Cerebellar Concussion
—Three Case Reports—

Hiroshi FUMEYA and Hiroshi HIDESHIMA
Division of Neurosurgery, Hideshima Hospital, Musashino, Tokyo

Abstract
Three patients with transient cerebellar dysfunction following head injury are described. Acute cerebellar signs, such as ataxia, nystagmus, and dysarthria, occurred just after trauma and resolved gradually, disappearing in every patient. Cerebrospinal fluid and computed tomography examinations were normal but magnetic resonance imaging and single photon emission computed tomography revealed cerebellar lesions. These findings distinguish cerebellar concussion from cerebellar contusion and suggest that the synergistic effect of trauma and ischemia may be the pathophysiological basis of this unusual syndrome.

Key words: cerebellum, concussion, head injury

Introduction
Cerebellar trauma often causes coma because an impact hard enough to injure the cerebellum, which is surrounded by compact bone structures, usually also damages the brainstem and the cerebrum. However, acute cerebellar symptoms may occur with intact consciousness and resolve rapidly and completely. Such cases are classified as concussion injuries of the cerebellum, but comparatively little is known about them, due to their infrequency.\textsuperscript{4,5,11} Cerebellar concussion is an appropriate term according to the definition of concussion of the International Neurotraumatology Committee.

We describe three cases of cerebellar trauma with associated lesions demonstrated by magnetic resonance (MR) imaging and single photon emission computed tomography (SPECT).

Case Reports
Case 1: A 49-year-old male fell down stairs while drunk, striking his left frontal region without loss of consciousness, but found himself grossly unsteady on standing up. Neurological examination disclosed coarse, frequent lateral gaze nystagmus, scanning speech, and marked titubation of the trunk. He could not stand with feet together with eyes open.

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Coordination tests revealed dysmetria and dysdiadochokinesia of the right hand. Lumbar puncture disclosed an opening pressure of 150 mmH\textsubscript{2}O of clear colorless fluid. T\textsubscript{2}-weighted MR imaging (repetition time [TR]/echo time [TE]/excitation 3000 msec/120 msec/1) showed a high-intensity area in the right superior cerebellar peduncle, although the CT scan was normal (Fig. 1A, B).

During the first 48 hours after admission, his dysarthric speech progressively resolved. Horizontal nystagmus largely disappeared on the 4th hospital day. The Romberg test was normal 10 days after trauma and he could perform tandem-walking in a straight line very well. MR imaging showed disappearance of the lesion (Fig. 1C).

Case 2: A 78-year-old male was hit by a motorcycle but suffered no loss of consciousness. He complained of vertigo and could not sit in a chair without support. Neurological examination revealed fine horizontal nystagmus, dysmetria of the right hand, and truncal ataxia. Plain skull x-ray films showed a linear occipital fracture overlying the inion. MR imaging revealed a high-intensity area in the right cerebellar hemisphere, but the CT scan was normal (Fig. 2A, B). \textsuperscript{123}I-iodoamphetamine SPECT demonstrated decreased regional cerebral blood flow (rCBF) in the lesion (Fig. 2C). Lumbar puncture showed an opening pressure of 130 mmH\textsubscript{2}O of clear watery fluid.

Nystagmus and dysmetria completely disappeared within 48 hours of trauma. He could walk with an
Figure 1 Case 1. A: T2-weighted MR image (TR/TE 3000/120 msec) on admission showing a high-intensity area in the right superior cerebellar peduncle. B: CT scan on admission showing no abnormality. C: T2-weighted MR image (3000/120 msec) 2 weeks after trauma showing disappearance of the lesion.

Figure 2 Case 2. A: T2-weighted MR image (TR/TE 2000/100 msec) on admission showing a high-intensity area in the right cerebellar hemisphere. B: CT scan on admission showing no cerebellar lesion. C: 123I-iodoamphetamine SPECT scan on admission disclosing decreased rCBF in the lesion. D: T2-weighted MR image (2000/100 msec) 10 days after trauma showing disappearance of the lesion. E: SPECT scan 2 weeks after trauma showing improved rCBF.
ataxic gait 1 week after injury. Follow-up MR imaging and SPECT showed that the signal abnormality and the impaired rCBF had improved in parallel with the clinical recovery (Fig. 2D, E). He was asymptomatic after 3 weeks.

