

Cook County Hospital, Chicago, IL  
Department of Emergency Medicine  
(RDT)

University of Illinois College of  
Medicine, Chicago, IL  
Department of Emergency Medicine  
(EPS)

## Nonhemorrhagic Pontine Infarct in a Child Following Mild Head Trauma

Ross D. Tannebaum, MD, Edward P. Sloan, MD, MPH

### ■ ABSTRACT

A child who presented with hemiparesis secondary to a delayed non-hemorrhagic pontine infarction following mild head trauma is described. The results of the child's workup, including computed tomography (CT), were negative. The diagnosis of nonhemorrhagic pontine infarct was made by magnetic resonance imaging (MRI). The diagnostic evaluation excluded other possible etiologies of cerebral infarction, including vasculitides, CNS infection, congenital heart disease, hypercoagulable states, and demyelinating diseases. Although trauma cannot be proven as the cause of the infarct, other known causes of infarct were excluded. There are few cases of traumatic nonhemorrhagic cerebral infarction among children in the literature; none describes diagnostic MRI findings. MRI is important in these cases, because it may reveal delayed infarction from small-vessel injury, which is not apparent on CT. This article discusses the etiology of and the diagnostic evaluation of pediatric cerebrovascular accidents and suggests the need for emergency physicians to consider trauma as a potential cause of delayed nonhemorrhagic cerebral infarct in children.

*Acad. Emerg. Med.* 1995; 2:523-526.

Received: March 8, 1994

Revision received: June 14, 1994

Accepted: June 16, 1994

**Acknowledgments:** The authors acknowledge the assistance of James Stone, MD, Department of Neurosurgery, and Ms. Velma Richmond, Department of Emergency Medicine, Cook County Hospital; and Arthur M. Heath, MD, and Joseph Porada, MD, Department of Radiology, Our Lady of the Resurrection Medical Center, Chicago, IL.

Address for correspondence and reprints:

Edward P. Sloan, MD, MPH  
Department of Emergency Medicine  
University of Illinois College of  
Medicine, Chicago  
Mail Code 724  
1819 West Polk Street  
Chicago, IL 60612

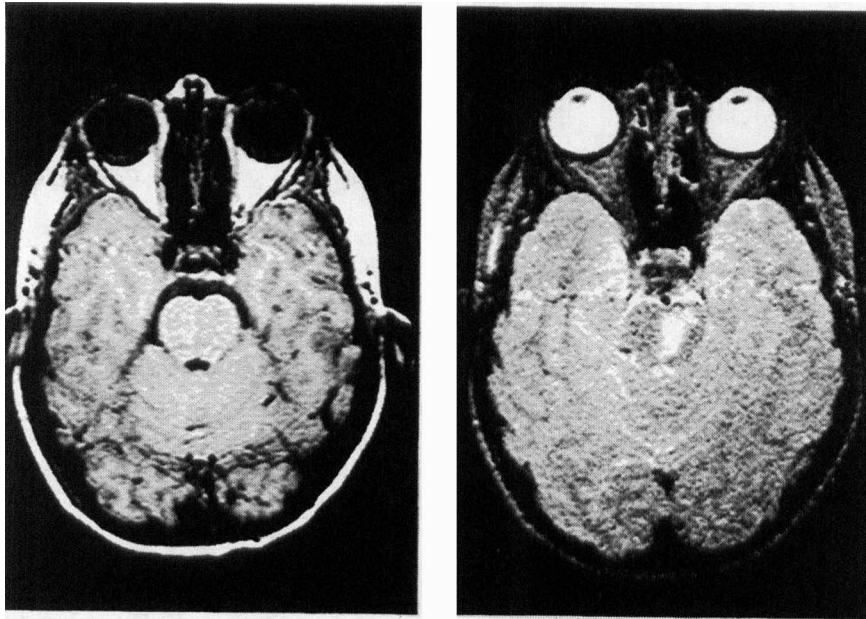
**Key words:** pediatric; head trauma; cerebrovascular accident; hemiparesis; infarction; magnetic resonance imaging

■ Serious sequelae from minor head injuries in children occur infrequently. Nonhemorrhagic strokes in children related to minor trauma have rarely been reported.<sup>1-5</sup> A case is described of hemiparesis developing after minor head trauma in a 9-year-old boy. Magnetic resonance imaging (MRI) showed a nonhemorrhagic lesion in the left pons in a vascular distribution suggestive of infarction.

