Altered vertebrobasilar flow in children: angiographic, MR, and MR angiographic findings.

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Altered Vertebrobasilar Flow in Children: Angiographic, MR, and MR Angiographic Findings

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PURPOSE: To characterize the clinical, MR, MR angiographic, and conventional angiographic findings in vertebrobasilar disease in children. **METHODS:** Eight children with posterior circulation ischemia and infarction had conventional spin-echo MR and MR angiography of the head and neck. Six patients had conventional angiography. **RESULTS:** Six patients had alteration of vertebral or basilar artery flow void on spin-echo images. MR angiography showed all six cases of angiographically proved vertebrobasilar dissection or occlusion despite overestimating the extent of arterial abnormality in two patients. In two patients the intracranial peripheral branch cutoff shown at angiography was correctly predicted on screening MR angiography. **CONCLUSION:** Posterior circulation infarction in children is usually secondary to traumatic injury to the vertebrobasilar circulation. MR and MR angiography noninvasively show vertebrobasilar flow disturbances and compare favorably with angiography in documenting dissection or occlusion of the vertebrobasilar circulation. MR angiography may obviate the need for invasive angiography in these children at diagnosis and during follow-up of anticoagulation therapy.

Index terms: Arteries, stenosis and occlusion; Magnetic resonance, in infants and children; Magnetic resonance angiography

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Posterior circulation vascular occlusive disease in children is a rare and uncommonly reported event. Unlike adults, in whom underlying vascular disease accounts for a high percentage of posterior circulation infarction, in children, traumatic injury to the cervical vertebral artery is the most commonly reported cause of vertebrobasilar occlusion (1). With the advent of noninvasive vascular imaging, particularly magnetic resonance (MR) imaging and MR angiography, there may be an increase in the incidence of detection of posterior circulation stenoses and occlusions in children. We report the clinical and neuroimaging findings in eight children with vertebrobasilar vascular occlusive disease detected with MR and MR angiography and confirmed with conventional angiography. The role of MR and MR angiography was assessed in relationship to the standard of reference of conventional angiography.

Patients and Methods

Between August 1991 and December 1993, eight children 2 to 16 years of age (mean age, 8.2 years) had MR and MR angiography detection of posterior circulation dissection or occlusion. Seven patients had clinical evidence of posterior circulation infarction. The eighth patient had clinical signs of transient posterior circulation ischemia. MR imaging and MR angiography were performed with a 1.5-T system. Sagittal T1-weighted imaging (450/11/1 [repetition time/echo time/excitations]) and axial T2-weighted imaging (2500/30,90/0.75) of the head was performed in each patient. Axial-T1 weighted imaging (550/10/2) of the neck was performed in six patients. Axial T1-weighted imaging (500/16/2) of the head was performed in four patients. All patients had three-dimensional time-of-flight MR angiography of the circle of Willis (36/6.9, 20° flip angle, spoiled gradient-recalled acquisition, 60-mm section, 1-mm partition thickness). Seven patients had 2-D time-of-flight MR angiography of the neck (45/8.7, 60° flip angle, spoiled gradient-recalled acquisition, 1.5-mm section thickness). Both individual source images and reconstructed maximum-intensity projection images

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were evaluated. Six children underwent conventional angiography with selective bilateral vertebral artery injection. Four patients had bilateral carotid conventional angiograms. Correlative angiography was performed within 24 hours of MR imaging in five patients. The sixth patient (patient 7) had angiography 6 days after MR imaging. Follow-up MR and MR angiography were performed in five patients. One patient (patient 4) had follow-up angiography to distinguish traumatic basilar artery injury from underlying fibromuscular dysplasia. Three patients (patients 3, 4, and 5) had plain radiographs of the cervical spine. In addition, patient 3 had lateral flexion-extension views of the cervical spine, whereas patient 5 had a CT examination of the upper cervical spine. Clinical follow-up ranged from 1 to 15 months. The study results were retrospectively reviewed by three radiologists experienced in pediatric neuroradiology.

