Cerebellar Infarction in the Young

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Background and Purpose: Ischemic cerebrovascular disease in children and young adults usually affects the anterior circulation.

Summary of Report: We describe two cases of cerebellar infarction in the territory of vertebral artery supply, associated with physical exertion, in a young adult and in a child. Review of 31 previous cases of cerebellar infarction occurring in the first 2 decades of life demonstrated a mostly obscure causation; where a likely cause was found, trauma was most frequent. In 12 of the 31 patients, a vertebral artery (usually the left) was occluded. Patients were sometimes predisposed to such occlusions by subluxation between the first and second cervical vertebrae, allowing abnormal neck movements that can cause arterial injury and thromboembolism. Some of these cerebellar infaracts, like those of our patients, have followed physical exertion.

Conclusions: Cerebellar infarction can be life-threatening, but half of the patients, including ours, have had complete or near-complete recovery. (Stroke 1992;23:763–766)

KEY WORDS • cerebellar infarction • child • young adults

Cerebellar infarction in adults is usually caused by atherosclerotic occlusion of an intracranial vertebral artery or by embolism to an intracranial vertebral artery from the heart or from an occluded extracranial vertebral artery.1–2 Ischemic cerebrovascular disease in children and young adults affects the anterior circulation much more often than the vertebrobasilar system.3 Posterior circulation strokes sometimes occur in the young, but the cause is often not determined, even after extensive evaluation.4–6 In adults, the vast majority of ischemic anterior and posterior circulation strokes occur at rest or during ordinary daily activities; onset during or after exertion is rare. Involvement of the posterior circulation in young patients is exemplified by lateral medullary ischemia reported after vigorous outdoor activity.3 We now describe major cerebellar infarction in relation to exercise in two previously healthy young patients with normal vertebral angiography and no known systemic disease or vascular risk factors.

Case Reports

Patient 1

A 9-year-old boy was well until 6/10/86 when, while riding a bicycle downhill, he tried to dodge a frolicking puppy. He fell from the bicycle but did not hit his head or neck. The next morning he awoke crying, with a severe pounding midoccipital headache. He vomited, and the headache subsided. The headache returned on 7/21/86, when he awakened with sharp, jabbing pain in the right frontal region; he felt dizzy and again became uncoordinated. He was readmitted to the Dartmouth–Hitchcock Medical Center, where examination showed slight dysarthria, a slightly smaller left pupil, and bilateral limb and gait ataxia. A repeat contrast-enhanced cranial CT scan on 7/21/86 showed nonenhancing bilateral cerebellar hemisphere infarcts; the right-sided lesion was larger than the month before (Figure 1). A week later, the right cerebellar infarct enhanced after contrast infusion, indicating recent ischemia. Repeat vertebral angiography on 7/24/86 was normal except for kinking of the upper cervical portion of the right vertebral artery. Transthoracic echocardiography was normal except for slight mitral valve prolapse. Coagulation, lipid, and metabolic studies were normal. He was treated with heparin, then warfarin (Coumadin, Du Pont Pharmaceuticals), and was discharged home on Coumadin on 7/29/86. One day in mid-August 1986, while running, he felt a “buzzing in the head” and fell to the ground, but rose quickly and felt fine afterward. On 9/19/86, his examination was normal except for slight limb ataxia, upper more than lower, and left more than...
right. Coumadin was stopped and daily aspirin (80 mg/day) was prescribed. Since then, he has done well on aspirin 80 mg/day, with no return of symptoms. He continues to be very active, with occasional headache at times of intercurrent illnesses. The mother sometimes has sinus headaches; other family members do not have headaches.

**Patient 2**

A 28-year-old athletic junior executive played five games of competitive softball on 6/22/86 without injury or incident. That night he was exhausted and went to bed with a headache. The next day, he had a severe headache over the vertex and behind his right eye, nausea, vomiting, spinning dizziness, and, later, transient tingling of his left face and arm. He was sent home from a local hospital after a normal examination and non-contrast-enhanced head CT scan. The next morning, he was difficult to arouse. A repeat noncontrast head CT scan showed acute hydrocephalus and effacement of the fourth ventricle. He was transferred to the New England Medical Center.

On admission, his blood pressure was 130/70 mm Hg, pulse was 80 beats per minute, and temperature was 38.6°C. He could be alerted temporarily with persistent prodding but gave his location as “mother’s house.” Speech was dysarthric, but language was normal. He held his head stiffly, slightly tilted to the right. He had bilateral ptosis; coarse horizontal nystagmus, greater to the right; and ocular skewing, with the right eye above the left. There was slight right peripheral facial weakness and dysmetria on finger-to-nose testing, especially on the right. Gait was ataxic. He had brisk deep-tendon reflexes and bilateral extensor plantar responses. He became more difficult to arouse under observation. Intravenous dexamethasone was given, and an external ventricular drain was placed into the frontal horn of the right lateral ventricle. Ten ml cerebrospinal fluid under high pressure was removed; it contained 280/mm³ red blood cells, 2/mm³ white blood cells, and 10 mg/dl protein. The patient became more alert within 30 minutes.

