Paramedian Thalamic Infarction Following Blunt Head Injury

—Case Report—

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Abstract

A 19-year-old male was admitted following a blow to the face. Computed tomographic (CT) scans 1 hour after injury revealed low-density areas in the bilateral thalami and midbrain, which were enhanced postcontrast except for the core 3 hours later. CT scans 2 days after injury revealed that the size of the low-density areas had increased. CT scans and magnetic resonance images 3 weeks after injury disclosed only small infarcted lesions in the bilateral thalami, the right side of the midbrain, and the left internal capsule. These findings suggest that the injury initially caused thrombus on the basilar arterial wall, leading to occlusion of the perforators, but almost all affected perforators were recanalized. Bilateral thalamic infarction resulting from head injury is unusual, as is the transient nature of the infarction in this case.

Key words: head injury, paramedian thalamic infarction, computed tomography, magnetic resonance imaging

Introduction

Thalamic infarction is frequently bilateral due to the nature of the vascular supply to the thalamic region,^{1,3-5,8-10)} and is usually caused by cardiogenic embolism or emboli originating from the proximal basilar or vertebral artery. This condition is known by various terms, including paramedian diencephalic syndrome,⁸⁾ syndrome of bilateral paramedian thalamic infarction,⁵⁾ syndrome of basilar communicating artery,⁹⁾ and syndrome of mesencephalic artery.¹⁰⁾ All these syndromes can be defined generally as "top of the basilar" syndrome.²⁾ The characteristic clinical symptoms and computed tomographic (CT) findings associated with bilateral thalamic infarction are well known. We recently encountered a patient presenting with bilateral thalamic infarction after head injury, with an unusual transient visualization by CT.

Case Report

A 19-year-old male was brought to our hospital on November 27, 1990, because he had been injured in a collision with a stationary truck while riding a motorcycle. On admission, he could open his eyes, but his speech was indistinct and he could not answer questions. There were no focal signs. The right upper eyelid and the left lower jaw were bruised. CT scans 1 hour after injury revealed bilateral low-density areas in the thalami and midbrain (Fig. 1). Anisocoria developed on the right, so repeat CT scans were performed 4 hours after injury. Contrast medium had been injected for abdominal examination 2.5 hours previously. The repeat CT scans showed enhancement of the low-density areas in the periphery but not the core (Fig. 2). CT scans 2 days after injury revealed that the size of the low-density areas had increased (Fig. 3). He was quite restless, requiring sedation, so the level of consciousness could not be determined during the first 5 days of hospitalization. The low-density areas gradually became smaller, and had almost disappeared 10 days

Fig. 1 Precontrast CT scans 1 hour after head injury, showing symmetrical and bilateral low-density areas in the thalami and midbrain.

Fig. 2 Postcontrast CT scans 4 hours after injury, revealing the enhanced low-density areas excluding the central core. The contrast material had been infused 2.5 hours before the examination.

Fig. 3 Precontrast CT scans 2 days after injury, showing the enlarged low-density areas in the thalami and midbrain.

Fig. 4 Vertebral angiogram 3 weeks after injury, revealing irregularity of the wall (arrowheads) and fusiform dilatation of the perforator (arrow).

of the midbrain, and the posterior limb of the left internal capsule (Figs. 5 and 6).

He was discharged 1 month after admission with hypersomnolence, apathy, and amnesia accompanied by mild paralysis of the right oculomotor nerve and right homonymous hemianopia. His level of consciousness was clear 9 months after injury, although mild lateral strabismus of the right eye and right homonymous hemianopia were still present.

Discussion

The present case is quite unusual, with transient extensive low-density areas in the thalami and the midbrain visible on CT scans after head injury. Transient bilateral thalamic lesions visualized by CT have been reported,⁷⁾ but the etiology was attributed to inflammatory lesions.

Whether the transient lesions in our case were due to direct or indirect injury is uncertain. The patient was conscious after the accident, so extensive direct damage to the brain is unlikely, and the lesions were probably secondary to the associated vascular injury. Mechanisms causing vascular damage following head injury include neurovascular friction,¹¹⁾ direct damage to the vascular wall, and compression or kinking of vessels.⁶⁾ In our patient, the blow to the face may have caused the clivus to compress the basilar artery, resulting in thrombus formation on the damaged vascular wall and leading to occlusion of the perforators from the terminal basilar artery. Subsequent recanalization in almost all perforators effectively limited the infarcted areas to the paramedian region of the bilateral thalami, the right side of the midbrain, and the posterior limb of the left internal capsule. Such a process would account for the

after injury. The auditory brainstem responses were unremarkable 2 days after injury. Vertebral angiograms 3 weeks after injury revealed irregularity of the basilar arterial wall and fusiform dilatation of the thalamoperforator (Fig. 4). CT scans and magnetic resonance (MR) images showed residual small infarcts in the bilateral thalami, the right side

Fig. 5 Precontrast CT scans 3 weeks after injury, showing localized low-density areas in the bilateral thalami, the right side of the midbrain, and posterior limb of the left internal capsule.

Fig. 6 T₂-weighted MR images 3 weeks after injury, clearly showing infarcted lesions in the paramedian thalami, midbrain, and posterior limb of the left internal capsule.

very unusual CT findings of an unenhanced central low-density core surrounded by enhanced low-density areas which later disappeared (Fig. 2). The core represents the area affected by permanent occlusion, while the latter represents the area reperfused after recanalization.

Bilateral thalamic lesions caused by obstruction of the single feeding artery to the medial thalamus, called the thalamosubthalamic or the paramedian artery, are characterized by hypersomnolence, apathy, amnesia, and impaired vertical gaze. In our patient, hypersomnolence, apathy, amnesia, paralysis of the right oculomotor nerve, and right homonymous hemianopia were present at discharge; but only the right lateral strabismus and right

homonymous hemianopia persisted 8 months after discharge.

Although various etiologies including embolism, atherosclerosis, and inflammation for bilateral thalamic infarction have previously been described, no case secondary to head injury has been documented.

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