Results

Clinical and neuroimaging data are summarized in the Table. Causes of vascular occlusion included trauma (five patients) and migraine (one patient); in two patients it was idiopathic. Five patients had clinical and MR evidence of repeated episodes of infarction. Progressive infarction was found in four of five patients with traumatic vertebral artery injury (patients 1, 2, 3, and 4) but in only 1 patient with idiopathic basilar artery occlusion (patient 6). No clinical or MR evidence of progressive vascular occlusion occurred in any patient after initiation of anticoagulation therapy.

High-signal changes on T2-weighted imaging in the brain stem, cerebellum, thalamus, splenium of the corpus callosum, or temporoccipital cortex compatible with infarction were found on initial MR in seven of eight patients (all but patient 5). Altered vertebral or basilar arterial flow void was found on axial T1-weighted images in six patients (all except patients 7 and 8). Patient 4 had focal narrowing of the basilar artery flow void, whereas five patients had high signal intensity in a vertebral or basilar artery (Fig 1) (patients 1, 2, 3, 5, and 6). Patient 2 had a bilateral vertebral artery flow void abnormality. Patterns of flow void high signal intensity included eccentric intraluminal high signal (two vessels, patients 1 and 2) and concentric intraluminal high signal (four vessels, patients 2, 3, 5, and 6) on axial T1-weighted images. Intraluminal high signal intensity correlated in all six patients with angiographically proved sites of intimal injury, thrombus formation, or vascular occlusion.

MR angiography showed all six cases of angiographically documented vertebrobasilar dissection or occlusion (sensitivity and specificity of 100%). MR angiographic findings included absence, narrowing, or irregularity of the vertebral or basilar artery on the maximum-intensity projection images (Fig 2). Given the high spatial resolution and thin sections of the individual source images, careful analysis of these images was required to confirm findings displayed on reconstructed maximum-intensity projection images. Peripheral branch occlusion (superior cerebellar artery or posterior cerebellar artery) was found on MR angiography in three patients (patients 3, 7, and 8) without obvious peripheral branch flow void abnormalities on conventional images. Cervical 2-D time-of-flight MR angiography overestimated the extent of angiographically proved vascular occlusion in two patients with vertebral artery dissection (patients 1 and 5). Specifically, the level of MR angiography abnormality exceeded the level of angiographic abnormality by four vertebral body levels in these two patients because of angiographically documented slow flow. Five patients had follow-up MR angiography ranging from 2 weeks to 15 months (mean, 7.3 months). A recanalized basilar artery was found in patient 6, whereas peripheral branch recanalization (superior cerebellar artery and posterior cerebral artery) was documented in patients 3 and 7 (Fig 3). Follow-up MR angiography showed interval decreased irregularity of both vertebral arteries in patient 2 and decreased basilar artery irregularity in patient 4.

