension of the neck has been considered as an important precipitating factor for occlusion and dissection, especially in the presence of vascular abnormalities and bony changes. Etiology usually remains obscure in most cases of spontaneous vertebral artery dissection. However, there does exist a striking discrepancy between minor force and degree of arterial vulnerability, thereby suggesting an inherent disturbance in the vessel architecture, although the exact arteriopathy remains elusive 13. Various predisposing conditions have been cited including fibromuscular dysplasia, hypertension and migraine etc. Recent infection 14 and hyperhomocystenaemia 15 have also been cited as potential risk factors.

Diagnosis of this condition can be established by both non-invasive and invasive means. Ultrasound examination of the neck vessels can suggest the presence of dissection, although the sensitivity to detect vertebral artery disease is low when compared with the carotids. However, duplex scans have been used to monitor healing of dissections and guide long term therapy. Typical findings include increased arterial diameter, decreased pulsatility, intravascular abnormal echoes and hemodynamic evidence of decreased or reversed flow 1, 16, 17. MRA is possibly the best technique to follow up the patients and monitor healing of dissections 18. It has been suggested that, in presence of a suspicious history, symptoms and signs of dissection, typical MRI findings of dissection, unusual location for atherosclerotic involvement and absence of coexisting atherosclerotic lesion, the diagnosis of arterial dissection may not be confirmed by a conventional angiogram. Standard catheter angiography still remains the gold standard for diagnosis, as it allows excellent characterization of the dissection 1, 7. Usual features observed are irregularity and/or stenosis of the vessel, 'string sign' (arising as a result of a dissection that extends circumferentially around the lumen over a long segment), 'double lumen', pseudoaneurysm formation or complete occlusion 19. Stenosis is by far the commonest finding, being caused by a subintimal hematoma 19. In most reported studies in literature, V2-V3 segment of the vertebral artery is the commonest to be involved in dissections. Management of this condition commonly involves anticoagulation with heparin followed by oral warfarin therapy, although no general agreement exists on the best management of this condition. Anticoagulation possibly aims at preventing thromboembolic complications, commonly seen to be associated with acute dissections 1.

Some authors consider intradural extension of dissection as a contraindication for anticoagulation, because of risk of precipitating subarachnoid hemorrhage. Although, conservative management of dissection presenting as SAH has been practiced, yet rebleeding is common and potentially fatal, making surgical intervention imperative and crucial for survival 20. Endovascular treatment is usually practiced in centers equipped with intervention facilities 21. Surgical treatment of dissections, consisting of an in-situ interposition graft or extra-cranial - intracranial bypass, is indicated only for patients with persisting symptoms, refractory to maximal non-invasive management and who are not candidates for endovascular therapy. Continuation of anticoagulation therapy may be guided by MRA or ultrasound. Persisting irregularity or stenosis at 3rd month necessitates continuation of therapy for another 3 months. If features of stenosis persist beyond 6 months, it is advisable to shift the patient on antiplatelet agents 22. Overall prognosis of this clinical entity is good and encouraging, although factors like severity of ischemic insult and extent of collateral flow do influence the overall outcome 1. Nearly 75% of patients make excellent recovery and overall death rate is less than 5%. Almost 90% of the stenosis resolve, two-thirds of the occlusions are recanalised and one-third of aneurysms reduce in size 1, 19. Extracranial dissections hold a better prognosis than the intracranial ones, because risk of aneurysmal bleeds is much lower 1, 7. Maximal improvement occurs in the first 3 months following a dissection. Risk of spontaneous dissection approximates 2% in the first month and decreases to about 1% per year 23, 24. Dissection usually does not recur in the same vascular territory 24.

CONCLUSION

It is worth realizing that vertebral artery dissection is an important cause of posterior circulation stroke in young and middle aged adults. There seems to be an important association between neck movements and trivial neck torsion precipitating this condition, although controversies exist. However, it is important that patients, presenting with this clinical symptomatology are questioned in detail about precipitating factors. Whether the so called 'spontaneous' dissections are truly spontaneous or effect of forgotten neck trauma is debatable. Still, many unanswered questions remain about this clinical entity and much remains to be learned about the pathogenesis and optimal treatment of this condition.

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