Vertebral artery dissection: a treatable cause of ischaemic stroke

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Vertebral artery dissection used to be an uncommon diagnosis, but it is now being diagnosed more frequently owing to the use of magnetic resonance imaging. We report on three patients with vertebral artery dissection to illustrate the importance of establishing this diagnosis by using magnetic resonance imaging in patients who present with cerebrovascular accident that involves the posterior territory. Treatment with heparin can help prevent recurrent embolic events and should be given in the absence of subarachnoid haemorrhage or other contra-indications.

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Introduction

Cerebrovascular accident is an important cause of morbidity and mortality. Most cases occur in elderly patients as a result of atherosclerosis. While vertebral artery dissection accounts for only 0.4% to 2.5% of the overall cases of cerebrovascular accident,[1] it accounts for 4% of cases of ischaemic stroke in patients younger than 45 years and 14% of cases of lower brainstem infarction.[1] Using magnetic resonance imaging (MRI), vertebral artery dissection can be diagnosed non-invasively. Three cases of spontaneous vertebral artery dissection have been diagnosed in the first year since the introduction of an MRI service at the Tuen Mun Hospital (TMH) in 1997. The following case reports illustrate the typical presentation and outcome of patients with vertebral artery dissection.

Case reports

Case 1

A 16-year-old boy was admitted to the TMH in April 1997 because of the insidious onset of right-sided clumsiness, vertigo, mild headache, and vomiting during the previous 2 weeks. He did not have paraesthesia or weakness, but had experienced a trivial neck injury a few days before the onset of his symptoms. Physical examination revealed right cerebellar signs. There were no other neurological signs and the cranial nerves were all intact. A computed tomography (CT) scan of the brain showed a hypodense lesion in the right cerebellar hemisphere. The T2-weighted images from a magnetic resonance imaging study showed a hyperintense signal in the right side of the cerebellum and the left side of the thalamus; these results were compatible with a diagnosis of infarction. Magnetic resonance angiography showed right vertebral artery dissection and thrombosis. The left thalamic infarction was probably due to embolisation across a perforator branch. Heparin 1000 U/h was given intravenously and followed by warfarin 2 mg/d. The patient's symptoms subsided 3 days later and no further cerebellar signs were detected.

Case 2

A 56-year-old man was admitted to the TMH in July 1997 because of a sudden onset of dizziness, headache, double vision, and unsteady gait. He had a 2-year history of hypertension and had been taking metoprolol and nifedipine. He denied having any history of head injury or neck trauma. On admission to hospital, the patient was fully conscious and afebrile, and the blood pressure was normal. There was nystagmus on left lateral gaze; loss of right lateral, upward, and downward gazes; left eye ptosis and a dilated left pupil; pin-point right pupil; and truncal ataxia. Long tract signs were absent. A CT scan of the brain revealed evidence of cerebellar and basal

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ganglia infarcts. Subarachnoid haemorrhage (SAH) was not detected following CT or lumbar puncture. The T2-weighted images from an MRI study showed a multifocal hyperintense signal in the left side of the cerebellum, pons, and left thalamus. T1-weighted images showed a hyperintense lesion and the narrowing of the lumen of the V4 segment of the left vertebral artery. There was evidence of left intracranial vertebral artery dissection that extended into the proximal basilar artery. The patient's condition deteriorated and there were signs of brainstem death; he died 1 week after hospital admission.

Case 3

A 44-year-old man presented to the TMH in September 1997 after having had a trivial neck injury on the previous day. He had had a right-sided headache and neck pain several hours after the injury, and had experienced dysphagia and numbness over the right side of his face and left arm 1 day after the injury. Physical examination showed right palatal paralysis, a loss of the gag reflex, a deviation of the uvula to left side, and an impaired touch sensation over the right side of the face and left arm. A CT scan of the brain showed no obvious abnormalities. Following a lumbar puncture, the pressure of the cerebrospinal fluid was found to be 24 cm H_2O and the fluid was found to be blood-stained. An MRI study revealed a focal T2 hyperintensity in the right-lateral medulla, which was consistent with a diagnosis of infarction. In addition, a high-intensity lesion and narrowing of the lumen in the V4 segment of the right vertebral artery were detected on the T1-weighted image (Fig 1). Magnetic resonance angiography confirmed the right vertebral artery dissection and the presence of a subacute intraluminal haematoma (Fig 2). Heparin was not given because of the SAH. The patient experienced progressive neurological deficit. Progressive dissection was suspected and the right vertebral artery was clipped by a neurosurgeon. The patient made an uneventful recovery but experienced residual numbness on the right side of the face.