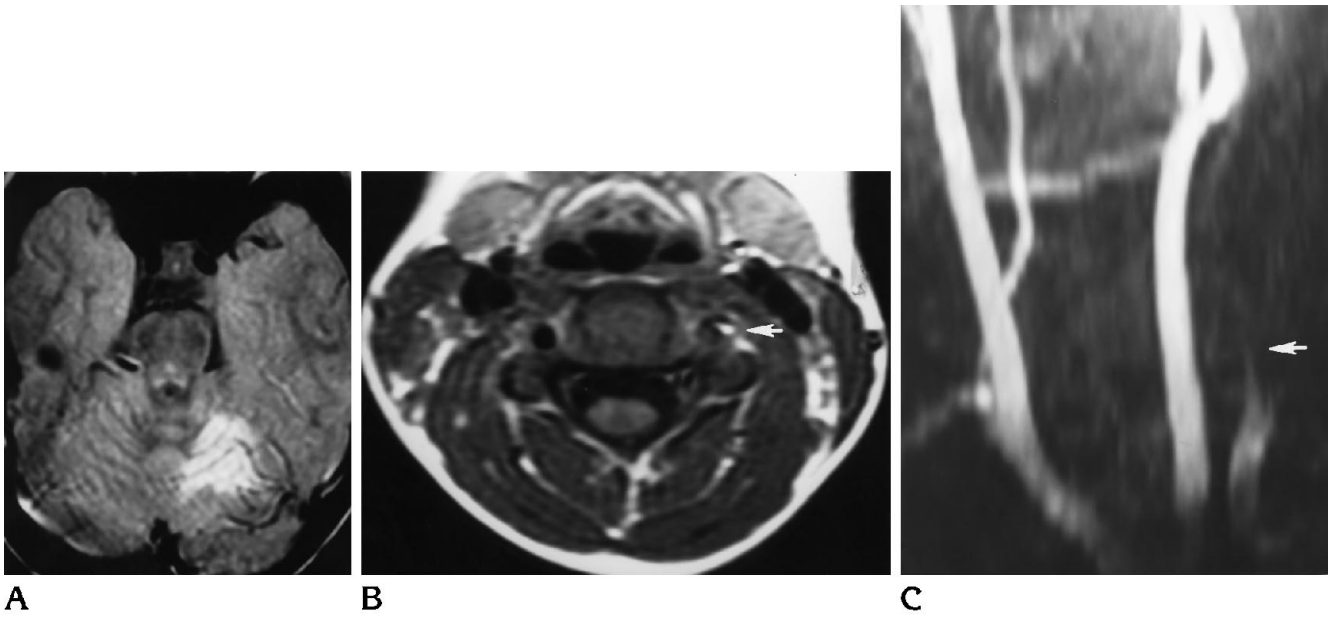
Angiographic findings included vertebrobasilar narrowing, irregularity, or occlusion. Angiography confirmed peripheral branch cutoffs in patients 3 and 7 and was not performed in patient 8, who had migraine headaches. MR angiography overestimation of angiographic abnormalities was shown by conventional angiography to be related to slow flow associated with dissection or occlusion (Fig 1). A follow-up angiogram in patient 4 confirmed interval resolution of basilar artery intimal injury. Intimal flaps were not seen in this series on MR angiography or at angiography.

Radiographic evaluation of the cervical spine was normal in patients 3 and 4. Patient 5 had a fracture of the C-2 vertebral body with extent into the right C-2 foramen transversarium.

Clinical and imaging findings in eight children with vertebrobasilar disease

Patient	Sex/ Age, y	Clinical Signs	Cause	MR Flow Void	MR Angiography	Angiography	Imaging Follow-up	Hospital Course	Treatment	Clinical Outcome
1	M/6	Vomiting, ataxia, dysarthria, L hemiparesis	Trauma	L vertebral: eccentric increased signal C-2 to C-4; R vertebral: normal	L vertebral: absent above C-6; R vertebral: normal	L vertebral: slow flow irregular at C-2; R vertebral: normal	None	Progression	Heparin, warfarin	1 mo: no deficits
2	M/6	Dizziness, ataxia, visual disturbance, R hemiparesis	Trauma	L vertebral: narrow, eccentric increased signal; R vertebral: narrow concentric increased signal	L vertebral: narrow, irregular at C-2; R vertebral: absent above C-2	L vertebral: narrow, irregular at C-2; R vertebral: slow flow narrow, irregular at C-2	1-y MR angiography: decreased bilateral vertebral irregularity	Progression	Heparin, warfarin	5 mo: no deficits
3	M/15	Headache, vomiting, dizziness, visual disturbance, dysarthria, dysphagia, ataxia	Trauma	Basilar: narrow, concentric increased signal	Basilar: narrow; Cutoff: bilateral SCA, left PCA	Basilar: narrow; Cut-off: bilateral SCA, left PCA	5-mo MR: improved basilar flow void; MR angiography: recanalized left SCA, left PCA	Progression	Heparin, warfarin	6 mo: dysarthria, ataxia decreased
4	M/16	Lethargy, R hemiparesis	Trauma	Basilar: narrow	Basilar: narrow	Basilar: narrow, irregular	2-wk MR angiography: decreased basilar irregularity; angiogram: decreased basilar irregularity	Progression	Observation	2 mo: no deficits
5	F/2.5	Decreased level of consciousness	Trauma	L vertebral: normal; R vertebral: concentric increased signal C-2	L vertebral: normal; R vertebral: absent above C-6	L vertebral: normal; R vertebral: Slow flow, occluded at C-2	None	Stable	Observation	3 mo: no focal deficits
6	F/5	Vomiting, seizure, dysphagia, R hemiparesis	Unknown	Basilar: concentric increased signal	Basilar: absent	None	15-mo MR: normal; MR angiography: normal	Progression	Heparin, warfarin	15 mo: persistent R hemiparesis
7	F/8.5	Headache, vomiting, visual disturbance, R hemiparesis	Unknown	Normal	Cutoff: left PCA	Cut-off left PCA	4-mo MR angiography: recanalized left PCA	Stable	Aspirin	4 mo: no deficits
8	M/9	Headache, R body sensory loss	Migraines	Normal	Cutoff: right PCA	None	None	Stable	Verapamil	3 mo: decreased headaches, no deficits

