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CASE REPORT

Vertebral Artery Dissection in a 5-year-old Child: Serial Magnetic Resonance Angiography Appearance

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ABSTRACT

This report is of a 5-year-old boy presenting with complex partial seizures. These were subsequently found to be due to vertebral artery dissection by magnetic resonance imaging and contrast-enhanced magnetic resonance angiography. Serial changes of contrast-enhanced magnetic resonance angiography appearance of vertebral artery dissection are presented. The imaging findings of this patients are quite unique in that they show that recanalisation of the dissected vessel may occur with occlusion of the previously unaffected segments. Risk factors, clinical features, and imaging tools useful for establishing the diagnosis are reviewed. This patient demonstrates the importance of considering vertebral artery dissection for paediatric patients with posterior fossa infarction and suggest that magnetic resonance imaging and contrast-enhanced magnetic resonance angiography of the neck is useful for establishing the diagnosis and management of these patients.

Key Words: *Magnetic resonance angiography, Magnetic resonance imaging, Pediatrics, Vertebral artery dissection*

INTRODUCTION

Posterior circulation infarction is uncommon in children. Vertebral artery dissection remains a rare diagnosis. Headache and seizure are the 2 commonest presenting symptoms.[1] Anticoagulation treatment for these patients remains controversial. The majority of patients have a favourable clinical outcome.[1] magnetic resonance imaging (MRI) and contrast-enhanced magnetic resonance angiography (CE MRA) are considered the modalities of choice for initial diagnosis and follow-up. Conventional angiography remains the gold standard but is reserved for selected patients due to its invasiveness.

CASE REPORT

A 5-year-old boy had an episode of complex partial seizure followed by lethargy and dizziness. At presentation, the seizure was described as a loss of responsiveness with an expression of a frightened stare and shoulder shrugging. The seizure lasted for 2 hours. The boy's unsteadiness when walking persisted for approximately 1 day. A significant improvement was seen 24 hours after the seizure. There was no cerebellar ataxia at follow-up. The patient had no history of trauma or systemic illness. He was born by normal vaginal delivery. There was no family history of migraine or cerebrovascular disease. Blood tests and urine tests at the time of acute illness showed no significant abnormality. Cervical X-ray was normal. Initial MRI of the brain showed infarction in the pons and left cerebellum (Figures 1 and 2). No MRA was performed at that time.

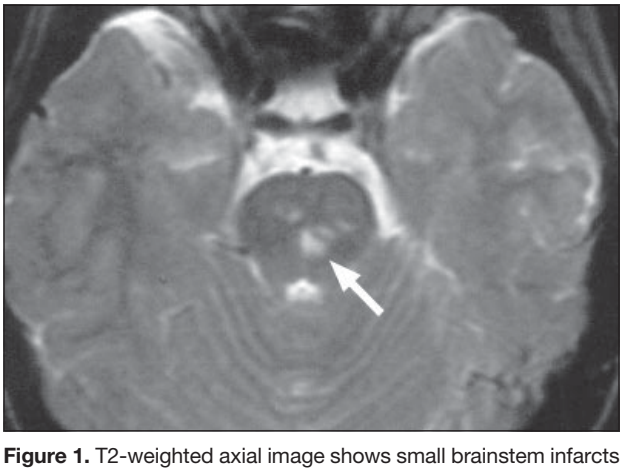

Figure 1. T2-weighted axial image shows small brainstem infarcts (arrow).

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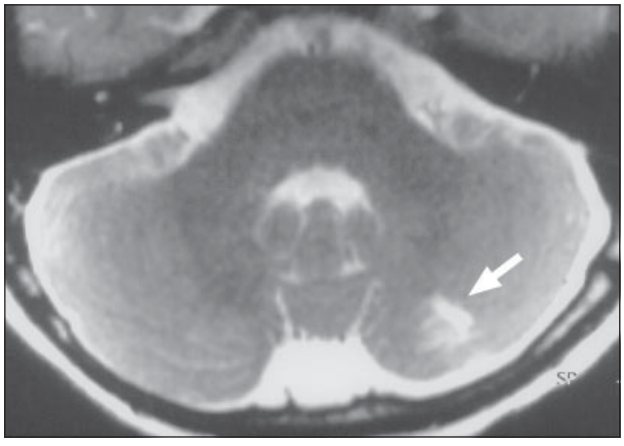

Figure 2. T2-weighted axial image shows infarct of left cerebellum (arrow).

He attended the neurological clinic of the Queen Elizabeth Hospital 2 months after the initial attack. No neurological abnormality was detected at physical examination. His heart sounds were normal with no murmur detected. His blood pressure was normal. After the acute illness, the patient did not experience another seizure or acute neurological disturbance. He did not receive anticoagulation therapy or antiepileptic treatment after the attack.

In view of possibility of vertebral artery dissection, CE MRA of the neck (Figure 3) and time of flight MRA of the circle of Willis (Figure 4) were performed.

