

Ischemic cerebellar infarct in a 5-year-old boy: Sequela to minor back trauma

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Minor trauma, a kick to the center of the back during roughhousing with an 8-year-old sibling, produced an ischemic cerebellar infarct in a 5-year-old boy. The boy was brought to medical attention because of vomiting and headache. He also had ataxia. Results of laboratory studies, structural examination, and cerebrospinal fluid analysis were non-contributory. An irregular, low-density region in the left cerebellar hemisphere of unknown cause was seen on computed tomography scans. Magnetic resonance imaging (MRI) revealed an area of signal alteration hyperintense on the T2-weighted image and mildly hyperintense on the proton-density image in the leftward cerebellar hemisphere. The distribution was most compatible with that of an acute superior cerebellar arterial infarction. No other abnormalities were detected. The patient was treated with oral prednisolone, recovered, and had no further episodes or sequelae. The authors recommend that post-traumatic stroke be considered early in the differential diagnosis of a child with the triad of symptoms seen in the patient described. They suggest that glucocorticoid therapy on a short-term basis may have a place in more rapid resolution of symptoms.

(Key words: Trauma, ischemic cerebellar infarct, childhood cerebellar stroke)

Childhood cerebellar stroke may follow relatively minor nonhead trauma. Clinical presentation may include ataxia, vomiting, and headache in a previously well child. We describe a 5-year-old

boy with these symptoms who had an ischemic cerebellar infarct after minor trauma to his back.

Report of case

A 5-year-old boy with a wide-based gait was seen because he had episodes of vomiting for approximately 12 hours. He was lethargic and complained of headache. He made intermittent inappropriate cognitive responses to questioning. His mother said that on the previous evening, the child suddenly started vomiting, had a severe headache, and had difficulty walking. Two hours before the onset of symptoms, the patient and his 8-year-old sister were scuffling, during which the patient was kicked in the center of his back. He did not lose consciousness and had no apparent head injury. The child had previously been healthy, with no other history of trauma or recent illness. He was taking no medications.

Initial vital signs were within normal limits. No evidence of trauma was found on physical examination. Structural evaluation of the cervical, thoracic, and lumbar spine revealed no tenderness or motion restriction. Tendon reflexes were symmetric and normal. Cranial nerves II through XII were intact. Abnormal physical findings included an impressive broad-based gait with deviation to the left. The child also had deviation to the left when standing with feet together. Finger-to-nose maneuvers on the left side were abnormal.

Results of routine laboratory studies were all normal. Computed tomography (CT) scans (*Figures 1 and 2*) revealed an irregular, low-density region in the left cerebellar hemisphere of unknown cause. Differential possibilities included low-grade neoplasm, atypical cyst, or early inflammatory process such as an abscess. An infectious etiology could not be ruled out, and a lumbar puncture was performed. Findings of cerebrospinal fluid analysis were unremarkable. Magnetic resonance imaging (MRI) revealed an area of signal alteration hyperintense on the T2-weighted image (*Figure 3, left*) and mildly hyperintense on the proton-density image in the leftward cerebellar hemisphere (*Figures 4, left and 5*). The distribution was most compatible with that of an acute superior cerebellar arterial infarction. No other abnormalities were detected.

In an attempt to prevent edema and its sequelae, oral prednisolone therapy, 2 mg/kg per day in divided doses, was started. The patient improved dramatically and was discharged on the third day with ataxia virtu-

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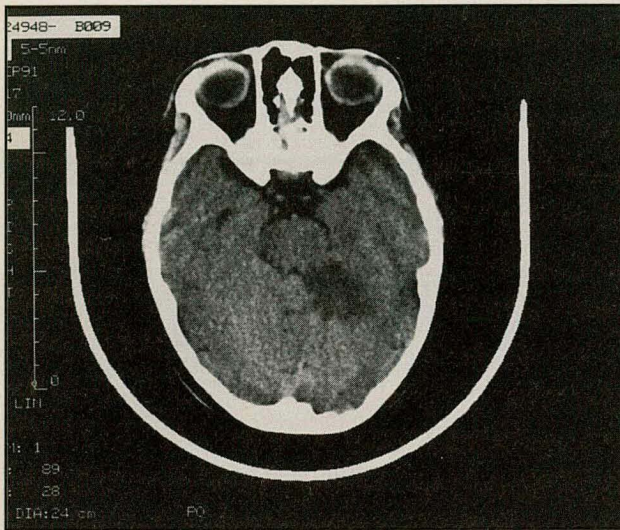