Note.—SCA indicates superior cerebellar artery; PCA, posterior cerebral artery.



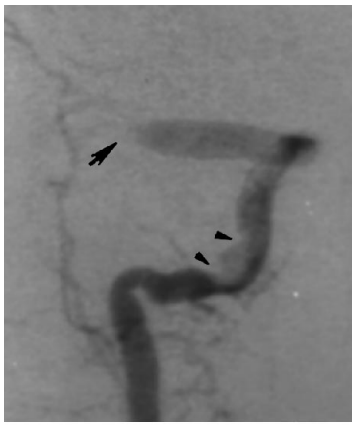


Fig 1. Patient 1, 6-year-old boy with acute ataxia and dysarthria after neck trauma.

A, Axial proton density–weighted image (2500/30/0.75) shows focal high signal in the left cerebellar hemisphere compatible with infarction.

B, Axial T1-weighted image (550/12/2) demonstrates eccentric high signal intensity within the left vertebral artery flow void (*white arrow*). Note normal right vertebral artery flow void.

C, Cervical 3-D time-of-flight MR angiography shows absent left vertebral artery signal above the C-6 vertebral level (*white arrow*).

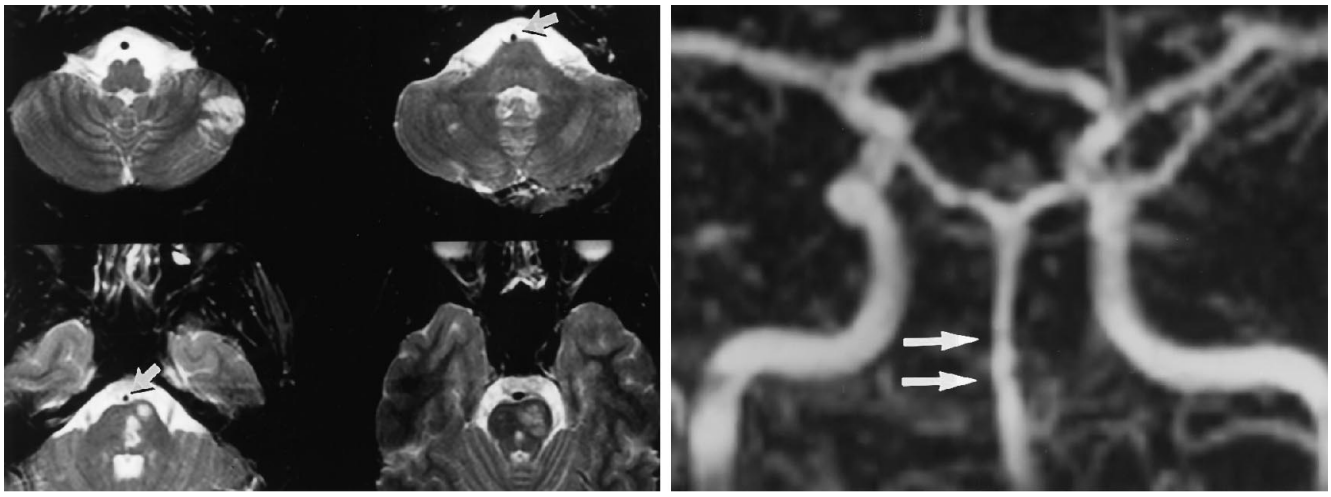
D, Digital subtraction left vertebral artery angiogram displays irregularity and multifocal narrowing of the left vertebral artery at the C-2 level (*black arrowheads*) consistent with intimal injury. Slow flow was noted throughout the left vertebral artery, which was occluded at the skull base (*black arrow*).