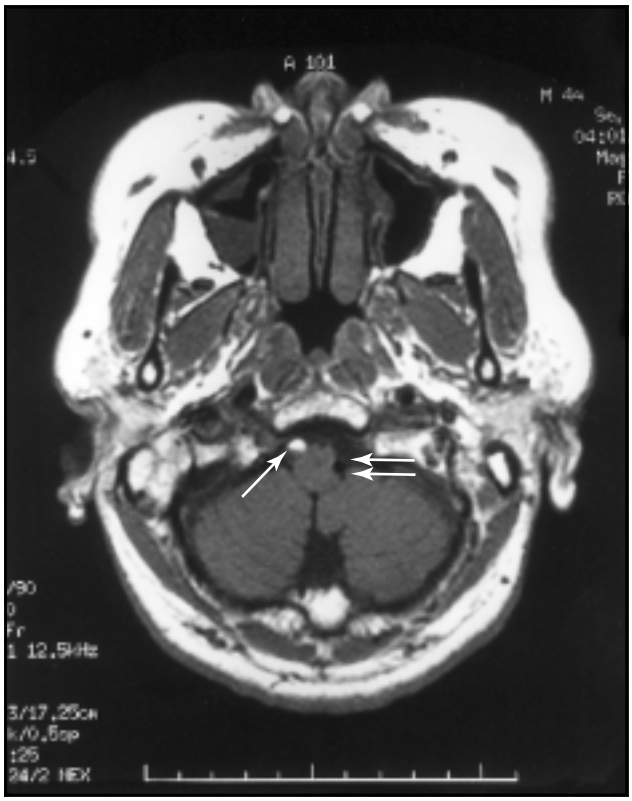

Fig 1. Axial T1-weighted magnetic resonance image showing a high-signal intramural haematoma and resultant narrowing of the right vertebral artery (arrow)
Note the lack of signal in the normal left vertebral artery (double arrow)

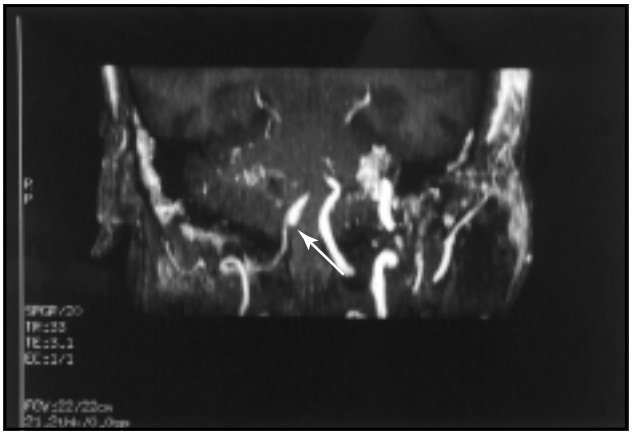

Fig 2. Three-dimensional magnetic resonance angiogram showing abrupt luminal stenosis of the right vertebral artery and subacute intramural haematoma (arrow)

Discussion

The two vertebral arteries, together with the basilar artery, are the main blood supply to the posterior circulation of the brain. Depending on the site of involvement, vertebral artery dissection is classified as V1 to V4, or extracranial or intracranial.[2] Extracranial dissection includes V1 (proximal to entry into the transverse foramen), V2 (within the transverse foramen from C6 to C2), and V3 (after exit from the C2 transverse foramen). Intracranial or V4 vertebral artery dissection refers to dissection that occurs after the vertebral artery has entered the dura.[2]

The spontaneous dissection of the vertebral artery was once considered to be a rare event, but it is now increasingly being recognised as a cause of stroke. Recent data have shown that 40% of posterior fossa infarctions are caused by vertebral artery dissection.[3] Common predisposing factors of vertebral artery

dissection include hypertension and fibromuscular dysplasia. Other risk factors are cystic medial necrosis, Ehlers-Danlos syndrome, Marfan's syndrome, and rheumatoid arthritis.[4]

The diagnosis of vertebral artery dissection requires a high index of suspicion when there are suggestive presentations, especially in young adults. Occipital headache and neck pain are common features, and occur in 70% of patients with vertebral artery dissection; 60% of patients have symptoms of vertebrobasilar circulation ischaemia.[4,5] There might be a history of neck trauma or manipulation. Extracranial vertebral artery dissection can be spontaneous or traumatic, whereas intracranial vertebral artery dissection is mostly spontaneous and presents with posterior circulation infarctions or SAH.[6]

In the past, angiography was needed to diagnose vertebral artery dissection. Typical angiographic findings consist of luminal narrowing or occlusion, the appearance of a double lumen, or the formation of a pseudoaneurysm.[2] Computed tomography can demonstrate the consequences of arterial dissection such as infarctions and subarachnoid haemorrhage. However, evaluating the condition of the posterior fossa is difficult owing to beam-hardening artefacts. Although using CT angiography can minimise these drawbacks, CT angiography requires the administration of intravenous contrast medium.