### ■ CASE REPORT

A healthy 9-year-old right-handed white boy fell while riding his bicycle on a gravel surface. He landed on the right side of his face and had no loss of consciousness. The fall was witnessed by two siblings. The boy was seen about 30 minutes later by his mother, who said that he was alert but "dazed" and could not recall the accident. The patient complained of headache that evening and for three days after the injury. He had normal speech, gait, motor function, and mental status. There was no other concussion symptom such as nausea, vomiting, or visual changes.

Ten days after the injury, the patient was noted to be walking "like he was drunk" and to have difficulty talking. His handwriting was illegible. The mother brought the child to the pediatrician, stating that the child had been a "straight-A" student, an athlete, and normally very talkative. The patient was alert and had no headache,



■ **FIGURE 1.** The initial magnetic resonance imaging scan ten days postinjury shows isodense pons tissue on the T<sub>1</sub> scan (left) and a hyperdense lesion in the left pons on the T<sub>2</sub> scan (right).

nausea or vomiting. The pediatrician ordered plain head computed tomography (CT). The radiologist interpreted the scan as negative.

The patient presented to the ED two days later because of persistent symptoms. Although still having difficulty with gait and speech, he denied the presence of headache or visual changes. The patient had no history of upper respiratory infection, febrile illness, drug ingestion, recent immunization, or other trauma. He had no known medical or developmental illness and no known medication or allergy.

On physical examination, vital signs were normal, as well as the examination of the lungs, heart, abdomen, and extremities. There were resolving ecchymosis and abrasions over the right malar eminence. The skull and facial bones were nontender and the neck was supple. The pupils were equal and reactive to light bilaterally. Funduscopy was normal with sharp disk margins and no retinal hemorrhages. No hemotympanum was noted. The patient was alert and oriented fully. His comprehension was excellent, but his speech was notably sparse. The examination of the cranial nerves was normal. The motor examination revealed 4/5 strength of the upper and lower right extremities. The sensory examination was normal for light touch, two-point discrimination, proprioception, graphesthesia, and stereognosis. The patient had slightly increased upper and lower deep tendon reflexes on the right, as well as a positive right-sided Babinski's reflex. Ataxia was noted with finger-to-nose and heel-to-shin movements on the right.

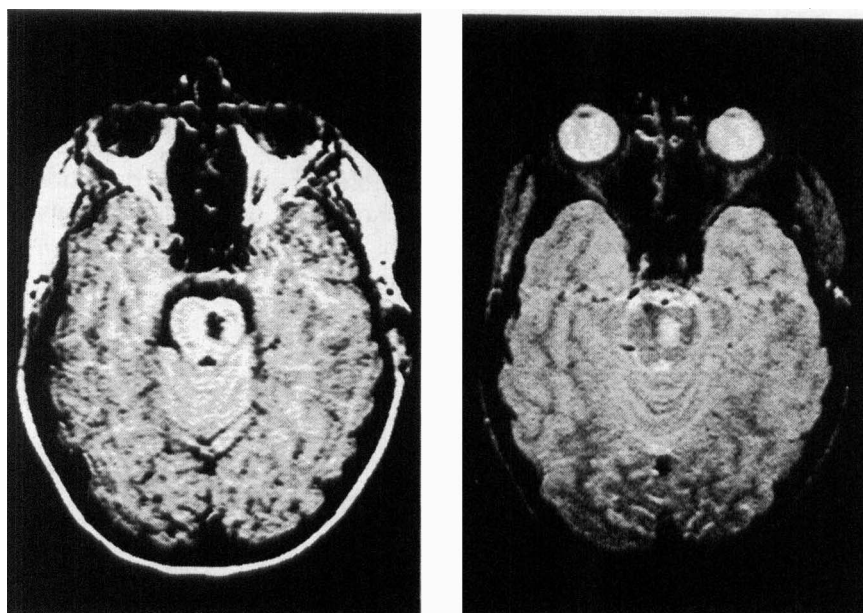
The patient was admitted to the pediatric service

with a diagnosis of right hemiparesis, etiology unclear, with consultation by pediatric neurology. Baseline chemistry and hematology tests were normal. No leukocytosis was noted.

The T<sub>1</sub> MRI showed an isodense pons, while the T<sub>2</sub> MRI revealed a solitary hyperdense lesion of the medial left pons (Fig. 1). These findings are consistent with a nonhemorrhagic infarct in the distribution of the paramedian branch of the basilar artery. This finding prompted a search for known causes of cerebral infarcts in children, including collagen vascular disease, infection, metabolic disorders, thromboembolic phenomena, vascular abnormalities, and demyelinating disease.