Discussion

Anterior circulation infarction in children is well known and has many possible causes, including an underlying vascular malformation, sickle cell disease, and emboli from cardiac or extracardiac sources (3, 4). Posterior circulation infarction in children is much less commonly reported than in adults, and indeed, very few cases were reported before the advent of computed tomography and MR imaging (5).

Causes of posterior circulation infarction in adults include underlying vascular disease such as hypertension, atherosclerosis, fibromuscular dysplasia, and migraine (6–8). Traumatic injury of the adult vertebral artery after cervical manipulation or minor trauma is well known (9, 10). In children, almost all reported cases with known causes have been secondary to trauma, as in five of our eight patients (1).

The cervical vertebral artery is protected from injury by the foramina transversaria of the second through sixth cervical vertebrae (11). At these levels vertebral artery injury is usually secondary to vertebral fracture as in patient 5 of our series. The most vulnerable location for intimal injury of the cervical vertebral artery is the point at which the artery exits from the C-2 transverse formamen before piercing the atlantooccipital membrane. Studies on cadavers have shown that severe extension, rotation, and longitudinal traction on the cervical spine can result in unilateral or bilateral occlusion of the vertebral artery at or above the C-2 level (12). Cervical spine anomalies and/or instability are thought to increase the risk of vertebral artery injury at this site after trauma, and



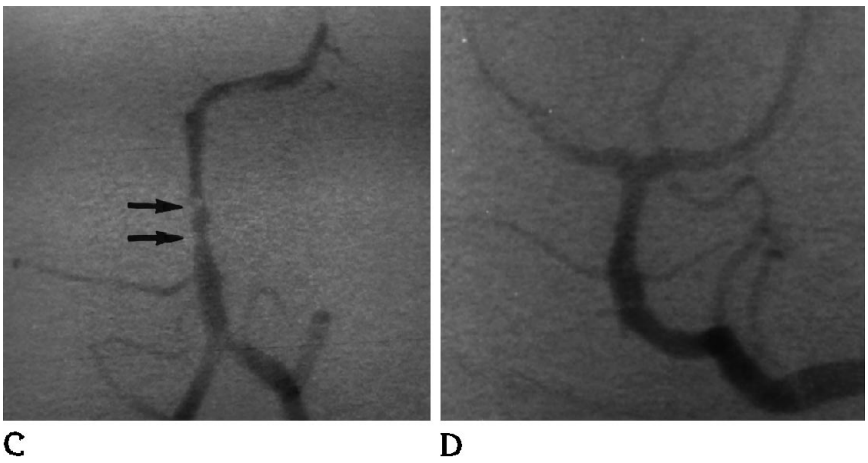


Fig 2. Patient 4, 16-year-old boy with abrupt onset of lethargy and right hemiparesis after a motor vehicle accident and severe blunt head trauma.

A, Axial T2-weighted image (2500/30/0.75) shows high-signal-intensity lesions in the left pons and left cerebellum. Narrowing of the midbasilar flow void is detected (*arrows*).

B, Intracranial 3-D time-of-flight MR angiography shows focal irregularity and narrowing of the basilar artery (*white arrows*).

C, Digital subtraction angiogram confirms focal irregularity and narrowing of the basilar artery (*arrows*).

