

# **Delayed, Transient Neurological Deterioration After Mild Head Injury**

## **—Case Report—**

Eiichirou URASAKI, Hideoki YASUKOUCHI, Akira YOKOTA,  
and Yoshihiro ARAGAKI\*

*Department of Neurosurgery, University of Occupational and Environmental Health,  
Kitakyushu, Fukuoka; \*Aragaki Neurosurgical Hospital, Kitakyushu, Fukuoka*

### **Abstract**

**A 16-year-old boy presented with delayed, transient neurological deterioration 18 days after mild head injury. Left hemiparesis and left homonymous hemianopsia appeared after right frontal contusional and mild subdural hematomas subsided. Neuroimaging examinations including cerebral angiography, magnetic resonance imaging, and single photon emission computed tomography showed vasodilation and hyperemia in the right cerebral hemisphere. The present case is not typical of acute “juvenile head trauma syndrome,” but may represent a possible pathophysiology of the delayed type of transient neurological deterioration after mild head injury.**

Key words: mild head injury, neurological deterioration, juvenile head trauma syndrome, hyperemia, status epilepticus

### **Introduction**

Delayed, transient neurological deterioration characterized by headache, vomiting, clouded consciousness, hemiparesis, cortical blindness, or seizures occasionally occurs after mild head injury in children and adolescents. This syndrome has been called “juvenile head trauma syndrome” (JHTS).<sup>7,8,25,27,29</sup> Almost all of the symptoms characteristically appear within 48 hours, usually within several hours, after craniofacial injury.<sup>7,8,25,27,29</sup> JHTS has recently been classified into acute and delayed types on the basis of whether symptoms appear before or after 48 hours.<sup>27</sup> However, any difference in the pathophysiologies for these types has not been identified.

Traumatic subdural hematoma sometimes causes delayed neurological deterioration more than 10 days after head injury.<sup>1,24</sup> The underlying condition is suspected to be cerebral edema or swelling due to disturbed cortical venous return.<sup>1,24</sup> We describe an interesting case of delayed and transient neurological deterioration after mild head injury associated

with subdural and contusional hematomas, and hemispheric hyperemia.