The lumbar puncture opening pressure was 18 cm H<sub>2</sub>O, and the cerebrospinal fluid (CSF) was clear, with a glucose of 3.3 mmol/L (59 mg/dL), a protein of 0.33 g/L (33 mg/dL), and no-WBCs or RBCs. The CSF Gram stain and acid-fast stains were negative, as were the cultures for aerobes, anaerobes, Mycobacterium, and fungus. The CSF VDRL and counterimmunoelectrophoresis (CIE) tests for *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitidis* were normal. The cryptococcus latex agglutination test was negative.

The Westergren sedimentation rate was 4 mm/hr. Serum collagen vascular tests, including lupus anticoagulant, rheumatoid factor, cardiolipin antibody, and streptolysin O titer were negative. Serum VDRL and Lyme disease serology tests were negative. A lipid profile, hemoglobin electrophoresis, and 24-hour urine for homocysteine were normal.



■ **FIGURE 2.** The repeat magnetic resonance imaging scan four months postinjury shows a hypodense pons lesion on the T<sub>1</sub> scan (*left*) and a resolving hyperdense lesion on the T<sub>2</sub> scan (*right*).

Echocardiography showed no valvular or left ventricular abnormality. Cerebral angiography was normal without any sign of arteriovenous malformation, aneurysm, vasculitis, or aberrant anatomy. As is normally the case, the branch arteries from the basilar artery to the pons, the area of infarction seen on the MRI, were too small to be visualized by angiography.

The patient improved over his eight-day hospital course without therapeutic intervention. At discharge he had a slightly ataxic gait with full motor recovery. One month after discharge the patient had an entirely normal neurologic examination. Four months after the injury, the repeat T<sub>1</sub> MRI showed a hypodense lesion, and the T<sub>2</sub> MRI a resolving hyperdense lesion in the same location in the left pons (Fig. 2). These findings are consistent with a resolving nonhemorrhagic infarct.

## ■ DISCUSSION

Up to 80% of head injuries in children are defined as mild, with a Glasgow Coma Scale (GCS) score >12, loss of consciousness <20 minutes, and postconcussion symptoms that last <48 hours.<sup>6</sup> Minor head trauma has uncommonly been associated with serious injuries. Children who sustain minor head trauma have developed transient blindness,<sup>7,8</sup> transient cerebellar ataxia,<sup>9</sup> and the syndrome of ischemic cerebrovascular accidents.<sup>1-5</sup>

Although reported only rarely, direct vascular injury can cause posttraumatic nonhemorrhagic brain injury following head trauma.<sup>1-5</sup> Dharker et al. recently re-

ported a series of 23 children aged 7 years or younger who had acute hemiparesis and hypodense basal ganglia lesions on CT immediately following minor head trauma.<sup>1</sup> Hilton-Jones and Warlow described 60 children and young adults who had cerebral infarction, 22% of which were posttraumatic.<sup>2</sup> In a review of 107 published cases of internal carotid artery occlusion in young adults, 20% had a history of trauma, and six of the 20 (30%) were traumatized >24 hours prior to the onset of symptoms.<sup>3</sup> In a series of 64 children who had vessel occlusion confirmed by angiography, 5% were believed to have had infarcts because of remote antecedent head trauma (>2 days prior to the onset of symptoms).<sup>4</sup> Maki et al. reported seven cases of posttraumatic, isolated basal ganglia infarct, four of which (60%) followed minor head trauma.<sup>5</sup> The authors hypothesized that a perforating branch of the middle cerebral artery was injured via a stretching force.

The child discussed in this case report had a lesion of the left medial pons, which corresponds to the vascular distribution of the left paramedian branch of the basilar artery. Infarction of this branch causes contralateral hemiplegia or hemiparesis, often with ataxia.<sup>10</sup> It is hypothesized that the shearing or stretching force caused intimal injury with delayed intimal dissection and infarction.

Other possible etiologies of childhood infarct were considered and excluded for this patient. The vasculitides associated with homocystinuria and collagen vascular diseases were ruled out. Infectious etiologies, such as neurosyphilis, Lyme disease, encephalitis, and men-

ingitis, also were excluded. Cyanotic congenital and rheumatic valvular heart disease, which predispose an individual to embolic phenomena, were not detected. Hypercoagulable states, such as sickle cell anemia, lipid disorders, and lymphoproliferative malignancies, which make thrombosis more likely, were not found. No vascular abnormality, such as an arteriovenous malformation or aneurysm, was seen. Additionally, moyamoya occlusive disease of the intracerebral carotid circulation, a cerebral angiography finding related to some childhood cerebrovascular accidents, was not seen.<sup>11</sup> Demyelinating disease, such as multiple sclerosis and Schilder's disease, which may manifest as neurologic deficits and nonhemorrhagic lesions on CT or MRI, were not likely, because MRI reveals multiple lesions for over 80% of patients.<sup>12,13</sup>