An enhanced CT scan of the posterior fossa on 6/25/86 showed a large area of low density in the right cerebellum and a small hypodense area in the paramedian left cerebellum. Ventricular size was decreased but not to normal. Bilateral vertebral arteriography was normal. The posterior inferior cerebellar arteries on both sides filled well. An avascular mass in the right cerebellum and cerebellar tonsillar herniation were present. Electrocardiogram and transthoracic echocardiogram were normal. Hemoglobin was 16.7 g/dl, and hematocrit was 51%. Blood chemistries and lipid and coagulation profiles were normal. The ventricular drain was removed on 6/28/86 without problem. A cranial magnetic resonance imaging scan done on 6/30/86 showed a large right lateral cerebellar high-signal lesion (Figure 2) and a smaller high-signal lesion in the left cerebellum. He continued to improve neurologically. At discharge, on 7/7/86, he had slight neck stiffness, right finger-to-nose dysmetria, and gait ataxia. One month later, examination was normal. Five months later, while bowling, he developed a sudden “light glare” in the center of vision lasting 20 minutes, without accompanying headache. He vaguely recalled similar episodes in...
his teens. The next month, after playing basketball, he had 20 minutes of "zig-zag" colored visual spectra in his left eye, with accompanying headache. In the 4½ years since his stroke, he has had a total of five classic migraine attacks but no other vascular events.

**Discussion**

Cerebellar infarcts cause symptoms and signs identical to cerebellar hemorrhages. Gait ataxia and vomiting are the two cardinal findings. Veering, leaning, toppling, and difficulty standing or sitting erect are also common. "Classical cerebellar signs," such as limb incoordination and dysmetria, are absent in cerebellar infarction, were present in both our patients. When cerebellar infarcts swell, a finding in our experience more common in young patients, mass effects develop. In the posterior fossa, this can be life threatening because of compression of the brain stem and fourth ventricle. Although acute cerebellar infarcts may not always image as low-density lesions on cranial CT scans, obliteration of posterior fossa cisterns (producing a "tight posterior fossa"), should lead to consideration of that diagnosis. When cerebellar infarction is complicated by obstructive hydrocephalus, as in our second patient, ventricular drainage may be a life-saving measure.

Brain infarcts caused by atherosclerosis usually develop upon awakening or during activities of daily living, but seldom during active exercise. Brain ischemia and infarction after vigorous exercise have been described in young people, some with migraine. Fisher described three examples: a 29-year-old man who had severe headache and left hemiplegia after trying to save a drowning boy (cerebellar angiography was normal); a 17-year-old boy who developed global aphasia after a 225-yard sprint; and a 16-year-old boy with previous headaches who developed right hemiparesis and aphasia after pushing a stalled car.

Although brain symptoms may be noted after exertion, the causative vascular injury probably occurs during physical activity. Proposed likely mechanisms of vertebral artery pathology are traumatic vertebral dissection; positional compromise of a vertebral artery with local thrombus formation and subsequent embolization of clot; and vasoconstriction, often with superimposed thrombosis. Congenital anomalies of the neck and hypoplasia of one vertebral artery or a posterior inferior cerebellar artery might predispose an individual to compression and thrombosis.

The vertebral arteries are common sites for congenital anatomic variations and anomalies. Often, one vertebral artery is hypoplastic in the neck or in its distal intracranial portion, when it is said to "end in posterior inferior cerebellar artery." Cerebellar or lateral medullary infarction in such patients during exercise might be explained by a mismatch between increased need for blood flow because of increased metabolic activity related to exercise and limited blood flow because of the hypoplasia. Small arteries are also more likely to occlude, especially in the presence of dehydration or increased coagulability. Our two patients, however, had normal vertebral artery anatomy.

For unknown reasons, intracranial posterior circulation arteries have a susceptibility to vasoconstriction. This is seen particularly in classic migraine, a disorder known to be associated with brain ischemia that typically begins in the posterior part of the brain, then spreads anteriorly, with prominent visual and other sensory symptoms referable to structures supplied by the posterior cerebral arteries. Basilar migraine presents with symptoms related to vasoconstriction of the basilar, vertebral, or posterior cerebral arteries, and is more common in adolescents. Stroke is an uncommon complication of childhood migraine but occasionally has been reported to complicate basilar artery migraine. Caplan has recently reported brain stem and cerebellar infarction in nine patients with migraine; one was a 6-year-old boy. Migraine is often precipitated by or follows exertion or sudden change in activity, and both of our patients had exercised (bicycle riding, softball) and the next day developed headache, then stroke. Patient 2 later developed classic migraine, with aura first noted months after the stroke. We postulate that the most likely explanation for cerebellar infarction in our patients was vasoconstriction. Dehydration may have also played a role in the illness of the second patient.

Cerebellar infarcts do occur in young adults and children, sometimes in relation to head and neck trauma and physical activity, as in our two patients. Except for two deaths reported by Fowler, all of the patients reported have survived their illnesses. Surprisingly, approximately half have either completely recovered or were left with only slight disability. We believe most previously unexplained examples of cerebellar infarction that occur in relation to exercise in the young are caused by dissection or vasoconstriction.

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**References**

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