Figure 1. Initial noncontrast axial computed tomography scan shows an oval hypodense area in the high left cerebellum effacing left portion of quadrigeminal plate cistern.

ally absent. The patient had no headache or emesis. Prednisolone therapy was continued for an additional 5 days.

Follow-up examination 4 days after discharge from the hospital revealed total resolution of abnormal gait, headache, and emesis. Nine days after discharge, follow-up MRI studies showed resolving cerebellar infarct (*Figure 3, right*) and a resolving small left thalamic infarct (*Figure 4, right*) that had not been noticed on the original studies. Computed tomography 7 months later showed evidence of the old infarcted regions without new signs of infarction or intracerebral changes. Ten months after the initial problem, the patient had no neurologic sequelae, and he has had no recurrence of ataxia or severe headache.

Discussion

When ataxia is accompanied by headache and vomiting, suspicion of a posterior fossa etiology must be raised, with cerebellar stroke or posterior fossa tumor as distinct diagnostic possibilities. The presence of nystagmus, which was not seen in our patient, is also consistent with a pathologic process of the posterior fossa. Childhood stroke is infrequent and etiologies are diverse (*Table*). Trauma-induced stroke in children is especially uncommon, and cerebellar stroke is rare.

A review of the literature since 1972 found only 19 cases of stroke related to trauma in children,¹⁻⁹ and only 7 of those involved the vertebrobasilar system, which includes the cerebellar arteries. Strokes are more commonly caused by occlusion in the anterior carotid system. Sometimes, the cause of the stroke becomes a diagnostic enigma, especially in previously well children. Baumann and associates¹⁰ reviewed data on 215 consecutively imaged children referred to them because of neurologic disease. Of those 215 children, 28 showed

an arterial infarction pattern. Of these, a third had no associated illness or adverse event. Thirty-four percent of the cases of ischemic stroke cited in the Mayo Clinic Rochester study on childhood stroke¹¹ had no identified etiology. Others^{4,6,12} likewise report childhood stroke of unknown cause in their data. Mitral valve prolapse,¹³ arteriosclerosis,¹⁴ and migraines¹⁵ are proposed by some authors as the pathologic process involved in these strokes of unknown origin. We propose that minor trauma may be implicated as a causal factor as well and that it may be more prevalent in its association with childhood stroke than is currently reported in the literature.

The temporal relationship between the event and the onset of symptoms can be varied and prolonged. Delays of 2 to 8 months have been reported.^{1,2} Others⁷ cite development of minor symptoms after 48 hours and more severe symptoms after 72 hours. Symptoms are more likely to develop in 2 to 24 hours, as was observed in our patient. Because of these delays, the traumatic event may not even be remembered or may be dismissed as trivial and not associated with symptoms. In the case reported here, a history of only a very minor trauma was elicited. The patient and his 8-year-old sister were playing, and she accidentally kicked him in the back. Debehnke and Singer⁷ reported cerebellar and pontine infarcts after a minor bicycle accident without head trauma or loss of consciousness. Dooley and Smith⁸ found a history of only having been jarred as a passenger in an automobile collision 8 months before onset of symptoms in a previously healthy 6-year-old stroke victim. Ko and Berbrayer³ describe cerebellar infarct in a 9-year-old boy 10 days after a day of tobogganing without specific head or neck trauma. These cases suggest that the

