

Vertebral Artery Dissection With Bilateral Hemiparesis

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Right vertebral artery dissection after trauma, with subsequent cerebellar and thalamic infarcts, is described in a 5-year-old child who presented with mild hemiparesis and ataxia. This vascular injury has been more commonly reported in adults. The diagnosis was confirmed with magnetic resonance imaging of the brain and cerebral magnetic resonance angiography. An arterial thrombus with multiple embolization was the cause of the child's neurologic deficits. Traumatic injury to the posterior circulation should be considered in children who have signs of vertebrobasilar insufficiency.

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Introduction

Cerebrovascular accidents are uncommon phenomena in children, occurring at a prevalence of 2.5 per 100,000 per year [1]. Events involving posterior circulation vessels are not common, especially arterial dissection. Dissections of the vertebral artery are documented secondary to chiropractic manipulation, sudden head turning, neck flexion during paroxysmal coughing, tobogganing, and amusement rides [2-5]. We report a case in which the initial history did not uncover trauma as a cause for neurologic findings. Nonetheless, a vascular etiology was investigated because of the multiple areas of infarction.

Case Report

A 5-year-old previously healthy boy was admitted because of right-sided weakness and limp to Cedars Sinai Medical Center after evaluation at an outlying clinic where he presented with left knee pain after losing his balance and falling. The parents had observed excessive giddiness, although there was no headache, emesis, visual disturbances, or neck

pain. Three days before admission the child had fallen, became less active, and developed a slight limp. Prior history was noncontributory. After admission, 1 parent reported that days earlier the boy had come crying and shivering complaining of falling backwards while descending the stairs and striking his occipital region.

On admission, his blood pressure was 128/76 mm Hg, pulse 88 beats/min, and temperature 36.7°C. On physical examination, his head was normocephalic and atraumatic. Neck was supple with full range of motion. Heart sounds were remarkable for vibratory grade 2 systolic murmur loudest at middle left sternal border. Bilateral extremity weakness was observed, right significantly greater than left with foot drop right greater than left. Deep tendon reflexes were equal and symmetric though slightly brisk without clonus. Babinski response was present bilaterally. His gait was ataxic with weight borne more on the left foot. Sensation to pinprick and touch were intact. He had a right central facial weakness and a rightward deviation of his protruded tongue. Mild dysconjugation with upward gaze (left higher than right) was observed. He demonstrated mild to moderate dysmetria with upper and lower extremities.

Results of laboratory studies were: hemoglobin 139 gm/L, hematocrit 0.41 (41%), leukocyte count 10.1×10^3 cell/ μ L, and platelet count 250×10^9 /L. Hemoglobin electrophoresis revealed a normal AA₂ pattern. Prothrombin time was 12.5 s (control, 11.9 s), partial thromboplastin time 34 s (control 24-37 s). A serum fibrinogen level was 2.26 gm/L, erythrocyte sedimentation rate 5 mm/hr (normal <20 mm/hr). Urine amino acids including cystine and urine organic acid screens were normal. Antinuclear antibody was negative. Urine toxicology screen was negative.

Cranial magnetic resonance imaging (MRI) with and without gadolinium on the second hospital day revealed subacute bilateral thalamic and cerebellar infarcts (Fig 1). Magnetic resonance angiography suggested poor flow in the posterior cerebral arteries. Later on hospital day 2, the patient was observed to have dysarthria and was transferred to the pediatric intensive care unit for closer neurologic assessment of possible stroke in progress. An echocardiogram was negative for vegetations. Serum levels of protein C, protein S, and antithrombin III were obtained and were subsequently normal. Cerebral angiography on hospital day 5 demonstrated sharp narrowing in the horizontal portion around C₂ with 90% stenosis compatible with vertebral artery dissection; both posterior cerebral arteries were occluded in the most proximal segments with good filling of the occipital lobes via middle artery collateralization (Figs 2 and 3). Arterial thrombus with multiple emboli as the cause of neurologic deficits was the likely diagnosis. After results of this study became available, anticoagulation with a continuous infusion of heparin was begun. The partial thromboplastin time was kept between 1.5 to 2 times the control value. Subsequent computed tomographic axial sections of the neck were negative for cervical fractures on day 6.