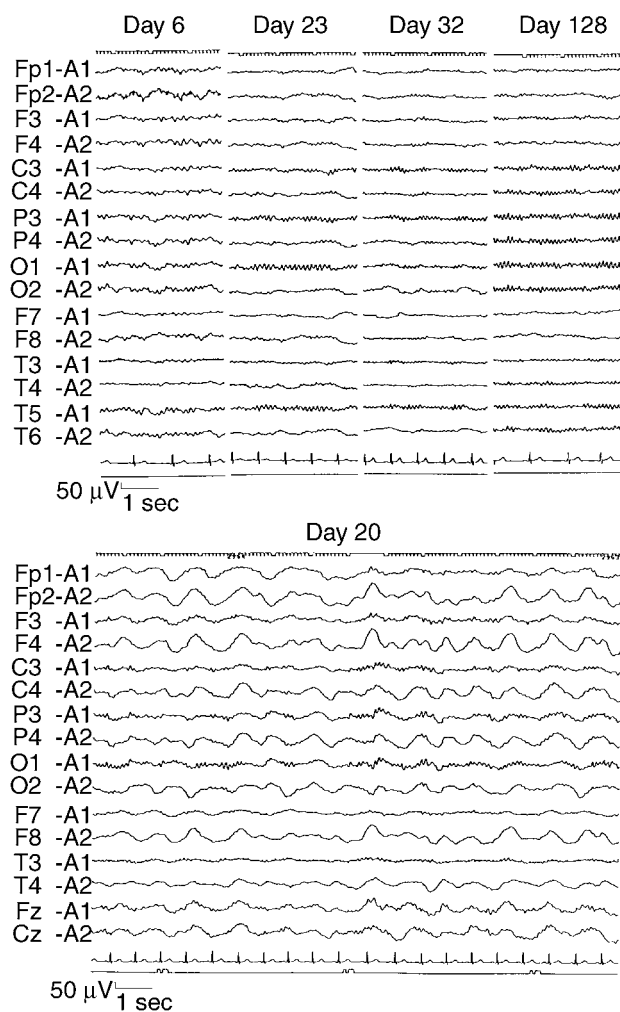
### **Case Report**

A 16-year-old boy, with no past personal or family history of epilepsy or migraine, struck the occipital area of his head on the ground after falling from a 2 m high wall while playing on a school ground (day 0). He did not lose consciousness but was admitted to a nearby hospital 3 hours after the accident because of continuous headache. Neurological examination found no abnormalities except for headache, and his score on the Glasgow Coma Scale (GCS) was the full 15 points. Skull radiography showed no fracture, but computed tomography (CT) detected an equivocal acute subdural hematoma in the right frontal lobe. The severity of the headache increased on the following evening, and he suffered nausea and vomiting. He was transferred to our department on day 3.

On admission, headache and nausea were the only positive neurological symptoms, and his GCS score again was 15. CT showed a right acute subdural hematoma, 5-mm thick, and a contusional hematoma in the right frontal base accompanied by mild

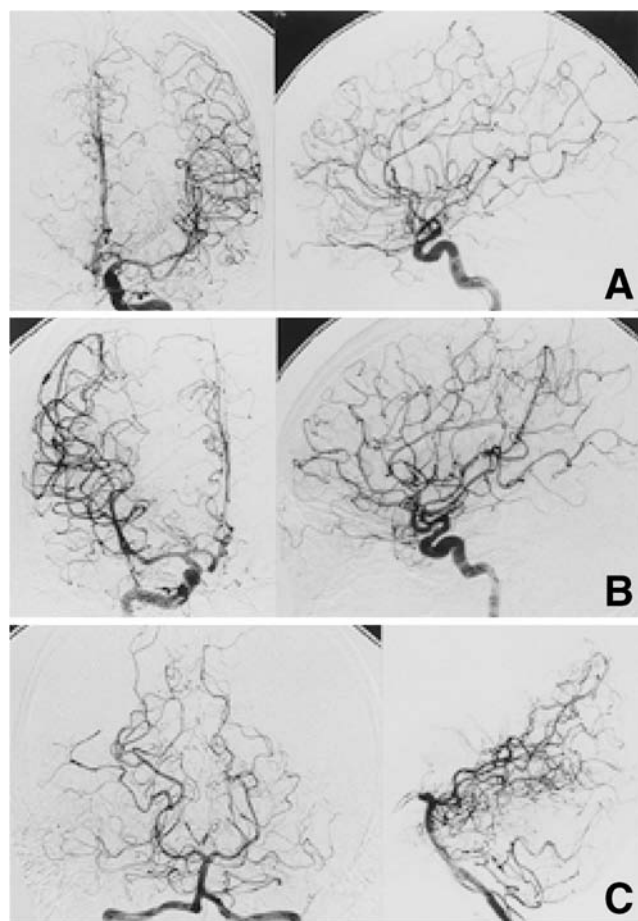
midline shift. Conservative therapy using glycerol, an antiemetic, and an analgesic was given, and his symptoms gradually subsided. Electroencephalography (EEG) on day 6 showed only mild, lazy activities in the right anterior and middle temporal regions (Fig. 1). Follow-up CT (day 12) showed that the subdural and contusional hematomas had almost disappeared, and hospital discharge was planned. His headache sometimes recurred, but was well controlled by oral administration of an analgesic.

He complained of progressive weakness in the left



**Fig. 1** Electroencephalograms recorded on days 6, 23, 32, and 128 (upper row) after head injury and on day 20 (lower row) when right hemiparesis and right homonymous hemianopsia appeared. Delta waves on day 20 and alpha wave suppression on days 23 and 32 are present predominantly in the right cerebral hemisphere. No spikes or sharp wave activities were recorded.

upper extremity on day 18, with positive Barré's sign (4/5). CT showed a post-contusional low density area in the right frontal base, slight right subdural fluid collection, and midline shift. Left hemiparesis including the face (upper 3/5, lower 4/5, face 4/5) became objectively apparent on days 19 and 20. Dysesthesia of the left extremities and left homonymous hemianopsia were also present. Angiography on day 19 showed vasodilation of the M<sub>3</sub> portion and the peripheral branches of the middle cerebral artery on the right as compared with the left (Fig. 2A, B), and vertebral angiography showed dilation of the right posterior cerebral artery trunk and right posterior temporal branch (Fig. 2C). No vasospasm was detected in the proximal or distal portions of the cerebral arteries. The capillary and venous phases