Examinations were performed on a 1.0 T imaging system (Magnetom Expert Plus; Siemens, Erlangen, Germany) with a 20 mT/m maximum gradient strength. Circular polarised (CP) head and neck coils were used. A transverse 3D time of flight sequence was used to

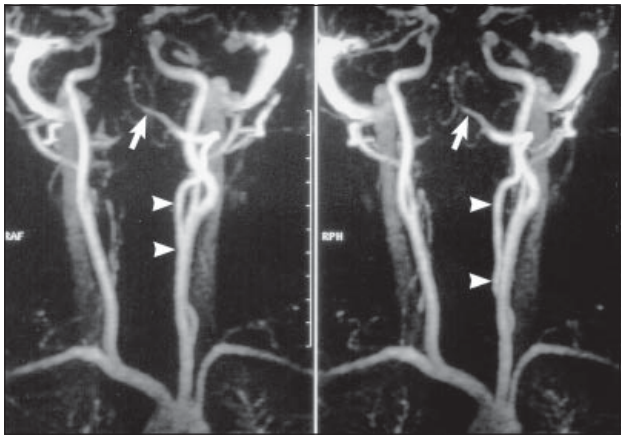

Figure 3. Contrast-enhanced magnetic resonance angiography shows abrupt tapering suggestive of dissection of the V3 segment of left vertebral artery (arrow). The V1 and V2 segments were normal (arrowheads).

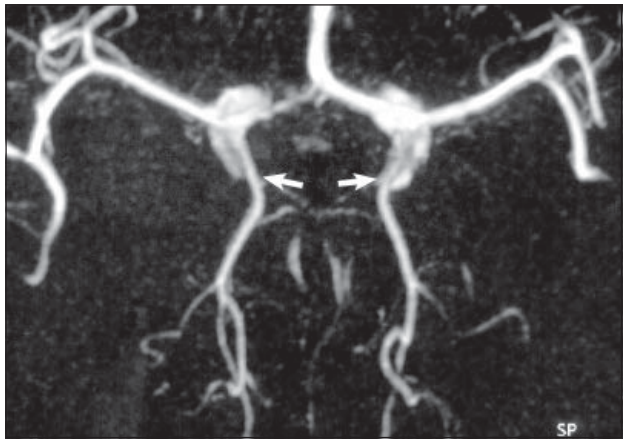

Figure 4. The intracranial portion of both vertebral arteries and basiliar artery were not seen on time of flight magnetic resonance angiography. The posterior cerebral arteries of both sides were supplied by posterior communicating arteries (arrows).

delineate the circle of Willis. The parameters were as follows: fisp sequence, TR/TE 37/9.6 ms; scan time: 3 minutes 11 seconds, flip angle 20°, slab thickness 48 mm, partition: 64 (interpolated), effective thickness 0.75 mm, rectangular FOV 20 cm, 6/8, matrix 200 x 512 (pixel: 0.75 x 0.39 mm). Contrast 3D MRA of the neck was performed in the coronal plane using a fast imaging sequence (Turbo Flash), with the following parameters: TR/TE 5.4/2.2 ms, flip angle 35°, scan time 10 second, slab thickness 72 mm, partition 48 (interpolated), effective thickness 1.5 mm, field of view 27 cm, 5/8, matrix 100 x 256 (pixel: 1.69 x 1.05 mm).

A bolus of 10 ml of gadolinium chelate (Gadodiamide Omniscan; Nycomed, Carrigtohill, Ireland) was given via injection. Four consecutive measurements were obtained 10 seconds after contrast injection started. The base images were then transferred to a workstation. MRAs were generated in lateral rotations by using a maximum intensity projection (MIP) algorithm and projections every 10° from -60° to +60° with 0° corresponding to the frontal plane.

CE MRA showed abrupt tapering suggestive of dissection of the V3 segment of the left vertebral artery (Figure 3). The V1 and V2 segments were of normal calibre. The basilar artery also appeared small. The right vertebral artery was small and appeared irregular. Other great vessels from the aortic arch were normal.

The intracranial portion of both vertebral arteries and the basilar artery were not seen in TOF MRA (Figure 4). The posterior cerebral arteries of both sides were supplied by posterior communicating arteries.

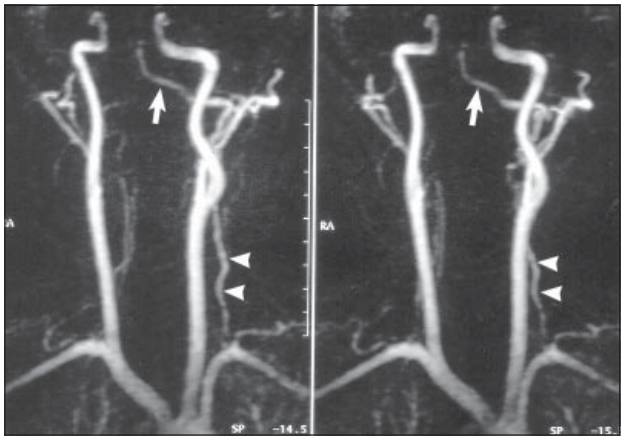

Figure 5. The V1 and V2 segments of the left vertebral artery could not be identified. The V3 and V4 segments of the left vertebral artery (arrow) were reconstituted by tortuous enlarged collaterals (arrowheads) arising from the left subclavian artery.

