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Isolated bilateral, large, basal ganglia haemorrhage following a traumatic brain injury: A case report

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Abstract

Traumatic basal ganglia haemorrhage is rarely seen in clinical practice. Bilateral basal ganglia hematoma without any other cerebral lesions due to trauma is extremely uncommon and has been reported only in a few cases. Although the mechanisms of this condition are unclear, haemorrhagic contusions are thought to arise as a consequence of a shearing strain on cranial blood vessels due to high-velocity forces at the time of the injury. Here we describe a 63-year-old female patient with an isolated bilateral, large, basal ganglia haemorrhage secondary to a road traffic accident. The patient was promptly diagnosed and conservatively treated and had fully recovered after two months.

Keywords

Basal ganglia haemorrhage, bilateral, traumatic brain injury

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Introduction

Basal ganglia lesions can be caused by many pathologies that can be separated into: inherited metabolic/genetic; acquired metabolic/toxic; inflammatory and infectious;

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vascular and ischemic; neoplastic.¹ A traumatic basal ganglia hematoma (TBGH) is defined as an intracerebral haemorrhagic lesion located in the basal ganglia (i.e., caudate nucleus, putamen and globus pallidus) and neighbouring structures such as the thalamus and internal capsule.² According to the diameter of the hematoma, a TBGH may be defined as 'small' when it is less than 2 cm, or 'large' when it is more than 2 cm.³ Bilateral TBGHs are extremely rare.⁴⁻⁹ Although the pathophysiology remains unclear, some theories suggest that the condition may arise from sudden acceleration or deceleration forces that result in shearing of the lenticulostriate or anterior choroidal arteries.4,10 Most cases are managed conservatively and the outcome is variable.^{7,11} Cognitive function may be affected and poor prognosis has been reported to be related to advanced age, associated intraventricular haemorrhage, ventilator dependence, large hematomas (volume >20 ml), poor Glasgow Coma Scale (GCS) on admission, and diffuse axon injury (DAI).9,11 Here we describe a 63-year-old female patient with an isolated bilateral, large, basal ganglia haemorrhage secondary to a road traffic accident.

Case Report

A 63-year-old woman was admitted to our emergency department in an unconscious state following a road traffic accident. On examination, her GCS was 9 (GCS ranges from 3 [completely unresponsive] to 15 [responsive]) and she had positive bilateral Babinski reflexes. Her pupils were 2mm bilaterally and reacted to light. Multiple abrasions over her left hand were noted. The patient's medical records showed well controlled hypertension and hepatitis B and hepatitis C virus infections. On admission, the patient's blood pressure (BP) was 150/ 90 mmHg, and she continued to receive her prescribed antihypertensive medication (i.e., perindopril/amlodipine 5/10 mg).

The initial computed tomography (CT) scan showed a haemorrhage within the basal ganglia bilaterally without skull fracture, epidural hematoma, subdural hematoma, brain contusion, or midline shift (Figure. 1a). The haemorrhage was located in lentiform nucleus and consisted of small hyperdense petechiae beside a large haemorrhage. Laboratory results were within the normal range including: complete blood cell counts; bleeding time; prothrombin time; activated partial thromboplastin time; liver function tests; blood glucose (i.e., 6.06 mmol/l). A second brain CT scan taken 6 hours after admission. showed low-density oedematous zones surrounding the haemorrhage areas (Figure 1b). A third brain CT scan was performed on Day 12 (Figure 1c). CT angiography did not show any vascular abnormalities (Figure 2). Magnetic resonance imaging (MRI) scans on Day 17 showed haemorrhages with surrounding oedema in both basal ganglia without any abnormal enhancement (Figure 3). CT scans of the patient's left hand showed a fifth metacarpal head fracture and ulna styloid fracture.

Following conservative treatment, the patient was discharged on Day 31 but had quadriplegia (i.e., muscle strength grades 3/5 on the right side and 4/5 in the left side) and aphasia. Her Mini-Mental State Exam (MMSE) score was 14/30 (i.e., moderate) and executive function score was 4/18. Two months after the accident, the patient showed good recovery of neurological function; she had normal strength in all four limbs, normal speech and reported no disturbance of daily activities. Her MMSE score was now 24/30 (i.e., mild) and executive function score was 15/18.

