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Case report

Bilateral traumatic hemorrhage of the basal ganglia

Yun-Xu Zhang*, Shu-Qin Wei, Yan-Yun Xing, Qi Liu, Wen-Jing He

Hiser Medical Group, Qingdao, China

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ABSTRACT

Hemorrhage of the basal ganglia is common in hypertensive patients, and most of the cases are spontaneous unilateral hemorrhage. Traumatic basal ganglia hemorrhage is uncommon, while bilateral hemorrhage of the basal ganglia after trauma is an extremely rare entity. This report described a rare case of bilateral hemorrhage of the basal ganglia after head trauma. We also analyzed the mechanisms and reviewed relative literatures.

Introduction

Intracerebral hemorrhage is a common symptom in stroke patients. The most common location is the basal ganglia and the common cause is hypertension. Traumatic basal ganglia hemorrhage is uncommon, while bilateral hemorrhage of the basal ganglia after trauma is an extremely rare entity. We described a rare case of bilateral hemorrhage of the basal ganglia after head trauma in this report and analyzed the possible mechanisms.

Case report

A 45-year-old female was transmitted to the emergency room, unconscious, after a conflict with others at the market. She was hit in the face and fell down with the head heavily stricken against the ground, resulting in immediate unconsciousness. On admission, the patient's blood pressure was 160/100 mmHg and blood sugar 5.9 mmol/L, together with negative results of electrocardiograph (ECG), complete blood count (CBC), prothrombin time (PT), and activated partial thromboplastin time (APTT). The patient had no history of hypertension or diabetes. Physical examination demonstrated that: (1) the Glasgow coma scale (GCS) score was 8; (2) a scalp hematoma appeared on the right side of the head; (3) the pupils were equal in size, 3 mm in diameter and brisk in light reflex; and (4) the Babinski sign was positive on both sides.

Computed tomography (CT) demonstrated bilateral hemorrhage of the basal ganglia, without skull fracture, epidural hematoma, subdural hematoma, or brain contusion (Fig. 1A). After primary process, the patient was transferred to the neurosurgical unit where routine treatment was initiated. Mannitol was used to control intracranial pressure and cerebral edema; phenobarbital was used to prevent seizure attack; and other medicines to promote recovery. The blood pressure of the patient was monitored by an electrocardiogram monitor. Throughout the hospital stay the patient's blood pressure was within the normal range without administration of any antihypertensive drugs. A second brain CT scanning was performed 48 h after admission, which showed that the high density areas were surrounded by low density edema zone (Fig. 1B). The third brain CT scanning was performed on day 21, and the images showed that most of the blood clots have been absorbed (Fig. 1C).

The patient was discharged on day 29 with hemiparesis of the left body. Unfortunately, she refused a fourth CT examination and digital subtraction angiography (DSA) of the intracranial vessels. An one-month follow-up was performed after discharge, and the patient's blood pressure was monitored closely which remains normal without taking any antihypertensive drugs.

Discussion

Intracerebral hemorrhage can be divided into traumatic and nontraumatic, and the latter is also known as spontaneous

* Corresponding author.
E-mail address: 13573826611@163.com (Y.-X. Zhang).

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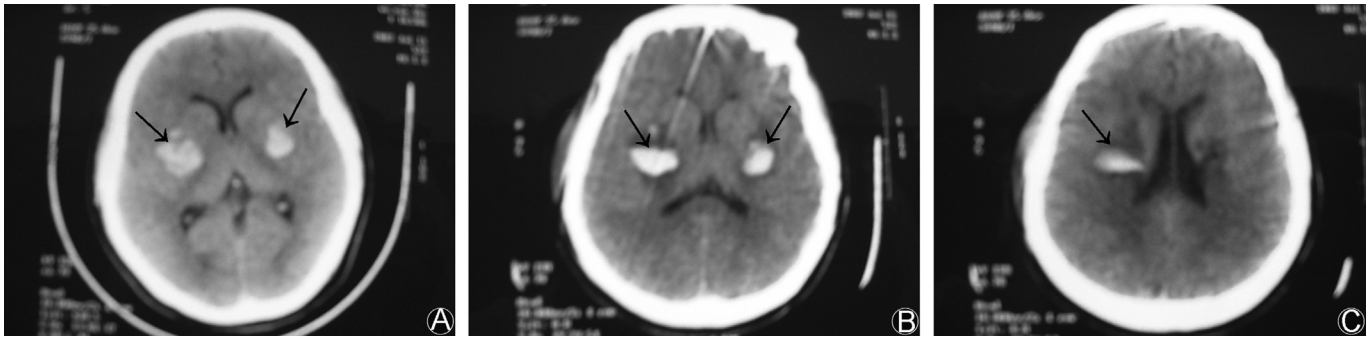

Fig. 1. CT images at admission (A), 48 h after admission (B) and day 21 (C).

intracerebral hemorrhage. Hemorrhage of the basal ganglia is common in hypertensive patients, and most of them are spontaneous unilateral hemorrhage. Traumatic basal ganglia hemorrhage is rare and its incidence in patients with closed head injury is reported as 3% in some case series.[1,2] Whereas in autopsy study, the incidence is higher, about 10%. Most of the traumatic basal ganglia hemorrhages are companied by skull fractures, epidural hemorrhage, subdural hemorrhage, subarachnoid hemorrhage or brain stem injuries, with poor prognosis. That is why the incidence is higher in autopsy series. Simple bilateral hemorrhage of the basal ganglia without other lesions is extremely rare.[3] There have been only two reports of three cases of bilateral traumatic hemorrhage of the basal ganglia to date,[4] and the underlying mechanism is still unclear.

At present, there are two hypotheses about the mechanisms of intracerebral hemorrhage of the basal ganglia after brain trauma, namely the “spontaneous or traumatic hemorrhage”. In spontaneous hemorrhage hypothesis, people believe that basal ganglion is a region predisposed to hypertensive hemorrhage. When the patient's blood pressure elevates abruptly after emotion stimulation or physical exertion, the lenticulostriate artery ruptures and hematoma forms. Congenital malformation or long-term hypertension and diabetes may be responsible for this propensity.

In traumatic hemorrhage hypothesis, “shearing force” is believed to be the cause of hemorrhage.[5,6] Lindenberg[7] histologically demonstrated a traumatic tear of a pallidal branch of anterior choroidal artery as the origin of pallidal hematoma in an autopsy case. Hemorrhage is caused by shearing injury of the lenticulostriate or anterior choroidal artery as a result of acceleration/deceleration forces.[3,5–7] 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. Some experts call this intermediary contusions.[8–10]

In this case, we took a detailed medical history of the patient, who reported no history of hypertension or diabetes. During her hospital stay and one-month follow-up after discharge, the patient's blood pressure and blood sugar were within the normal range without taking any drugs. So hypertension and diabetes can be ruled out from the predisposing factors. Since there were no predisposing factors, spontaneous hemorrhage can be excluded. The relative higher blood pressure (160/100 mmHg) at admission could be the result of stress reaction to head trauma and intracerebral hemorrhage. The history of head trauma was clear, and confirmed by the results of physical examination. Shearing force seemed to play a critical role in the hemorrhage, and traumatic hemorrhage was the diagnosis.

In conclusion, we believe that bilateral basal ganglia hemorrhage in this patient was the result of head trauma. Since there wasn't a digital subtraction angiography examination of the intracranial vessels, we couldn't figure out if there was any defect with the patient's lenticulostriate or anterior choroidal artery, which is a limitation of this report.

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