Another possibility that must be considered is whether this patient had an idiopathic nonhemorrhagic infarct. Whether such an entity exists in children has been considered.<sup>14-19</sup> However, the number of patients in any series with nonhemorrhagic brain infarct who have been found to have no risk factor varies inversely with the intensity of investigation.<sup>14-19</sup> A study of 54 pediatric patients with ischemic strokes found 15 thought to have no risk factor or laboratory abnormality. However, minor head injury history beyond the few hours preceding the onset of neurologic deficit was not addressed. Notably, ten patients in the series had a history of preceding minor head injury in the several hours preceding the event.<sup>17</sup> In two case series of 14 and eight children who had "idiopathic infarct,"<sup>14,15</sup> the question of previous minor head injury was not addressed.

## CONCLUSION

The patient in this case report developed delayed hemiparesis following minor head trauma. A single nonhemorrhagic infarct in the pons was detected via MRI. An exhaustive search for other known causes of childhood stroke determined no other likely etiology. Although a causal relationship cannot be proven, the brain stem infarct in this child was most likely due to the antecedent minor head trauma. Although an idiopathic cerebrovascular accident could have caused the fall (and the head trauma), the fact that our patient did not have neurologic findings immediately following the head injury makes this possibility remote. Additionally, whether the entity of "idiopathic brain infarct" occurs in children is controversial.

This case underscores the need to inquire about prior

minor head injury in children with neurologic deficits. Magnetic resonance imaging should be considered for these children when CT evaluation is nondiagnostic. Computed tomography should be done prior to MRI, because CT is readily available in the ED setting and is more sensitive in the detection of acute intracranial hemorrhage. Last, this case supports the use of head injury instructions for all children with minor head trauma.

## REFERENCES

1. Dharker SR, Mittal RS, Bhargava N. Ischemic lesions in basal ganglia in children after minor head injury. *Neurosurgery*. 1993; 33:863-5.
2. Hilton-Jones D, Warlow CP. The causes of stroke in the young. *J Neurol*. 1985; 232:137-43.
3. Humphrey JG, Newton TH. Internal carotid artery occlusion in young adults. *Brain*. 1960; 83:565-78.
4. Hindfelt B, Nitron O. Brain infarction in young adults. *Acta Neurol Scand*. 1977; 55:145-57.
5. Maki Y, Akimoto H, Enomoto T. Injuries of basal ganglia following head trauma in children. *Child's Brain*. 1980; 7:113-23.
6. Miller JD. Minor, moderate, and severe head injury. *Neurosurg Rev*. 1988; 9:135-9.
7. Slayer EJ. Does mild mean minor? Recovery after closed head injury. *J Adolesc Health Care*. 1989; 10:237-40.
8. Yamamoto LG, Bart RD. Transient blindness following mild head trauma. *Clin Pediatr*. 1988; 27:479-83.
9. Mandel S. Minor head injury may not be "minor." *Postgrad Med*. 1989; 85:213-25.
10. Carpenter MB. *Core Text of Neuroanatomy*. Baltimore: Williams and Wilkins, 1978, pp. 330-2.
11. Schoenberg BS, Mellinger JF, Schoenberg DG. Cerebrovascular disease in infants and children: a study of incidence, clinical features, and survival. *Neurology*. 1978; 28:763-8.
12. Stewart JM, Houser OW, Baker HL, et al. Magnetic resonance imaging and clinical relationships in multiple sclerosis. *Mayo Clin Proc*. 1987; 62:174-84.
13. Gebarski SS, Gabrielsen TO, Gilman S, et al. The initial diagnosis of multiple sclerosis: clinical impact of magnetic resonance imaging. *Ann Neurol*. 1985; 17:469-74.
14. Curless RG. Idiopathic ischemic infarction of the brain stem in children. *Child's Nerv Syst*. 1991; 7:305-8.
15. Kappelle LJ, Willemsse J, Ramos LMP, et al. Ischaemic stroke in the basal ganglia and internal capsule in childhood. *Brain Devel*. 1989; 11:283-92.
16. Powell FC, Hanigan WC, McCluney KW. Subcortical infarction in children. *Stroke*. 1994; 25:117-21.
17. Satoh S, Shirane R, Yoshimoto T. Clinical survey of ischemic cerebrovascular disease in children in a district of Japan. *Stroke*. 1991; 22:586-9.
18. Trescher WM. Ischemic stroke syndromes in childhood. *Pediatr Ann*. 1992; 21:374-83.
19. Luchini P, Guida F, Paola F. Vertebrobasilar infarcts in infancy: a case report and review of the literature. *Child's Nerv Syst*. 1991; 8:426-9.