

Bilateral Traumatic Hemorrhage of the Basal Ganglia

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Bilateral traumatic hemorrhage of the basal ganglia is an extremely rare neuropathologic entity. This report describes a 50-year-old man with bilateral basal ganglia hemorrhage with occipital fracture of the skull after head trauma. The mechanism of development of traumatic hemorrhage of the basal ganglia has been not clear. But, it is presumed to be secondary to rupture of the lenticulostriate or anterior choroidal artery by shearing as a result of acceleration/deceleration forces. We briefly summarize our uncommon case and discuss its possible mechanisms.

KEY WORDS : Basal ganglia · Hemorrhage · Contusions · Head trauma.

Introduction

Traumatic basal ganglia hematoma (TBGH) is a rare entity defined as a hemorrhagic lesion located in the basal ganglia or neighboring structures such as the internal capsule and the thalamus^{1,2)}. It can be classified as “large” if it is more than 2 cm in diameter or as “small” if it measures less than 2 cm in diameter¹⁾. Its incidence is about 3% of patients after a closed head injury^{5,9)}. The mechanism of TBGH has not been clear, but, it is believed to arise from a shear strain in the ganglionic region. TBGH can occur as an isolated lesion or as another associated intracranial injury such as diffuse axonal injury, cerebral contusion, and subdural or epidural hematoma. TBGHs are associated with a worse prognosis than other types of posttraumatic intracranial hemorrhages^{2,6,7,9)}. But, when TBGHs occur in isolation, they show good outcome^{5,7)}.

We briefly summarized our uncommon case of bilateral TBGH and discussed its possible mechanisms.

Case Report

A 50-year-old man complained of headache and mild occipital swelling after fall down during fight.

His medical histories were not remarkable. Blood pressure of the patient was within normal range on admission. He had no history of a loss of consciousness after head injury. Neurological examinations revealed no abnormalities. The Glasgow Coma Scale score on admission was 15. Skull x-rays showed a vertical, linear fracture line at the midline of occipital bone of the skull (Fig. 1). Computerized tomographic (CT) scans showed small bilateral hemorrhage of the basal ganglia, punctuate hemorrhage in genu of left internal capsule and traumatic subarachnoid hemorrhages in both frontal region and occipital, clival fractures of the skull (Fig. 1). CT angiography revealed no vascular abnormalities. Magnetic resonance (MR) images taken two days after head injury showed acute hemorrhage with surrounding edema in both basal ganglia without abnormal

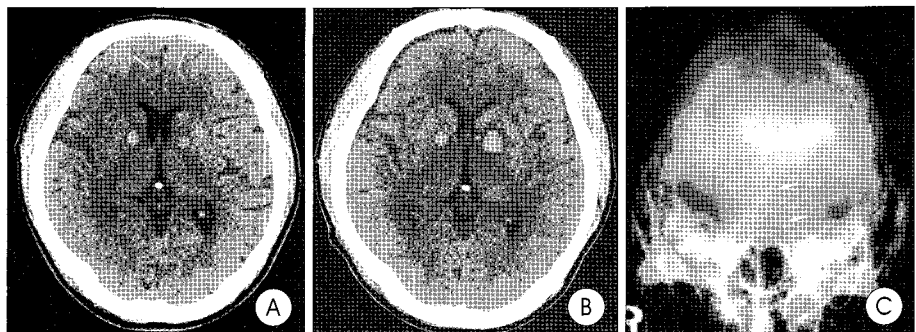


Fig. 1. Brain computerized tomography scans and skull x-ray. A, B : Non-enhanced axial images show bilateral hemorrhage of the basal ganglia, punctuate hemorrhage in genu of left internal capsule and traumatic subarachnoid hemorrhage in both frontal region (white arrows). C : Towne's view of the skull reveals a vertical, linear occipital fracture of the skull (white arrows). Note that the fracture line is at the midline of occipital bone of the skull. The strong impact at this midline might have caused bilateral hemorrhage of the basal ganglia.

• Received : November 10, 2006 • Accepted : February 14, 2007

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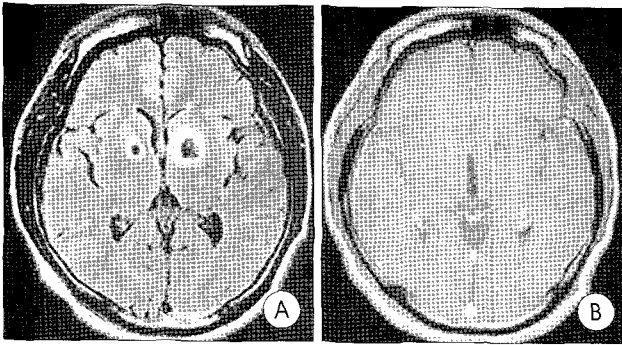


Fig. 2. Brain magnetic resonance images 2 days after head injury. A : Axial fluid-attenuated inversion recovery image shows acute hemorrhage with surrounding edema in both basal ganglia. B : Axial T1-weighted image after gadolinium injection shows no abnormal enhancements.

enhancements (Fig. 2). The laboratory results were within normal ranges, including complete blood cell counts, bleeding time, prothrombin time, activated partial thromboplastin time, liver function tests, and blood glucose level. Blood pressure of the patient had been within normal range through a 2-week admission period. With regards to an etiology of bilateral hemorrhage of the basal ganglia, we could not disclose any possible culprit except head injury in spite of full diagnostic work-up including a careful history-taking and through imaging studies. Our final diagnosis was bilateral traumatic hemorrhage of the basal ganglia. After conservative treatment was carried out and the patient was discharged 14 days after injury with no neurological deficits.