Case 3: A 18-year-old male was involved in a motorcycle accident with a truck, losing consciousness for 5 minutes. Neurological examination revealed coarse, frequent lateral gaze nystagmus, and explosive speech. Lumbar puncture disclosed an opening pressure of 200 mmH2O of clear colorless fluid. Plain skull x-ray films showed a fracture of the right petrous pyramid. MR imaging revealed a high-intensity area in the right cerebellar hemisphere, whereas CT showed no traumatic lesion (Fig. 3A, B). 123I-iodoamphetamine SPECT demonstrated decreased rCBF in the lesion (Fig. 3C).

Nystagmus disappeared 12 days after injury and slurred speech resolved 1 month later. MR imaging showed that the lesion had disappeared completely (Fig. 3D).

Discussion

Cerebellar concussion is not a definite entity. However, a syndrome consisting of transient cerebellar dysfunction following head trauma has been reported in the literature. Cantu described two patients with clear cerebrospinal fluid who developed dysarthria, truncal ataxia, and rotatory nystagmus after occipital head trauma. He regarded the transient dysfunction as midline cerebellar concussion. Gerstenbrand et al. reported that three head injury patients showed transitory cerebellar symptoms of the hemispheric type. Nakamura described seven patients with post-traumatic cerebellar dysfunction, but found that CT revealed no lesions because cerebellar concussion caused the syndrome. These authors concluded that clear cerebrospinal fluid and complete recovery distinguish cerebellar concussion from contusion. In our patients, the normal CT scan and the clinical features indicated concussion rather than contusion.

Sudden neuronal depolarization, followed by secondary cerebral vasoconstriction, evokes the transient neurological dysfunction characteristic of cerebral concussion. Neuronal mitochondrial swelling and extracellular edema are observed in rat concussion models. CT does not demonstrate the local edema associated with concussion, but T2-weighted MR imaging reveals it as a high-intensity area and SPECT as a region of decreased CBF. The MR imaging and SPECT findings in our patients were compatible with local edema and ischemia, so cerebellar concussion may be pathophysiologically analogous to cerebral concussion.

Recent studies show that CBF is reduced soon after head injury. General hypotension, catecholamine surge, and disturbance of autoregulation are thought to be responsible for the changes in global or hemispheric flow, but are unlikely to evoke the changes in regional flow seen in our patients. Possible causes include cerebral vasospasm and decreased metabolic demand. Peripheral arterial spasm may reduce regional flow and propagate local edema. Posttraumatic vasospasm usually occurs in patients with subarachnoid hemorrhage, but transcranial Doppler ultrasound monitor can detect vasospasm in patients with no CT evidence of hemorrhage. Although our patients demonstrated clear cerebrospinal fluid, arterial vasospasm may have occurred and resulted in cerebellar ischemia.

Follow-up MR imaging and SPECT showed that...
neither trauma nor ischemia was so severe as to cause persistent abnormality. Unless cerebellar contusion or infarction develops, patients are unlikely to be affected for a long time. Nevertheless, symptoms lasted for several weeks in our patients. This implies the synergistic effect of the two mild insults. Traumatized brain is very vulnerable to ischemia, so mild concussive brain trauma, associated with a simultaneous ischemic insult, can induce prolonged functional impairment. Our findings suggest that the combination of mild injury and simultaneous local ischemia may cause cerebellar concussion to become symptomatic and delay functional recovery.

References


Address reprint requests to: H. Fumeya, M.D., Division of Neurosurgery, Hideshima Hospital, 3-14-4 Kichijo-ji-minami-cho, Musashino, Tokyo 180, Japan.

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