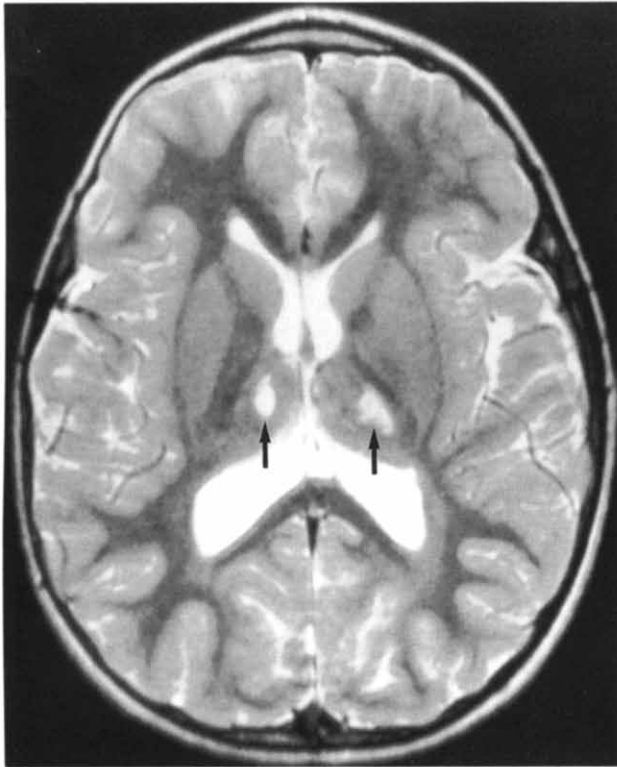
His hemiparesis, ataxia, and speech improved throughout his hospital course although foot drop persisted, right more than left. He was discharged from the hospital on aspirin and received neurologic follow-up and physical and occupational therapy. For 1 month after discharge he complained of migraine quality headaches. Three months after discharge, he began riding his bicycle and mild amnesia of the hospitalization resolved. Five months after discharge, his examination is remarkable for minimal coarse tremor and unchanged foot drop.

Discussion

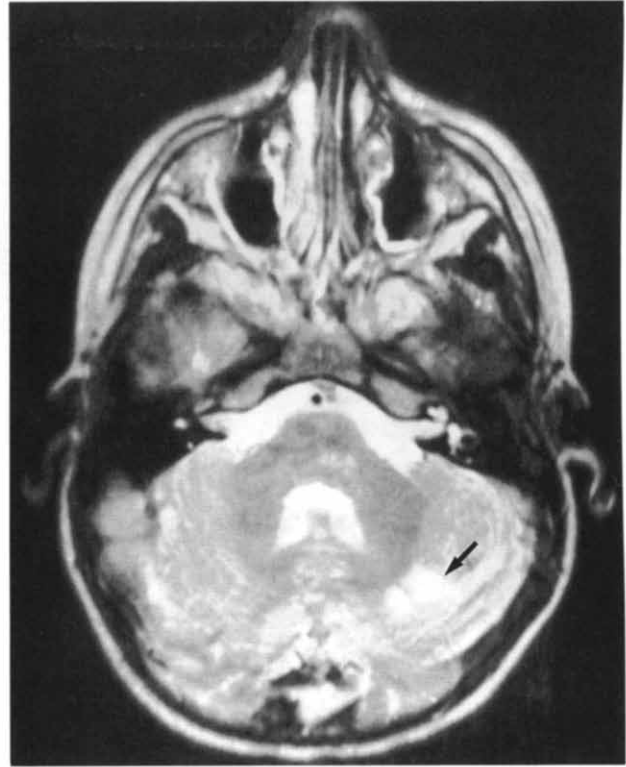
Cerebrovascular infarctions are rare events in childhood and adolescence. Many times, an etiology cannot be found [6]. These events occur more commonly in the distribution of the middle cerebral artery. Thrombotic causes include

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A



B

Figure 1. Cranial MRI axial T₁-weighted images demonstrate a low intensity signal that did not enhance. Axial T₂-weighted images (TR: 3,600 ms, TE: 102 ms) display high signal intensity in the bilateral thalami (arrows in A) and left cerebellum (arrow in B) consistent with infarction.

arteriosclerosis (premature onset with lipid abnormalities), coagulopathies, sickle cell disease, meningitis, moyamoya syndrome, lupus cerebritis, Fabry disease, and homocystinuria. Embolic causes include cardiac disease, and trauma to the neck with arterial thrombus formation and embolization, usually with involvement of the carotid artery.

Carotid and vertebral artery dissection is a relatively common cause of strokes and transient ischemic attacks in young and middle-aged individuals [7]. Pain usually accompanies both traumatic and spontaneous dissections, but may be absent. Because of its relative immobility, the vertebral artery is prone to injury at the C₁-C₂ joint com-

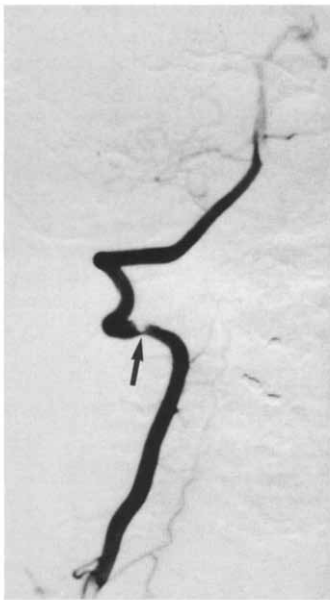


Figure 2. Right vertebral angiogram demonstrates extended intraluminal filling defect at C₁-C₂ level (arrow).