D, Follow-up angiogram at 2 weeks shows interval resolution of the prior basilar artery irregularity and narrowing. The diagnosis of resolving traumatic intimal injury to the basilar artery was made.

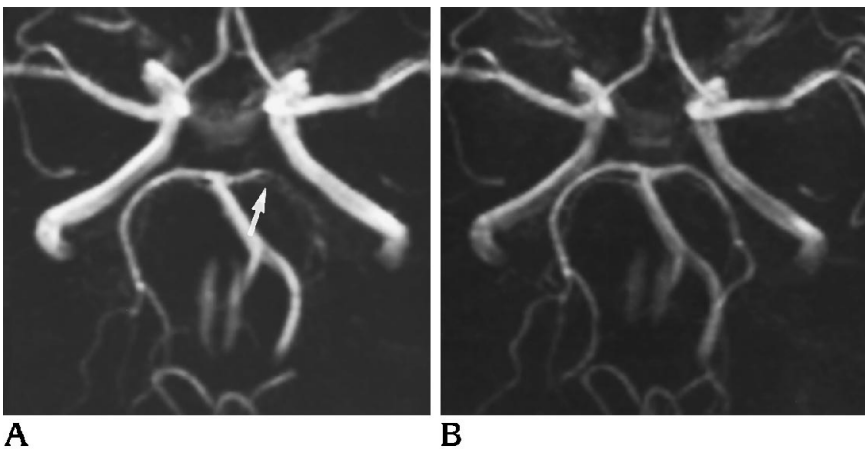


Fig 3. Patient 7. Abrupt onset of headache, visual disturbance, and right hemiparesis in an 8-year-old girl with no history of head or neck trauma.

A, Collapsed image from 3-D time-of-flight MR angiography shows irregularity and cutoff of the left posterior cerebral artery (*white arrow*).

B, Four month follow-up MR angiography displays interval recanalization of the left posterior cerebral artery in this patient, who received aspirin therapy (from Allison et al [2]).

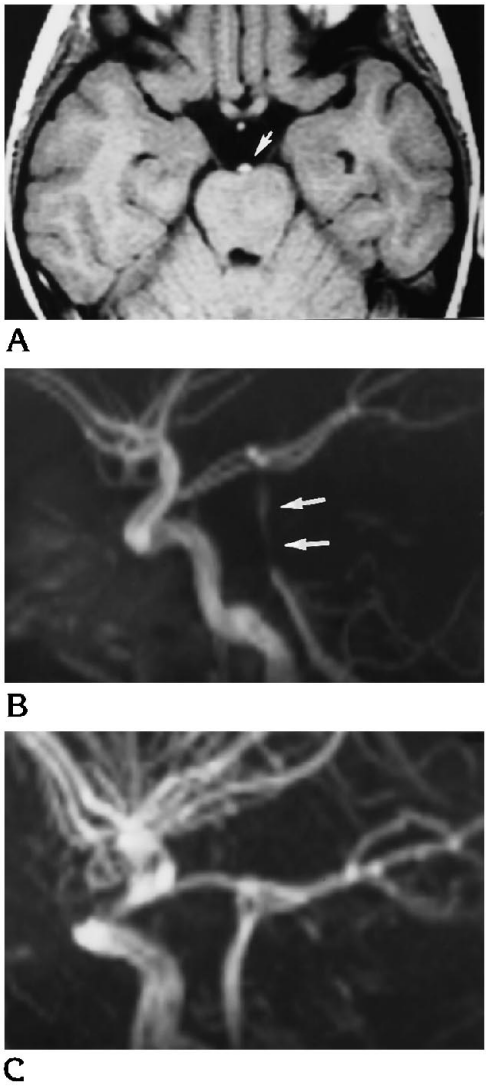


Fig 4. Patient 6, 5-year-old girl with vomiting, seizure, and right hemiparesis.

A, Axial T1-weighted image shows abnormal high signal intensity within the basilar artery (*white arrow*).

B, 3-D time-of-flight MR angiography confirms occlusive disease of the basilar artery (*white arrows*).

