Spontaneous basilar artery dissection

Dissection of the basilar artery is rare. We report a 51-year-old man who developed acute pontine infarction associated with dissection of the distal basilar artery. There was no trauma or unaccustomed movement of the head and neck prior to the stroke. The dissection was diagnosed non-invasively by magnetic resonance imaging and magnetic resonance angiography. Cervicocerebral artery dissection is a common cause of stroke in young patients particularly when conventional cardiovascular risk factors are absent. Magnetic resonance angiography combined with magnetic resonance imaging is a useful diagnostic tool prior to invasive angiography.

Introduction

Cervicocerebral artery dissection is an uncommon aetiology in ischaemic stroke and accounts for approximately 2% of all cases. However, in patients younger than 45 years, arterial dissection is the second leading cause, accounting for 10 to 25% of ischaemic strokes in this age-group. Previous reports of cervicocerebral arterial dissection predominantly involved the extracranial artery, with the carotid and vertebral arteries constituting the majority of cases (80-90%). With advances in magnetic resonance imaging (MRI), dissections of intracranial arteries are increasingly being recognised. The suprachiasmatic segment of the internal carotid artery and the middle cerebral artery stem are sites with a predilection for intracranial arterial dissection. Isolated dissection of the basilar artery is rare. We report a patient with spontaneous dissection of the basilar artery resulting in pontine infarction and present the clinical and radiological features.

Case report

In May 2006, a 51-year-old Chinese man developed acute and progressive left hemiparesis and dysarthria in the early morning. He reported suffering a dull occipital headache a day before. There was no history of trauma, unaccustomed extension, rotation or chiropractic manipulation of the neck. He had obstructive sleep apnoea syndrome (OSAS) for more than 10 years and had been on nocturnal continuous positive airway pressure. He had also been in advanced renal failure from chronic glomerulonephritis, which had been successfully treated with a cadaveric renal transplant 2 months before admission. Common cardiovascular risk factors, like hypertension, diabetes mellitus, and hyperlipidaemia, were absent. His usual medications were prednisolone and azathioprine. On admission, he was afebrile, with a blood pressure of 140/80 mm Hg, and a pulse rate of 120/min. He was orientated but drowsy. Dysarthria, a dense left hemiparesis and an impaired lateral gaze were evident. The plantar response was normal on the right and up-going on the left. Pupillary light reflexes were normal bilaterally. His neck was soft and without local tenderness. The heart sounds were normal and the electrocardiogram showed a sinus tachycardia. The blood results included a haemoglobin of 83 g/L, a haematocrit of 41%, a leukocyte count of 13.2 x 10⁹/L, a platelet count of 310 x 10⁹/L, a serum fibrinogen level of 4.00 g/L, an erythrocyte sedimentation rate of 5 mm/hr, a fasting plasma glucose of 10.5 mmol/L, and a glycosylated haemoglobin of 6.5%. The coagulation profile, fasting cholesterol, liver and renal function tests were unremarkable. There were no autoimmune markers. Non-contrast cranial computed tomography revealed no acute intracerebral haemorrhage. Transthoracic echocardiography was normal. Carotid duplex ultrasound showed normal extracranial carotid arteries, and the flow in both vertebral arteries was antegrade and normal. Diffusion-weighted MRI showed restricted diffusion at the right pons, suggestive of acute pontine infarction. The contrast-enhanced magnetic resonance angiography (MRA) showed smooth tapering of the distal basilar artery (Fig 1). The dissection was confined to the basilar artery; both vertebral arteries were unremarkable. The diagnosis was spontaneous basilar artery dissection resulting in acute pontine infarction. Anti-coagulant therapy (subcutaneous low-molecular-weight heparin, followed by warfarin aiming to achieve
the international normalisation ratio of 2.0-2.5) was then initiated. The blood pressure was carefully monitored. The patient remained stable without further neurological deterioration.

Discussion

Isolated basilar artery dissection is uncommon. In the majority of patients with posterior circulation dissections, the basilar artery is usually involved by extension of distal vertebral artery dissection.6

Previously, a definitive diagnosis of vertebrobasilar artery dissection could only be based on invasive angiography. With advances in radiology, MRI combined with MRA has now become the investigation of choice because of their non-invasiveness and comparable sensitivity and specificity. Nonetheless, in difficult cases conventional digital subtraction angiography remains the gold standard for defining the exact level and arterial territory of the dissection.7-9 As shown in Figure 1, an intimal flap with the double-lumen sign, a crescent-shaped intramural thrombus, and an eccentric flow void of the patent lumen can be clearly demonstrated in the axial T2-weighted MRI, and these features are highly specific for dissection. The acute thrombus in the false lumen may appear more hyperintense, and thus more conspicuous in T1-weighted images. However, the concurrent hyperintense signal from the sluggish flow in the compromised true lumen may actually obscure the intimal flap, rendering the ‘double-lumen’ indistinct. This interference is an important consideration in a small calibre artery. Therefore, dissection of an intracranial artery can be more readily appreciated on T2-weighted images.

Clinically, most people with symptomatic basilar artery dissections present with brain stem or cerebellar ischaemic strokes, or less commonly, subarachnoid haemorrhages.10,11 The mean age of presentation is in the late 40s.12 In contrast to extracranial carotid or vertebral artery dissections, basilar artery dissection affects more men than women.12,13 One characteristic of the stroke related to basilar artery dissection is a close temporal relationship to unaccustomed neck movement, cervical spine manipulation or trauma,14 but this was absent in our patient. It has been postulated that underlying arteriopathies, such as fibromuscular dysplasia, connective tissue disorders (eg Ehlers-Danlos syndrome and Marfan syndrome), cystic medial necrosis and moyamoya disease, cause structural weakening of the arterial wall, predisposing to arterial dissection.15
In our patient, there was no evidence of an underlying arteriopathy. Other risk factors for dissection such as family history or recent infection were also absent. He had a long history of OSAS and although no literature has so far reported any association between cervicocerebral artery dissections and OSAS, Sampol et al. found that patients with aortic dissection had a higher apnoea-hypopnoea index than a control group with hypertension. This suggests that OSAS may be an additional risk for aortic dissection.

In conclusion, cervicocerebral artery dissection should be considered as a possible aetiology in young patients with stroke, particularly when conventional cardiovascular risk factors are absent. In combination, MRI and MRA form a useful non-invasive diagnostic tool for suspected basilar artery dissection.

References