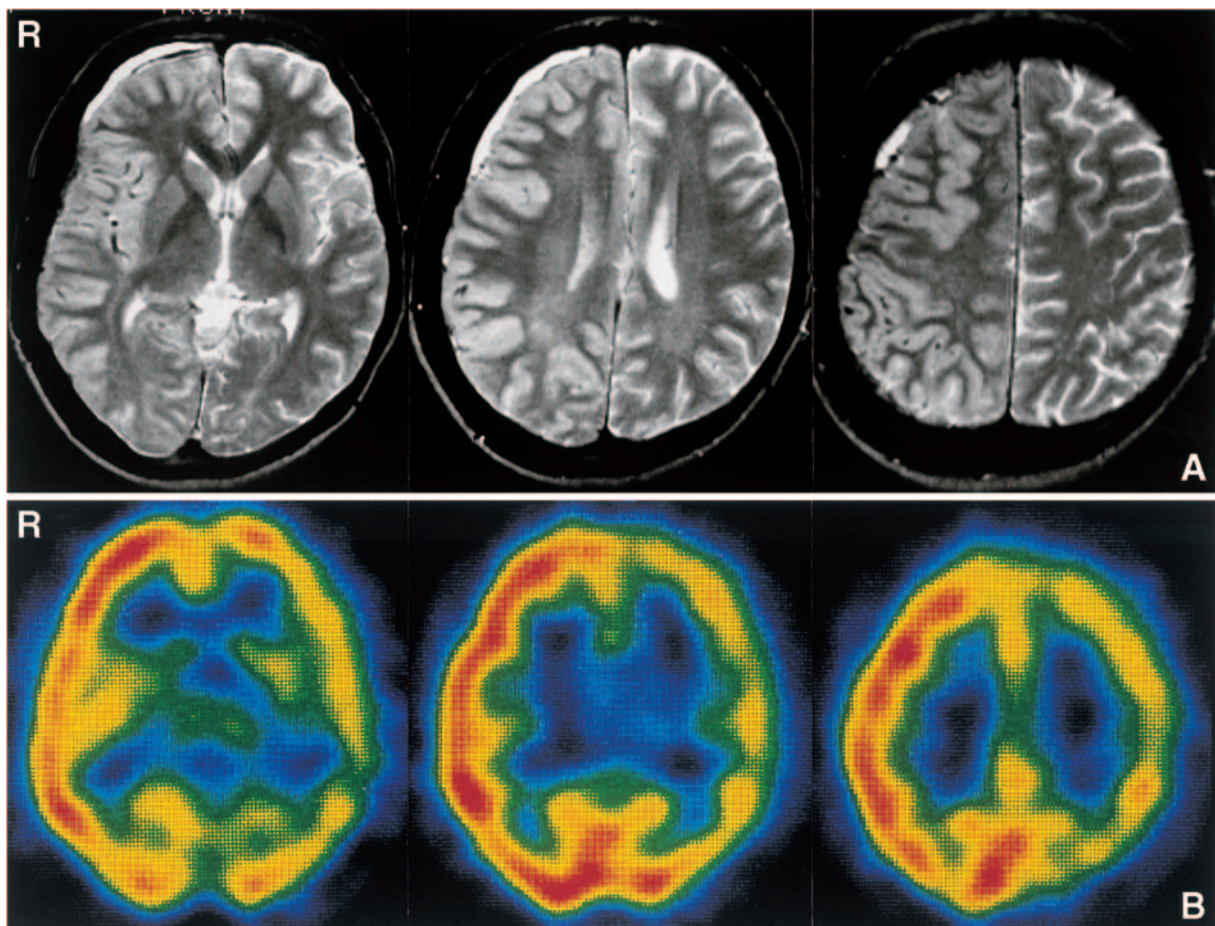
**Fig. 2** Left (A) and right (B) carotid angiograms showing relative vasodilation of the M<sub>3</sub> portion and peripheral branches in the territory of the right middle cerebral artery. Vertebral angiograms (C) showing vasodilation of the right posterior cerebral artery trunk and right posterior temporal branch.