Discussion

Traumatic basal ganglia hematoma (TBGH) is an uncommon event, which is observed in about 3% of patients with a closed head injury^{5,9)}. But, in autopsy series, its incidence is higher, ranging between 10% and 12%¹⁾, this discrepancy reflects a high mortality of patients with TBGH. Adams et al.¹⁾ reported 63 patients with basal ganglia hematomas in an autopsy series of 635 fatal non-missile head injuries. In patients with basal ganglia hematomas, contusions were more severe, there was an increased incidence of traffic accidents, gliding contusions and diffuse axonal injury than in patients without this type of hematoma. In this case, another interesting aspect is the bilateral occurrence of the TBGH with mirror image. There is only one report of two cases of bilateral TBGH by Yanaka et al.¹²⁾ to date.

There is a controversy whether a deeply located hematoma such as the basal ganglia is spontaneous or traumatic in origin. However, in this case, the patient has previously been in good health and his medical history including hypertension was not evident. His blood pressure had been within normal ranges during admission period. He did not have any other possible

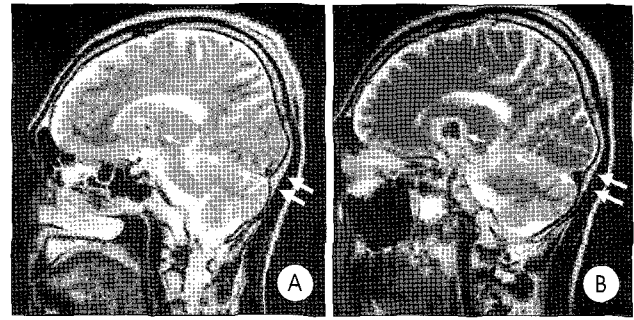


Fig. 3. Brain magnetic resonance images showing a correlation of the direction of the impact and hemorrhage site. Sagittal T2-weighted image shows the direction of an occipital impact (large white arrows) and the tentorium (small white arrows) corresponds with hemorrhage site of the basal ganglia. A : right, B : left.

causes of bilateral basal ganglia hemorrhage except head injury as evidenced by full diagnostic work-up studies that did not show other causes of intracerebral hemorrhages such as aneurysm, arteriovenous malformation, coagulopathy, vasculitis, intoxication. Brain CT revealed small hemorrhages of both basal ganglia, traumatic subarachnoid hemorrhages in both frontal region and an associated occipital fracture of the skull, which were other evidences of head injury. Therefore, bilateral TBGH was made as a final diagnosis.

The mechanism of TBGH has not been clear, but, it is believed to arise from a shear strain in the ganglionic region. Mosberg and Lindenberg¹⁰⁾ histologically demonstrated a traumatic tear of a pallidal branch of anterior choroidal artery as the origin of pallidal hematoma in an autopsy case. TBGH is caused by shearing injury of the lenticulostriate or anterior choroidal artery as a result of acceleration/deceleration forces^{1,9,10)}. When the strong impact is applied to the vertex, forehead, or occipital area and directed toward the tentorium, there would be a shift of the brain through the tentorial notch with stretching and tearing of vessels by shearing forces, resulting in hemorrhages in the basal ganglia region^{2,4,8,10)}. We have demonstrated that the direction of an occipital impact and the tentorium that corresponded with hemorrhagic site of the basal ganglia in the T2-weighted sagittal image of our patient (Fig. 3). Also, the impact at the midline might have added the causes of bilateral hemorrhage of the basal ganglia.

Contusions are bruises of the neural parenchyma as a result of head injury. Because deeply located hemorrhages such as the basal ganglia develop in the parenchyma between coup and contrecoup contusions, they have been considered as intermediary contusions³⁾, although the mechanisms of contusions remain poorly understood, which might result from ischemia by the compression of vessels irrigating the basal ganglia region as a result of shearing injury¹⁰⁾. In this case, if occipital fracture is presumed to be coup injury, traumatic subarachnoid hemorrhage in both frontal regions can be

considered a contrecoup injury, and finally, bilateral basal ganglia hemorrhage is likely to be intermediary contusions. Basal ganglia hemorrhages of our patient are classified as "small" hemorrhages because they measure less than 2 cm in diameter. These "small" TBGHs could be considered to be not hematomas, but hemorrhagic contusions, suggested by Mosberg and Lindenberg¹⁰. "Small" TBGHs are likely to be closely associated with diffuse axonal injury, which is associated with contusions and/or small hemorrhage in the corpus callosum, basal ganglia, tegmentum of pons, intraventricular, and acute brain swelling¹¹. "Small" basal ganglia hemorrhage is likely to be one of CT findings of diffuse axonal injury¹¹. The final prognosis of patients with TBGH is strongly related to the existence of associated diffuse axonal injury^{5,7}. In our case, diffuse axonal injury was not considered clinically because the patient had no history of a loss of consciousness after injury. The recognition of the existence of associated diffuse axonal injury seems to be very important in the predilection of outcome of patients with TBGHs.

In addition, since the basal ganglia is a region predisposed to hypertensive hemorrhage, when head injury and hematoma are simultaneously present. Despite thorough evaluations for its etiology, however, it may still be difficult to reach a definitive conclusion. For this reason, differential diagnosis of traumatic or spontaneous basal ganglia hematomas can be a crucial medicolegal issue. Therefore, a very careful evaluation of both past medical histories and physical examination, along with imaging study modalities, are mandatory for correct diagnosis and proper management.

Conclusion

We believe that this case might be ascribed to head trauma as an etiology of bilateral basal ganglia hemorrhage. "Small" hemorrhages of the basal ganglia seem to be hemorrhagic contusions as a result of a shearing injury after an occipital impact.

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