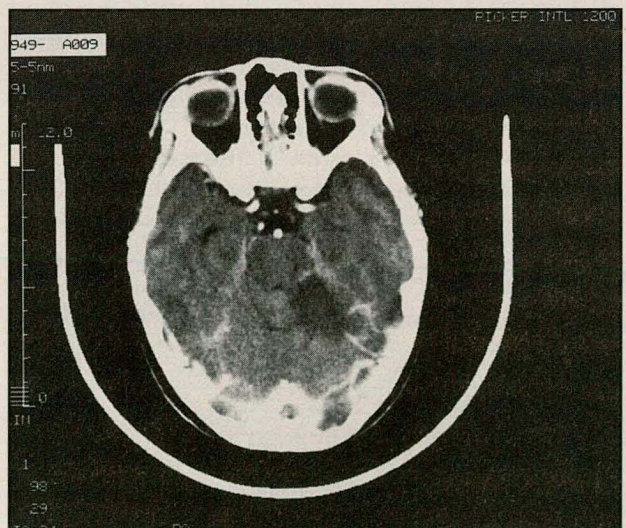


Figure 2. Initial contrast axial computed tomography scan shows no enhancement of lesion.

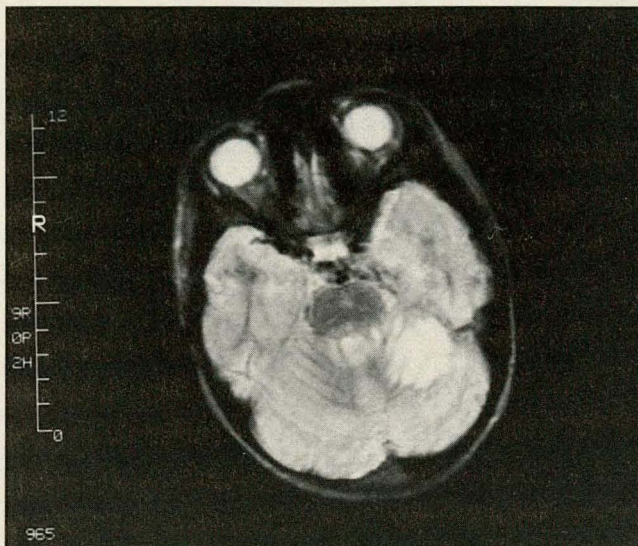


Figure 3. Left: Initial axial T2-weighted magnetic resonance image (MRI) demonstrating focal increased signal involving white and gray matter of left superior cerebellar hemisphere. Right: Axial T2-weighted MRI 9 days later demonstrates less

hyperintense edema in the left superior cerebellum when compared with earlier study (left). Lack of progression and, in fact, resolution of edema and size of lesion over time is consistent with infarct.

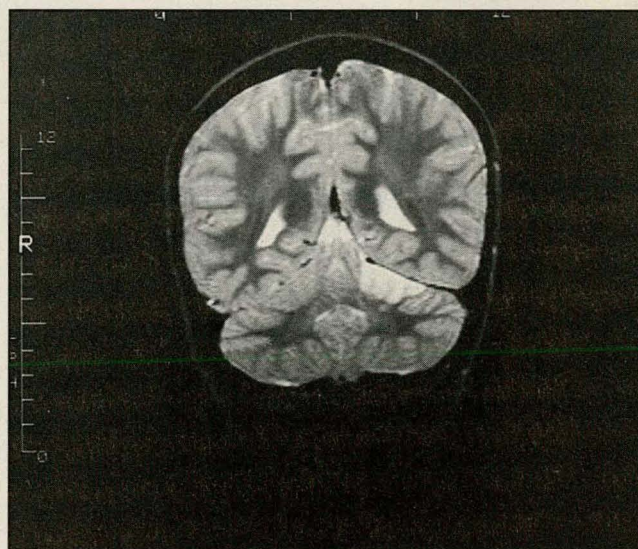


Figure 4. Left: Initial coronal T1-weighted magnetic resonance image demonstrating a well-demarcated hypointense lesion in the left superior cerebellar hemisphere. Right: Coronal T2-weight-

ed MRI 9 days later. Hyperintense lesion involving white and gray matter in the selective distribution of the left superior cerebellar artery.

traumatic etiology for childhood stroke may be underestimated and that children who have even minor head trauma may sustain cerebellar injury.