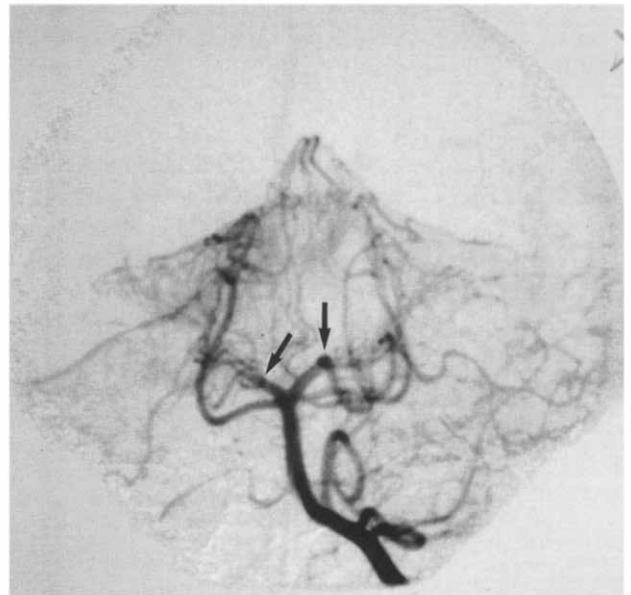


Figure 3. Left vertebral angiogram (anterior-posterior view) demonstrates absence of filling of basilar artery from right vertebral artery with no flow from the posterior circulation above tentorium (arrows).

plex, from rotation or hyperextension due to stretch and mechanical trauma [3]. However, embolism from dissected areas of this vessel is not usually the pathologic process involved although various authors have found evidence of this [5,8,9,]; rather thrombus extension or dissection occluding the origin of artery branches is responsible [10]. Sometimes, an obvious pathologic process is not apparent and both thrombus propagation/dissection progression or embolization are suggested as explanations [11,12]. Either pathologic process would cause some degree of basilar artery occlusion and might involve a large number of brainstem structures. Many brainstem syndromes resulting from particular infarction patterns have been described (medial and lateral medullary; inferior/middle/superior pontine; midbrain tectum and tegmentum; posterior cerebral territory) and labeled with eponyms such as Wallenberg, Benedikt, Weber, Parinaud, top-of-the-basilar [13]. Any of these hypoperfusion syndromes may instead be symptoms of vertebrobasilar migraine [9]. Of course complete obstruction of basilar flow will result in coma and probably death, if collateral flow from the anterior circulation does not come through the circle of Willis.

In our patient, the physical findings of bilateral weakness, right greater than left, were presumably due to thalamic infarcts which bordered on, and probably included, the posterior limb of the internal capsule. This pattern is consistent with the clinical presentation of mild hemiparesis that persisted. Hemiparesis due to cortical infarction in children tends to be less persistent. Conversely, the limb dysmetria and gait ataxia which were prominent on admission were probably due to cerebellar infarcts. These findings have essentially resolved, consistent with a relatively good prognosis of cerebellar infarction in children. No sensory abnormalities were ever demonstrable, presumably because of the difficulties of a reliable detailed sensory examination in a young child. Initial dysconjugate upward gaze was probably due to asymmetric involvement of the most rostral midbrain, that is cranial nerve III or its nucleus.

Children that present with posterior circulation deficits should be investigated for vertebrobasilar lesions, because these patients may have sustained only minor trauma that is not easily recalled by interview, and further impairment from emboli may be preventable. Arteriography will delineate the vascular injury, while MRI rather than CT scan will demonstrate posterior fossa infarcts.

In our review of the literature, there is no consensus therapy for infarction associated with posterior circulation dissection in children. We perceive a reluctance to acutely

anticoagulate with heparin but a preference for antiplatelet therapy. Chronic anticoagulation therapy with aspirin does seem to be of benefit to prevent further infarctions [3,11]. Long-term outcome in adults treated with anticoagulants appears to be good [14]. Children recovered relatively well in all reports whether they received heparin, aspirin, or no medication. Although therapy considerations must be based on adult literature, we believe aspirin is probably sufficient as the primary and perhaps the sole treatment.

Strokes in children are uncommon, especially in the posterior circulation. In these reports, patients frequently have congenital abnormalities of the vertebral vessels or normal anatomy but a strong history for trauma. When children have findings of posterior circulation impairment, a history of trauma should be pursued so that appropriate investigation and therapy is not delayed.

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