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Case Report

Vertebral Artery Dissection to Perform Anticoagulation or not

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Abstract

Stroke is the leading cause of death and serious long-term disability. One person dies from stroke every 4 minutes in the United States. Ischaemic stroke and haemorrhagic stroke account for 85% and 15% of strokes, respectively. Symptoms of the two types of strokes can overlap and treatment is different. This makes early diagnosis essential so that the patient can receive treatment in a timely manner as time is brain. Haemorrhagic stroke has 4 subtypes, which include subarachnoid, subdural, epidural and parenchymal. Causes include hypertension, anticoagulation, arterio-venous malformation, cancer, metastasis, or amyloid angiopathy.

Ischaemic stroke causes include venous or arterial thrombosis. Arterial stroke accounts for the majority of ischaemic stroke, which includes thrombotic, cardioembolic, cryptogenic or genetic. Age has traditionally been used to identify stroke subtype as large vessel atherosclerosis stroke was common in the elderly who have risk factors such as hypertension, diabetes, obesity, obstructive sleep apnoea and smoking. Cryptogenic and dissection strokes are common in young patients. As the population lives longer, age becomes an unimportant factor in identification of stroke type.

Keywords: Vertebral artery dissection, Ischaemic stroke, Haemorrhage, Anticoagulation

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Introduction

A 40-year-old woman had been admitted to the medical ward because of dizziness, nausea, vomiting and severe spinning and unsteadiness. Her past medical history was notable for hypertension, type II diabetes and hypercholesterolemia **[1].**

Case Presentation

She is on hydrochlorothiazide, metformin and atorvastatin. She denied any history of recent travelling or contact with sick people. She presented to ED at 9 am (12 hours since her symptoms started). On examination she was irritable, tried not to move her head, temperature was 37.2°C, and respiratory rate was 18/minute. Her blood pressure was 130/80 and radial pulse was 70/minute with good volume and equal in both sides. Absent neck venous distension due to dehydration. Examination of the eyes showed downbeat nystagmus towards the left side. Examination of the cranial nerves showed impaired sensation of light touch in the distribution of the left trigeminal nerve in V1 and V2 distribution and left-sided Horner syndrome. She also had left-sided ataxia in the form of dysarthria and wide based gait, with tendency to lean towards the left side **[2, 3].**

Patient had loss of pain, superficial touch, and temperature in the right arm, body, and leg. Examination of strength and reflexes, including plantar, were unremarkable in both sides. Blood tests for full blood count, liver function tests, urea and electrolytes, metabolic panel, autoimmunity and vasculitis, sepsis screen, troponin, brain natriuretic peptide, D-dimer and procalcitonin were either normal or negative.

Results

CT of the head, chest, abdomen and pelvis did not show any abnormalities and transthoracic echo ruled out vegetation, clots and regional wall abnormalities with normal systolic function of the left and right ventricles. Repeated transthoracic echo with agitated saline ruled out patent foramen oval or left to right shunt. MRI stroke protocol and MRA confirmed dissection in the left vertebral artery and left sided lateral medulla and cerebellar infarction. The patient was diagnosed with incomplete lateral medullary syndrome due to left vertebral artery dissection. She was treated with fluids, loading dose of aspirin and clopidogrel, metoclopramide, ondansetron and atorvastatin.

The patient was assessed by a speech therapist who advised thickened fluids due to high risk of aspiration and the physiotherapy team was involved from early treatment. The patient was seen by neurology who advised against anticoagulation and to continue dual antiplatelet for 6 months and repeat MRI and MRA in three-and-6-months' time. The patient improved and was able to return home after two months. Her repeat MRAs at 3 and 6 months did not change or show signs of recanalisation.

Discussion

Vertebral artery dissection is not uncommon in the young population. It is classified as traumatic or spontaneous **[4]**. Patients with spontaneous dissection should be investigated for underlying collagen disease, fibromuscular dysplasia, Marfan syndrome, Ehlers-Danlos Syndrome type IV, osteogenesis imperfecta, polycystic kidney disease, pseudoxanthoma elastica and hereditary haemochromatosis.

The vertebral artery is referred to as the cervical artery in literature as it arises from the subclavian artery and ascends into the cervical foramina transversarium at the level of C6, making segment V1. Segment V2 ascends to C2, segment V3 extends from the transverse foramen of C2 to the posterior arch of the atlas after piercing the dura and segment 4 is intracranial, starting at the foramen magnum basilar, and combines with the other vertebral artery to form the vertebrobasilar artery.