The patient was followed up approximately 18 months later. He was asymptomatic with no neurological deficit. This time, MRI showed no new infarcts. CE MRA performed with the same parameters showed no significant interval change in the appearance of the right vertebral artery. The previously normal looking V1 and V2 segments of the left vertebral artery could not be identified while improvements of the V3 and V4 segments were seen. They appeared to be reconstituted by tortuous enlarged collaterals arising from the left subclavian artery (Figure 5). These findings were verified by reviewing the base and maximum intensity projection images of the MRA.

DISCUSSION

Posterior circulation infarction is uncommon in children. Vertebral artery dissection is considered rare, especially in the absence of direct trauma to the neck.[2] Risk factors associated with vertebrobasilar ischaemia include systemic hypertension, underlying arteriopathy, sepsis, anomalies of the cervical spine or posterior circulation, the thermolabile methylene tetrahydrofolate reductase gene mutation, or factor V Leiden mutation.[1,3,4] Underlying cardiac abnormalities are rare.

Headache is the most common presenting symptom.[1] This patient presented with seizure, which is also a common presenting symptom.[1] Other reported clinical presentations include neck pain, serial transient ischaemic attacks, clinical stroke, and a mixture of brainstem and cerebellar signs.[1] The majority of these patients are boys with multi-focal vascular abnormalities. Clinical outcomes for the majority of patients are good. More than half of the patients in Ganesan et al's series showed no neurological deficit.[1] This patient was clinically asymptomatic largely because of the good supply of posterior cerebral arteries from the posterior communicating arteries. The most common advocated treatment for patients with proven dissection is anticoagulation. However, the risk and benefits of this remains controversial.

Ganesan et al predefined the radiological criteria for making the diagnosis of arterial dissection as follows:[1]
- dissection flap on catheter angiography (CA)
- tapering arterial occlusion ('string sign') on CA
- aneurysmal dilatation on CA (dissecting aneurysm)
- intramural haematoma on MRI.

These strict criteria tend to underestimate the actual incidence of dissection. This patient demonstrated the string sign Ganesan et al defined for catheter angiography. Because MRI and CE MRA are widely employed as the initial imaging investigations, these criteria may need further modification.

MRI and MRA are considered modalities of choice for initial evaluation of vertebral artery disease in most centres.[2] They are non-invasive and can simultaneously evaluate all major cervical and intracranial arteries as well as the brain. Typically, MRI reveals a narrowed flow void surrounded by a crescentic or circumferential intramural or periarterial haematoma. The haematoma is usually subacute and appears hyperintense on both T1- and T2-weighted images. Haematoma signal intensity may decrease as it resolves and appear intermediate in signal intensity making it less conspicuous. Fat-suppressed images may be useful in increasing the conspicuity of the haematoma relative to the hyperintense signal of adjacent fat. Enhancement of the haematoma may be seen. Gradient echo images may be useful for demonstrating the intimal flap as a hypointense band separating the haematoma and the intraluminal flowing blood.

MRI has some limitations in the diagnosis of cervicocephalic artery dissection, including false-positive results related to technical causes (flow-related enhancement, even echo rephrasing). Also, MRI may not demonstrate intramural haematoma in smaller vertebral arteries.[2] The small irregular right vertebral artery seen at MRA in this patient may represent hypoplasia or dissection. It would be difficult to differentiate between the 2 conditions solely on MRA findings. The unchanged imaging appearance of the narrowed and irregular right vertebral artery on follow-up MRA

does not provide more information with regard to the cause. Since no catheter angiography was performed, it would be difficult to ascertain whether bilateral vertebral dissections were not present at initial presentation. The widely patent posterior communicating artery on both sides may complicate the issue of actual posterior circulation compromise in the event of vertebral artery dissection. The initial private MRI was reviewed for any thrombus at bilateral vertebral arteries. Unfortunately, the cervical region was not included in the scan.

A recent study of Husson et al compared the results of MRA with those of CA in children with arterial infarction.[5] In this series of 26 brain infarcts, MRA was concordant with CA in detecting defects in large cerebral vessels. However, MRA was found to overestimate the degree of stenosis [up to 25%] and underdiagnose distal arterial lesions. The spatial resolution of MRA, which is inferior to CA, can lead to an imprecise description of the nature of the lesion. The extent of collateral flow may be not accurately demonstrated by MRA.[5]

This patient demonstrates the importance of considering vertebral artery dissection in patients with posterior fossa infarct. MRI and CE MRA are useful for initial investigation and follow-up study. The MRA findings of this patient are unique in that they show that recanalisation of the dissected vessel may occur with occlusion of the previously unaffected segments. Follow-up angiography/MRA in other studies of vertebral artery dissection have shown either occluded vessels or arteries that have returned to normal.[6,7]

To conclude, vertebral artery dissection should be considered for patients presenting with posterior fossa infarct. MRI and MRA, including the neck region, is useful for initial diagnosis and follow-up.

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