showed a slight increase in capillary brush and slight dilation of the cortical veins in the right cerebral hemisphere, but the cerebral circulation time was almost equal for both hemispheres (not shown). Magnetic resonance (MR) imaging on day 19 showed high intensity, thick cortical ribbons along the gray matter of the right cerebral hemisphere on the  $T_2$ -weighted image (Fig. 3A), which appeared iso-intense on the  $T_1$ -weighted image. Many flow voids were present in the gray matter. These findings were compatible with vasodilation and cortical hyperemia. Hyperemia in the right cerebral convexity was confirmed on day 20 by single photon emission computed tomography (SPECT) with technetium-99m-ethyl-cysteinate-dimer (Fig. 3B). EEG findings on day 20 showed diffuse delta activities, predominantly in the right cerebral hemisphere (Fig. 1), but no spikes or sharp waves were detected. A generalized convulsive seizure occurred on day 21. Oral administration of val-

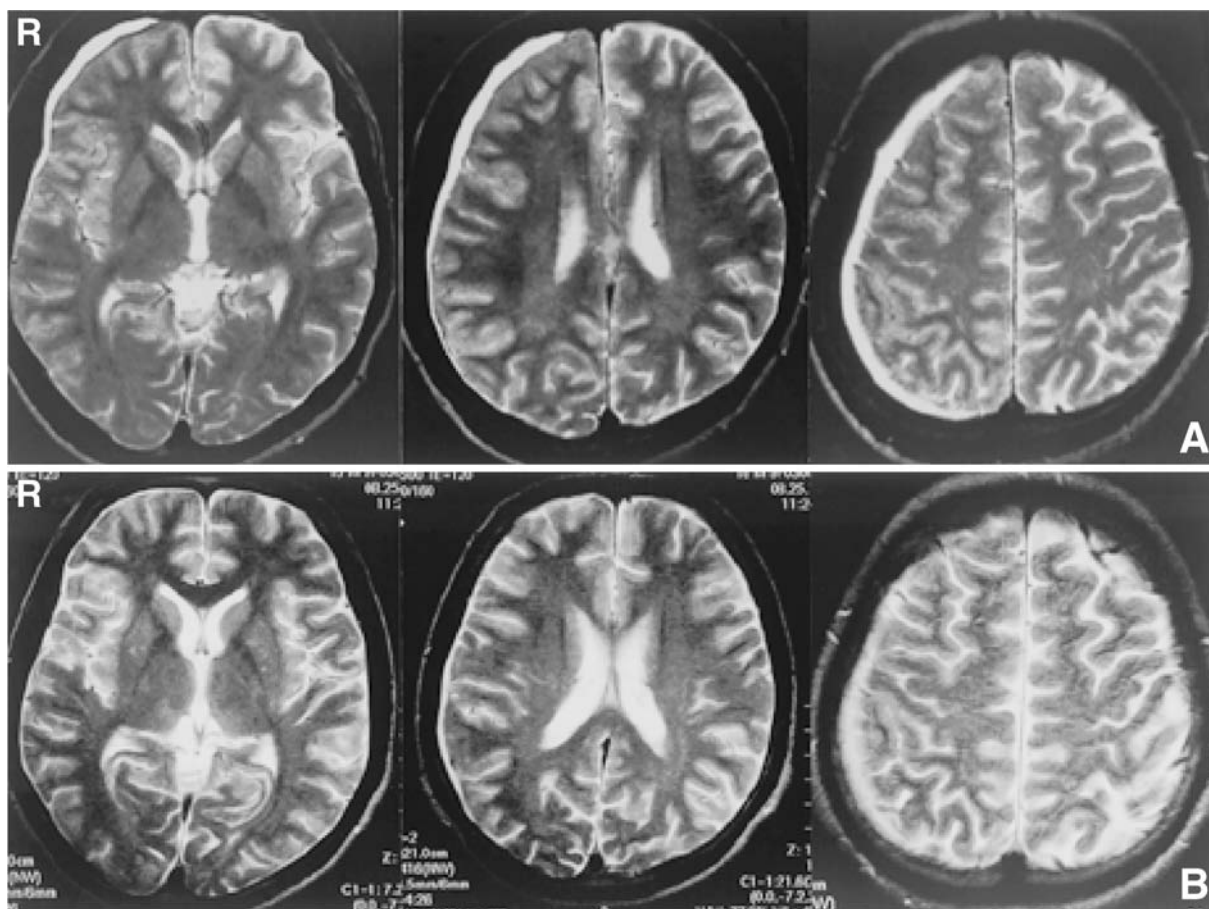
proic acid at 800 mg/day was started after this seizure and was discontinued at discharge (day 33). Transient neurological deterioration continued for a week. The neurological symptoms gradually subsided and disappeared on day 25. EEG on days 23 and 32 showed that the delta wave had disappeared but that the alpha wave was still suppressed in the right centro-parieto-occipital and temporal regions (Fig. 1). Follow-up MR imaging showed residual hyperintense cortical ribbons in the right hemisphere on day 37, and normality on day 91 after injury (Fig. 4). The EEG on day 128 was normal (Fig. 1).