The V4 segment is intracranial with a thin adventitia and lacks supporting tissue such as external elastic tissue, hence dissection in segment 4 causes subarachnoid haemorrhage. Dissection in the vertebral artery is common in segments 2 and 4 due to the pars transversaria and atlas loop, respectively **[5]**. Symptoms of vertebral artery dissection are very non-specific, such as dizziness, vertigo, diplopia, dysarthria, neck pain, and ataxia; therefore, diagnosis of vertebral artery dissection is challenging **[6]**. Early diagnosis and treatment of vertebral artery dissection could reduce the risk of vertebrobasilar stroke and serious sequela like locked in syndrome or death **[7]**.

Evidence of intravenous thrombolysis (IVT) in patients with ischaemic stroke with either alteplase or Tenecteplase is highly efficacious, however evidence of thrombolysis in patients with ischaemic stroke due to cervical or vertebral artery dissection remains very limited and mostly includes observation studies. However, in all studies with thrombolysis of ischaemic stroke, cervical and vertebral artery dissection were not excluded **[8]**. In all studies, the rate of intracranial haemorrhage was similar in patients with and without vertebral artery dissection **[9]**.

In the absence of safety concerns and given the efficacy of IVT in eligible patients with ischaemic stroke, it makes sense not to withhold thrombolysis in patients with cervical artery dissection if they meet all the criteria for thrombolysis **[10]**. In patients with segment 4 vertebral artery dissection, the risk of subarachnoid haemorrhage is high, and the safety and benefit are not well established for thrombolysis **[11]**.

Most ischaemic strokes due to vertebral artery dissection are caused by artery-to-artery embolisation, hence administration of anticoagulation is commonly used to prevent artery-to-artery embolisation and arrest the progression of ischaemic stroke. Two randomised trials, CADISS (Cervical Artery Dissection in Stroke Study) and TREAT-CAD (Biomarkers and Antithrombotic treatment in cervical artery dissection) examined anticoagulation versus antiplatelet treatment.

CADISS was a UK-based multicenter open-label, randomised controlled trial and had 250 patients with cervical artery dissection. Patients were randomly allocated to be treated with an antiplatelet or a vitamin K antagonist with bridging-heparin in 124 patients including 12 without bridging and antiplatelets in 126 patients. TREAT-CAD was a multicenter open-label, randomised controlled, non-inferiority trial

comparing the efficacy of aspirin and vitamin K antagonists.

The two trials concluded that if patients have a high risk of bleeding based on radiological criteria, they should not be treated with anticoagulation or antiplatelets. Radiological criteria included dissection of intracranial segment 4 vertebral artery, ruptured vessel wall, intramural haematoma, intimal flap, double lumen, pearl and string sign (focal aneurysmal dilation, pearl) adjacent to long irregular stenosis (string), suboccipital rind sign (hyperintense on T1 or thickened on CT rim around the vessel), representing hematoma and development of aneurysm.

Both trials concluded that 3-to-6-month duration of antithrombotic is reasonable and longer antithrombotic may be reasonable taking into consideration bleeding risk and neuroimaging signs **[12]**.

Conclusion

Clinical diagnosis of vertebral artery dissection is very challenging and it is not uncommon that neuroradiology diagnoses dissection before the clinician. Radiologic red flags are intimal flap, double lumen, dissecting aneurysm, intramural haematoma, stenosis and complete occlusion. Dissection has predilection to segments 2 and 3 of the vertebral arteries.

There are no clear guidelines for the treatment of cervical artery dissection. Vertebral artery dissection is common in the young person who may be asymptomatic with no restriction defect in DWI (diffuse-weighted imaging) or presenting with cerebral or brain stem stroke, complete or incomplete lateral medullary infarction. In contrast, internal carotid artery dissection usually presents with ipsilateral Horner syndrome and anterior stroke features.

Patients presenting before 430 hours are suitable recipients of intravenous thrombolysis unless there is dissection of segment 4, due to increased risk of subarachnoid haemorrhage. Patients with high bleeding risk will be treated with single or dual antiplatelet. In the absence of clear guidelines, the clinician is left to provide the best care based on their experience, taking into consideration the radiological findings and bleeding risk.

Additionally, secondary causes need to be ruled out including collagen disease, patent foramen ovale, fibromuscular dysplasia, Marfan syndrome, homocystinuria, and genetic stroke, such as CADASIL (autosomal dominant arteriopathy, subcortical infarct and leukoencephalopathy) due to mutation in NOTCH3.

Conflict of interest

All authors report no conflict of interest.

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