The mechanism of injury in our patient was thought to be possible vertebral artery dissection resulting from a whiplash kind of impact or trauma to the back. We also postulated that a sudden force could have potentially slammed the superior cerebellar artery up against the tentorium that divides it from the posterior cerebral artery. Others¹ have suggested that stretching of the vertebral arteries with platelet aggregation and thrombosis secondary to traction, hyperextension, or

rotational injuries to the neck might be the mechanism of injury. Fraser and Zimble² discovered anterior subluxation of vertebra C-1 on vertebra C-2, a common congenital anomaly, in their patient. They proposed that this anomaly caused mechanical deformation and occlusion of the vertebral arteries leading to stroke in their 6-year-old patient.

Diagnostic evaluation of children with ataxia, vomiting, and headache begins, as always, with taking a thorough history and completing a physical examination. Subtle historical clues must be sought, such as history of antecedent viral illness or minor trauma. Etiologies that may require imme-

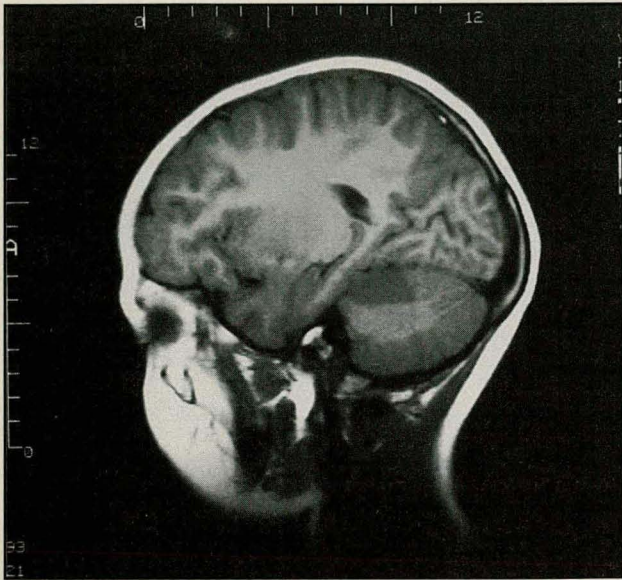


Figure 5. Initial sagittal T1-weighted magnetic resonance image shows focal wedge-shaped hypointense lesion in the left superior cerebellar hemisphere.

diate medical or surgical intervention should be considered first, including infection, stroke, hydrocephalus, posterior fossa neoplasm, and cervical vertebral fracture or instability

Findings consistent with acute cerebellar disease should have early evaluation by CT or MRI. Gentry and colleagues¹⁶ have shown MRI to be highly sensitive in detecting both hemorrhagic and nonhemorrhagic lesions. They compared MRI and CT examination to determine their relative sensitivities. They report that CT has not been particularly useful for classifying and staging injury and that it has low sensitivity for nonhemorrhagic lesions. They state that MRI offered distinct advantages over other imaging methods for classifying traumatic cerebral lesions. The ability of MRI to image in multiple planes was cited as particularly beneficial for localization of the lesion. Computed tomography has a diagnostic accuracy of approximately 60% in the first week after the episode, increasing to 100% in the second week.¹⁷ Computed tomography findings in our patient were inconclusive, and MRI was needed to make the final diagnosis. In the case of the bicycle accident reported by Debehnke and Singer,⁷ the CT scan showed no abnormality while MRI revealed cerebellar and pontine infarcts. The findings cited by Gentry and associated¹⁶ and the implications drawn from these two cases suggest that MRI be the initial diagnostic test when an ischemic event is suspected and that CT is more appropriate for follow-up evaluation 2 or more weeks later. In our institution, CT is more readily available and therefore was performed first.