## Discussion

Prolonged ictal paralysis and cortical blindness are occasional forms of nonconvulsive simple partial seizure.<sup>2,3,16,28,32,33</sup> The convulsive seizure that occurred 2 days after the onset of symptoms, and the



**Fig. 3**  $T_2$ -weighted magnetic resonance images on day 19 (A) showing a flow void and hyperemia in the right cerebral hemisphere. Single photon emission computed tomography images (B) showing apparent hyperemia in the right cerebral hemisphere.



**Fig. 4** Follow-up magnetic resonance images on days 37 (A) and 91 (B) showing gradual normalization of hyperemia in the right cerebral hemisphere.

post-traumatic clinical course together with the neuroimaging findings required us to exclude the diagnosis of status epilepticus of the nonconvulsive type.

Cerebral angiography has detected vascular hyperemia, capillary brush, and early draining veins in patients with status epilepticus,<sup>17,21,34</sup> which are compatible with the increased blood flow in SPECT that is generally seen in status epilepticus.<sup>11,16,32</sup> Neuroimaging signs of transient abnormalities in patients with status epilepticus characteristically involve a focal area either in the white matter or in the combined white and gray matter, appearing as low density areas by CT, and as decreased signal intensity on T<sub>1</sub>-weighted images and/or increased signal intensity on T<sub>2</sub>-weighted ones by MR imaging, without a mass effect.<sup>9,13,31</sup> Such changes are thought to be due to vasogenic edema induced by hyperemia. Increased oxygen consumption in the cortical epileptogenic zone and the resulting hypoxia are assumed to lead to an accumulation of CO<sub>2</sub> and lactic acid, which in turn causes vasodilation, loss of autoregulation, and increased cerebral blood

flow.<sup>13,17</sup>

Such neuroradiological findings mentioned above were similar to those of the present case, but no EEG findings to support the diagnosis of epilepsy were obtained. EEG findings of status epilepticus may be normal when the epileptic focus is small<sup>11</sup> or deep in the brain structure. However, the diffuse slow waves in the entire unilateral hemisphere found in our case without epileptic discharges tended to exclude status epilepticus.

Vasospasm was not the direct cause of delayed neurological deterioration in our patient, although hypoperfusion, hyperemia, and vasospasm are well known factors in neurological deterioration after head injury.<sup>5,12,18-20,22,23,26,30</sup> Complete neuroimaging examinations including CT and MR imaging, cerebral angiography, and SPECT during the period of neurological deterioration found no traces of vasospasm.

The time course in this case is unique because, except for occasional headache, the neurological symptoms appeared after a far longer interval than

in previously reported cases of JHTS in which the delay between head injury and the onset of the neurological episodes varied from several seconds, minutes, and hours up to 2 days,<sup>7,8,25,27,29)</sup> and the episodes lasted from several minutes to several days. Presumably, the majority of the acute type of JHTS, especially those at intervals of several seconds or minutes, involve electrical and/or neuronal reflex phenomena such as cortical spreading depression<sup>25,29)</sup> or a trigeminovascular response, because these phenomena appear immediately after the input of stimuli.<sup>7,27)</sup> Our case cannot be classified as the acute type of JHTS.