In contrast, the use of MRI can enable a diagnosis to be made non-invasively. The MRI findings of vertebral artery dissection include an intramural haematoma, an intimal flap, and the enhancement of the artery wall and septum.[6] Magnetic resonance angiography can clearly show abrupt luminal stenosis and the disappearance of flow signal at and distal to the dissection.[6] Magnetic resonance angiography can also enable the age of the haematoma to be estimated. An intramural haematoma has an intermediate signal on T1-weighted images and a high signal on T2-weighted images in the first few days of dissection. Subsequently, there is a high signal on both T1- and T2-weighted images and finally, the hyperintensity resolves.[7]

Multivessel dissection occurs in two thirds of cases[2,6] and should be investigated carefully. Duplex colour-flow imaging can be used to diagnose extracranial vertebral artery dissections non-invasively. Ultrasonographic findings include stenosis, intimal flap with true and false lumens, pseudoaneurysm, intramural haematoma, and occlusion.[8] The V2 and V3 segments, however, may not always be well visualised. Errors may also occur in the presence of a hypoplastic vertebral artery. Furthermore, imaging the vertebral artery requires an experienced sonographer.

Based on MRI findings, vertebral artery dissection can be classified into three groups. The first group is symptomatic cases with evidence of cerebrovascular ischaemia. The second group is symptomatic cases (eg neck pain) but without symptoms or imaging evidence of cerebrovascular ischaemia. The third group belongs to incidental findings from MRI of the brain or neck for unrelated symptoms. Treatment is indicated in the first group, as was the case for all three patients in this report. However, it is controversial as to whether treatment should be given in the second and third groups of disease. In general, the decision to start treatment depends on the presence of a recent clot and how recently the symptoms started.

Aspirin is currently the standard treatment for acute ischaemic stroke. Subcutaneous unfractionated heparin is not associated with a reduction in the number of early deaths or the incidence of recurrent stroke.[9,10] In contrast, the treatment of spontaneous vertebral dissection remains controversial. A wide variety of treatment regimens have been advocated for extracranial dissection, including conservative therapy, antiplatelet regimens, anticoagulation therapy, and surgical resection of the involved artery.[11] Anticoagulation is usually advised for patients with extracranial vertebral artery dissection in the absence of SAH, because of the risk of recurrent ischaemic events. As in case 1 of this report, heparin is given intravenously at a standard dose of 1000 U/h and titrated according to the activated partial thromboplastin time (APTT). Heparin treatment is then changed to warfarin therapy 5 days later. Anticoagulation should be continued for 6 months, when the majority of the dissections have recanalised. Accordingly, low molecular weight heparin, by virtue of its biological properties, can be given subcutaneously without the need of APTT monitoring. However, clinical data of its efficacy in the treatment of vertebral artery dissection is lacking. The role of anticoagulation in intracranial vertebral artery dissection is not well defined and surgical intervention is usually advocated. Some patients may experience repeated embolic events despite receiving anticoagulation therapy.

Endovascular treatment has been used successfully to prevent the formation of further emboli. Occlusion of the parent artery can be achieved by inserting a balloon or coil proximal to the dissection site. Surgical intervention of intracranial vertebral artery dissection includes proximal clip-occlusion of the affected

artery when the opposite vertebral artery is equal in size or larger. The dissection is wrapped if the affected artery is dominant.[10]

The prognosis of vertebral artery dissection is generally benign.[4,5,12] Ischaemic events commonly occur within the first week of the dissection and sometimes up to 1 month later. The dissected artery recanalises within the next few weeks or months.[12] However, extension into the basilar artery and SAH are associated with a poorer prognosis and increased mortality. The risk of recurrence of dissection is 1% per year.[4]

In conclusion, the use of MRI can help in the diagnosis of vertebral artery dissection, which should be considered as a diagnosis for patients with infarctions in the posterior territory. The administration of anticoagulants decreases the risk of recurrent embolic event and improves the prognosis of some patients.

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