Other diagnostic tests may need to be done, as

Table
Major Etiologies of Childhood Stroke¹⁴

■ **Arterial thrombosis and embolism**

- Arteriosclerosis
- Vasculitis—systemic lupus erythematosus
- Fibromuscular hyperplasia
- Sickle cell diseases
- Cardiac disease
 - Cyanotic congenital heart disease
 - Mitral valve prolapse
 - Myocardial infarction
- Infectious disorders
 - Meningitis
 - Bacterial infections
 - *Mycoplasma spp* infections
- Neurofibromatosis
- Moyamoya syndrome
- Homocystinuria
- Vertebrobasilar occlusion
- Trauma

■ **Venous thrombosis**

- Infant dehydration
- Polycythemia associated with cyanotic heart disease
- Pregnancy
- Oral contraceptives
- Nephrotic syndrome
- Purulent venous thrombosis
 - Mastoiditis
 - Otitis media
 - Scalp infections
 - Facial infections
- Sagittal sinus thrombosis with acute lymphoblastic leukemia

■ **Intracranial hemorrhage**

- Arteriovenous malformation
- Galen's vein malformation
- Aneurysm
- Intracerebral hemorrhage
 - Hypertension
 - Trauma

■ **Vascular disease of the spinal cord**

- Thrombotic infarction of the spinal cord from sickle cell disease

dictated by historical and clinical findings.

The major goal of therapy in stroke includes treatment to preserve brain function by maintaining circulation and oxygenation and prevention of subsequent neurologic deficits through the use of positioning, hyperventilation, and hyperosmolar agents. Steroid therapy is effective in treating cerebral edema that occurs after a stroke, and aspirin therapy has been shown to prevent recurrent ischemic events.¹⁸ The value of steroids in preventing neurologic deficit has not been proved, and their use in the pediatric stroke patient has not been established. However, we elected to admin-

ister oral prednisolone to our patient in an attempt to prevent further sequelae from edema. Clinical improvement was rapid and dramatic, and our patient has had no neurologic sequelae or recurrent attacks.

Ko,³ Rothrock and coworkers,¹⁵ and Jain and colleagues¹⁹ report pronounced improvement after the administration of steroids to their pediatric stroke victims as well. We propose that glucocorticosteroid administration on a short-term basis may bring about rapid resolution of symptoms in childhood stroke victims and that its efficacy in this setting warrants further study. Aspirin therapy was not used in our patient. Patients with a more advanced pathologic process may require surgical intervention.

Children seem to recover from strokes differently than adults. It is thought that this may be due to the development of axonal sprouting and the formation of new synapses or functional reorganization of normal structure in the young brain.¹⁷ Children can, however, still have devastating sequelae.

Comment

A physician must consider a vertebrobasilar or posterior fossa pathologic process when evaluating a child who is seen with ataxia, vomiting, and headache. Thorough history taking and physical examination are essential for discovering sometimes subtle clues as to the etiology of the symptoms and for dictating diagnostic studies that should be done. It is prudent to include CT or MRI in the initial diagnostic workup in order to expeditiously diagnose potentially lethal conditions that may require immediate medical or surgical interventions. It is suggested that MRI is more sensitive for evaluating patients with suspected ischemia and, where possible, should be a diagnostic consideration even if there is history of only minor nonhead trauma. Rapid diagnosis and treatment may prevent further damage and subsequent morbidity. Glucocorticosteroid therapy may have a place in more rapid resolution of symptoms and its use needs further study. Children seem to recover better from strokes than do adults.

The Strokes in Children Study Group, a section of the Joint Committee for Stroke Facilities, suggested in 1973 that strokes in children should be reported to a registry to permit better understanding of the natural history of these disorders. To our knowledge, this practice was never established. It would seem advisable that this project again be suggested. Perhaps with widespread reporting, we would have better insight into the prevalence and

etiology of stroke in children and spark interest in exploring treatment possibilities, such as glucocorticosteroid administration early in the course of the episode. Any progress in improving morbidity for these children would make this an important project.

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