Vasoconstriction and hypoperfusion may be causes of neurological deterioration, as partial peripheral arterial obliterations were found in two cases of JHTS by cerebral angiography performed on the 7th and 2nd days after onset of the hemiparesis.<sup>8)</sup> Cerebral angiography found no abnormalities in six of 37 patients with JHTS.<sup>25)</sup> In contrast, investigation of migraine suggested that vasodilation and hyperemia are the underlying causes of JHTS, but there was no radiological evidence.<sup>27)</sup> The present case did not fit the profile of typical acute JHTS, but our findings suggest that vasodilation and hyperemia are the baseline conditions for some cases of delayed neurological deterioration after mild head injury.

CT showed mild subdural and contusional hematomas in our patient, but no abnormalities in most reported cases of JHTS.<sup>6,7,25,29)</sup> CT found no pathological abnormalities in nine of 12 cases of JHTS.<sup>27)</sup> Exceptional CT findings in one case with diffuse cerebral swelling and in another with hemispheric swelling after mild head injury were considered to reflect vasodilation and not edema.<sup>29)</sup> JHTS probably includes a variety of pathological states.<sup>29)</sup>

Subdural hematoma is sometimes associated with delayed neurological deterioration 10 or more days after head injury.<sup>1,24)</sup> Cerebral hemispheric swelling and ischemia due to disturbed cortical venous flow may be the causes of this neurological deterioration.<sup>1)</sup> Cerebral angiography in our patient showed no such abnormal findings of the cerebral venous system when the neurological deterioration occurred. SPECT showed decreased cerebral blood flow in a patient with subacute subdural hematoma.<sup>1)</sup> Our patient suffered subacute neurological deterioration similar to these subdural hematoma cases,<sup>1,24)</sup> but harbored different pathological conditions, such as vasodilation and hyperemia.

Disturbed cerebral autoregulation after mild head injury was recently found in 28% of patients with GCS 13–15, who had poor or absent functioning of cerebral autoregulation.<sup>10)</sup> This finding suggests that

hyperemia is involved in mild head injury, although the majority of patients regain normal autoregulation.<sup>7,8,25,27,29)</sup>

Post-traumatic hyperemia can be divided into malignant and benign types depending on the association with intracranial hypertension.<sup>4,12–15,19,23,26,27)</sup> Analysis of intracranial pressure and cerebral blood flow in 59 patients with moderate or severe head injuries showed that the median duration of hyperemia associated with intracranial hypertension was longer (median 6 days, range 1–14 days) than that of hyperemia without intracranial hypertension (median 2.5 days, range 0–5 days).<sup>12)</sup> Our findings indicate that certain cases of mild head injury may represent a more delayed or prolonged hyperemia phase linked to a favorable outcome.

Hyperemia also frequently occurs focally or globally in the unilateral hemisphere, when cortical contusion and laceration or subdural hematoma are present, respectively.<sup>18,26)</sup> Hyperemia in our case was interpreted as the benign hemispheric type induced by metabolic recovery from the damage done by the mild contusional and subdural hematomas. The benign type of hyperemia is considered to be secondarily induced by metabolic demands on the release of metabolic mediators during reestablishment of membrane ionic homeostasis after head injury.<sup>12,26)</sup>

Delayed neurological deterioration after mild head injury involves a variety of pathological causes from early neuronal reflex phenomena to late cerebral edema due to hyperemia or ischemia. The baseline condition may be disturbed autoregulation of cerebral vasoreactivity. The present case can be classified as the delayed type of JHTS, or subacute subdural hematoma, in which neurological deterioration is caused by hyperemia as a result of “overregulation” during the recovery from head injury. Immature autoregulation may be a factor in such delayed neurological deterioration after head trauma.

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*Address reprint requests to:* E. Urasaki, M.D., Department of Neurosurgery, University of Occupational and Environmental Health, 1-1 Iseigaoka, Yahata Nishi-ku, Kitakyushu, Fukuoka 807-8555, Japan.