Written informed consent for the publication of this report was obtained from the patient and it was subsequently reviewed and approved by the local ethics committee



Figure I. (a) The initial computed tomography (CT) scan showed a haemorrhage within the basal ganglia bilaterally without skull fracture, epidural hematoma, subdural hematoma, brain contusion, or midline shift. The lesion consisted of small hyperdense petechiae next to a large haemorrhage. (b) A second brain computed tomography (CT) scan was performed 48 h after admission and showed low density oedematous zone surround by haemorrhagic areas. (c) The third computed tomography (CT) scan was performed on Day 12 showed that most of the hematomas were absorbed.

of the Vietnam Military Medical University. This report adheres to CARE guidelines.¹²

Discussion

The prevalence of TBGH has been estimated to between 2.4–3% in closed head

injuries.^{2,13} However, in autopsy series, the prevalence has been found to be higher at 10–12%.^{3,13} Bilateral TBGH is extremely rare and a limited number of cases have been reported.^{4–9}Although the mechanism of TBGH remains unclear, two hypotheses have been suggested, namely spontaneous,



Figure 2. The computed tomography (CT) angiography did not show any vascular abnormalities.



Figure 3. Magnetic resonance imaging (MRI) scans on Day 17 showed haemorrhage with surrounding oedema in both basal ganglia without abnormal enhancement.

or traumatic haemorrhage. According to the spontaneous haemorrhage hypothesis, it is thought that the basal ganglia is a region predisposed to hypertensive haemorrhage. Following an abrupt increase in BP after emotional stimulation or physical exertion, the lenticulostriate artery ruptures and a hematoma is formed.⁶ In the traumatic haemorrhage hypothesis, a strong impact applied to the vertex, forehead, or occipital area and directed toward the tentorium, causes a shift of the brain through the tentorial notch with stretching and tearing of the lenticulostriate or anterior choroidal artery by shearing forces.^{2,3,13,14} Shearing of the blood vessels caused by rapid acceleration and deceleration forces at the time of trauma lead to parenchymal coup and countercoup contusions.^{3,15} While TBGHs are thought to be small, multiple, sometimes bilateral, and located in the zone of lentiform nucleus and external capsule, spontaneous haemorrhages are believed to be solitary and in the region of the thalamus and internal capsule.⁷

Although there is a controversy whether basal ganglia hematoma is spontaneous or traumatic in origin, we believe that the bilateral, large, basal ganglia haemorrhage in our patient was the result of her head trauma. She showed no evidence clinically or radiologically to suggest that there was any other cause for the lesion. For example, her hypertension was well-controlled. Apart from a relatively high BP immediately after her trauma, the patient's BP was within normal range throughout her hospital admission. The increase in BP around the time of her accident was probably due to the stress reaction to the trauma and the intracerebral haemorrhage.⁶ Despite a history of hepatitis B and C virus infections, her liver function tests and other blood tests indicated no coagulation disorders. She had no history of substance abuse or toxic inhalation. In addition, the patient had symptoms of haemorrhage immediately after her trauma.

Variable outcomes have been reported for patients with a TBGH.⁵ One study in children found that the presence of TBGH in severely head-injured patients worsens the prognosis and outcome. ¹⁶ Indeed, TBGH is thought to be associated with a worse prognosis than other types of posttraumatic intracranial haemorrhages.⁸ Almost all patients who suffer simultaneous bilateral basal ganglia hematoma have a poorer outcome compared with solitary intracerebral haemorrhage.¹⁴This is mainly due to the destruction of crossing and noncrossing fibres, bilateral diaschisis phenomenon, and development of severe disturbed consciousness, quadriparesis and pseudobulbar palsy.¹⁴ In one study of 41 patients with TBGH, 38% had a favourable prognosis and the mortality rate was 10%.¹¹ By contrast, other studies in smaller numbers of patients have found favourable outcomes in all patients with no mortality.^{5,7,13}

Several prognostic factors are thought to be associated with a poor outcome and include: advanced age; post-resuscitation GCS <7; abnormal pupillary response; abnormal response to pain; ventilator dependence; large hematoma volume (>20 ml); diffuse axonal injury (DAI); association with parenchymal injuries; intraventricular haemorrhage or brain stem haemorrhage; association with coagulation disorder.^{9,11,17} In the case reported here, despite the large size of the basal hematoma, the patient showed good recovery one month after hospital discharge and, two months later, had resumed normal life. Her mild cognitive deficits were probably associated with the DAI. We believe the favourable outcome in this patient was related to a purely basal ganglia hematoma with no other injuries in other intracranial locations.

In conclusion, although TBGH is rare and bilateral TBGH is extremely uncommon, they should be included in a differential diagnosis of basal ganglia haemorrhage. Moreover, it is crucial to differentiate the TBGH from a spontaneous, nontraumatic basal ganglia haemorrhages which may be secondary to cerebrovascular disease, so that treatment may be timely and appropriate. Conservative management can give good results but prognosis is variable and dependent on many prognostic factors.

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Declaration of conflicting interests

The authors declare that there are no conflicts of interest.

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