Delayed nonhemorrhagic encephalopathy following mild head trauma

Case report

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Delayed nonhemorrhagic encephalopathy following mild head trauma is a rare condition with an unknown etiology. The few cases reported in the literature are in young adults, all of them in the era before computerized tomography (CT) became available, and all had a devastating clinical course with multifocal ischemia or necrotic lesions found at autopsy. A case is presented of a young man with this syndrome who survived the acute encephalopathic phase with severe residual neurological deficits. Repeat CT scans during and following the acute phase as well as magnetic resonance imaging showed diffuse multifocal lesions compatible with ischemic changes and demyelination in the “watershed” areas of the brain.

KEY WORDS • encephalopathy • posttraumatic encephalopathy • blindness • hemiplegia

Delayed nonhemorrhagic encephalopathy following mild head trauma is rare. The reported patients have been young people who suffered a mild head injury and, after a symptom-free interval lasting from minutes to several hours, developed neurological deficits ranging from cortical blindness to severe focal deficits and deep coma. The clinical course of these patients was usually devastating, with high mortality rates or very severe residual neurological deficits. The pathogenesis of this condition is unknown; however, autopsy findings in four such patients2,9,10 have shown multifocal ischemic or necrotic lesions with no clear geographical distribution. The radiological data of this condition are extremely limited as the reported cases are from before computerized tomography (CT) became available. The clinical course as well as CT and magnetic resonance (MR) studies of a young man with this condition are described.

Case Report

This 29-year-old man suffered a blow to the head during a fist fight. He did not lose consciousness and, apart from a mild headache, was able to continue his daily routine as a salesman. Two hours later he started to complain of increasingly blurred vision which within minutes progressed to complete blindness. On his way to the hospital he noticed weakness of his left hand and had trouble in speaking.

Admission. On arrival at the emergency room, the patient was alert, although drifting to sleep when left alone. Examination revealed moderate mixed aphasia, paresis of central origin of the facial nerve on the right, and hemiparesis on the left side. The pupils reacted to light although the patient was totally blind. Funduscopic examination was normal. Thirty minutes after admission the patient went into deep coma with dense hemiplegia on the left side which was soon replaced by bilateral decerebrate posturing. A skew deviation of the eyes was noted. Vital signs remained normal and stable. Computerized tomography scans (both unenhanced and enhanced) showed no intracranial abnormalities.

Lumbar puncture disclosed normal pressure and normal composition of the cerebrospinal fluid (CSF). Blood count and chemistry were within normal limits. Arterial blood gases were normal except for mild metabolic acidosis (pH 7.316, base deficit of 6.5).

The patient was treated with hyperventilation, ci-
Encephalopathy following mild head trauma

FIG. 1. Upper Pair: Unenhanced computerized tomography (CT) scans of the brain on admission with a normal gray/white matter differentiation. No changes were observed after the administration of contrast material (not included). Lower Pair: Unenhanced repeat CT scans 3 days after admission. Large hypodense regions in the watershed areas of both hemispheres are evident, with edema and a slight symmetrical ventricular compression. Again, no changes were noted after intravenous injection of contrast material (not included).

FIG. 2. Upper Pair: Magnetic resonance (MR) images of the brain (spin-echo, T₁- and T₂-weighted images) obtained 21 days after admission. Scattered small hyperintense lesions in the white matter or both hemispheres are seen. A symmetrical enlargement of both ventricles is also present. Lower Pair: Computerized tomography scans obtained on the same day as the MR images. The hypodense white matter areas are barely visible. A symmetrical enlargement of both ventricles with deep sulci is evident secondary to moderate brain atrophy.

metidine, and minidose heparin. Antibiotic agents were added for treatment of a lung infiltrate which was attributed to aspiration. Repeated analysis of arterial blood gases showed good oxygenation. His condition remained unchanged for 3 days when repeat CT scans were obtained (Fig. 1 lower pair). The CT showed hypodense lesions in the “watershed” areas of both hemispheres, most prominent in the frontal and occipital lobes. Neurological examination revealed coma with decerebrate posturing, Cheyne-Stokes respiration, and medial longitudinal fasciculus dysfunction on the right.

A tracheotomy was performed and the patient was successfully weaned from the respirator. A repeat lumbar puncture revealed normal pressure, and CSF analysis disclosed normal chemistry values.

Course. On Day 9 the patient started to open his eyes to command, but no voluntary motor response was observed. Optokinetic nystagmus was elicited and pursuit of objects with his eyes was observed. Decerebrate posturing in response to painful stimuli was noted even at times when the patient was obviously awake. Five days later the patient started to communicate with his family and the staff by speaking and crying. Neurological examination disclosed flaccid paralysis of both upper limbs with slight paresis of the lower limbs. Respiration returned to normal and oral feeding was instituted. Repeat CT and MR imaging were performed on Day 21 (Fig. 2). While CT scanning showed disappearance of the previously described hypodense lesions, some degree of brain atrophy was noted with slight ventricular and cisternal dilatation (Fig. 2 lower pair). Magnetic resonance imaging, however, still showed regions of hyperintensity in the watershed areas compatible with demyelination (Fig. 2 upper pair).

The patient was discharged to a rehabilitation center on Day 30 posttrauma. Six months later he still had severe paresis of both upper limbs, but was otherwise neurologically intact.

Discussion

Acute encephalopathy associated with severe head injury has been extensively described in the literature. Most of the reported cases were linked to severe head injury causing an immediate and prolonged period of unconsciousness without evidence of intracranial mass lesions. These patients were left with neurological deficits of variable severity. Cortical blindness was a common finding, but other deficits included motor dysfunction, severe dementia, and a chronic vegetative
Most of these reports suggested a vascular etiology for these residual neurological deficits.

Delayed encephalopathy following mild head injury is much rarer. In contrast to the so-called "posttraumatic stupor" or transient blindness following trivial head injuries not uncommonly reported in children, which carry a favorable prognosis, delayed posttraumatic encephalopathy in adults usually has a devastating clinical course with death or severe neurological dysfunction as the outcome. The clinical course of our patient is highly suggestive of this condition, of which very limited data exist.

The head injury itself was very mild, not sufficient even to cause temporary loss of consciousness. Symptoms became evident only after a period of 2 hours and progressed from focal deficits such as blindness, aphasia, and hemiparesis to severe involvement of the brain stem. The initial CT scan was normal, but 3 days later marked hypodense regions in the watershed areas were seen, suggesting involvement of the white matter perhaps due to ischemia. These changes were gradually disappearing on a CT scan obtained 18 days later, correlating with clinical improvement of the patient. Magnetic resonance imaging demonstrated the sequel of the acute event in the form of hyperintense, diffuse lesions in the white matter compatible with areas of cell death and demyelination. Moderate brain atrophy was also a late finding in both CT and MR imaging.

The poor clinical outcome reported in patients with delayed nonhemorrhagic encephalopathy after mild head trauma was also demonstrated in our patient. Although surviving the acute encephalopathic event, he was left with a severe, apparently permanent, neurological deficit.

The pathogenesis of this condition is unknown. The autopsy reports in four such patients which revealed multifocal ischemia or necrotic lesions suggest a link to the functional cerebrovascular abnormalities that follow trauma. The radiological data from our patient may be compatible with this hypothesis. The hypodense lesions in the watershed areas may indicate diminished blood flow in major intracranial vessels, both in the anterior and posterior circulations. Interestingly, some of these lesions resolved with time, suggesting a mechanism of transient vasospasm unrelated to subarachnoid hemorrhage.

References

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