C, Follow-up 3-D time-of-flight MR angiography at 15 months shows a normal basilar artery; this patient received anticoagulation therapy.

surgical stabilization may be a consideration (13, 14). Although less well described, traumatic injury to the basilar artery, as in patient 4, may be encountered.

Vasoreactivity caused by migraine has been shown to be associated with posterior circulation infarction. One study correlating angiography with MR angiography in migraineurs reported basilar artery narrowing or occlusion and peripheral branch obstruction in one child (8).

In addition to parenchymal infarction, conventional spin-echo MR can show large-vessel arterial obstruction or slow flow by showing intraluminal high signal on short-repetition-time/-echo-time or long-repetition-time/-echo-time pulse sequences (Fig 4) (15). Although intraluminal high signal with technical causes (flow-related enhancement, even-echo rephasing, etc) may be occasionally encountered in normally patent vertebral or basilar arteries, in our patients intraluminal signal abnormality was noted on multiple axial images in conjunction with clinical and MR evidence of ischemia (16). The use of MR angiography in the evaluation of cervical carotid stenosis and occlusion in adults is well documented. MR angiography has been recently reported to have a sensitivity of 97% and a specificity of 98.9% in detecting angiographically proved stenoses and occlusion in adult patients with cerebellar or brain stem ischemia (17). Little information, however, is available in children. Noninvasive detection of posterior circulation vascular occlusion in children is significant because of the greater difficulty in obtaining vascular access in younger patients, the need for general anesthesia during many angiographic procedures in children, and the need for noninvasive follow-up examinations. All six angiographically proved posterior circulation vascular dissections and occlusions in this series were first detected with a combination of conventional spin-echo imaging and 2-D or 3-D time-of-flight MR angiography. Analysis of individual source images in addition to the reconstructed maximum-intensity projection images improved visibility of MR angiography findings. MR angiography overestimated the angiographic extent of disease in patients 1 and 5, relating to slow flow, a phenomenon well known in carotid disease in adults. Overestimation of the extent of occlusion because of slow flow might be decreased by the use of phase-contrast MR angiography with low-velocity flow encoding, a technique not used in this patient group.

Initial treatment of vertebral artery dissections includes restriction of activities, limitation of neck motion via a cervical collar, and anticoagulation and antiplatelet therapy (18). In adults, endovascular or surgical treatment of the vertebral artery may be indicated after repeated embolic events despite anticoagulation therapy, subarachnoid hemorrhage, or aneurysm formation (19, 20). Most children with

posterior circulation infarction can be expected to recover without surgery (1). Although there are little data about the need for anticoagulation in children with posterior circulation infarction, four children in this study (patients 1, 2, 3, and 6) were anticoagulated for vertebral or basilar artery dissection or occlusive disease detected with MR angiography. Confirmatory angiography was performed in three of these patients before anticoagulation (patients 1, 2, and 3). Symptoms of progressive posterior circulation ischemia abated in these patients after onset of anticoagulation therapy.

When a child presents with abrupt onset of neurologic signs and symptoms indicative of posterior circulation ischemia, a history of traumatic injury to the neck and head should be sought. A migraine history should be elicited. Cervical spine radiographs to detect a fracture or underlying cervical spine instability should be performed. The imaging work-up should proceed to MR imaging and MR angiography to define those patients with infarction in the posterior circulation distribution. MR and MR angiography can show vertebral and basilar artery dissection caused by trauma or peripheral branch cutoff from a migranous or unknown cause. MR and MR angiography allows noninvasive assessment of vessel status during treatment. Conventional angiography should show more vertebral artery intimal flaps than cervical MR angiography; however, no intimal flaps were identified at angiography in our patients. Fortunately, a good neurologic outcome and low likelihood of long-term recurrence can be expected (21). The need for invasive angiography in this select group of children may be limited to those in whom surgical or neurointerventional therapy is being considered. In addition to the noninvasive advantage of MR angiography over angiography, economic benefit should also be realized, because the approximate cost of MR angiography is much lower than the cost of posterior circulation angiography.

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