

CASE REPORT

Traumatic bilateral basal ganglia hematoma: A report of two cases

Pranshu Bhargava, Sarvpreet Singh Grewal, Bharat Gupta, Vikas Jain, Harman Sobti

Department of Neurosurgery, Christian Medical College, Brown Road, Ludhiana, Punjab, India

ABSTRACT

Traumatic Basal ganglia hemorrhage is relatively uncommon. Bilateral basal ganglia hematoma after trauma is extremely rare and is limited to case reports. We report two cases of traumatic bilateral basal ganglia hemorrhage, and review the literature in brief. Both cases were managed conservatively.

Key words: Basal ganglia hematoma, bilateral, traumatic

Introduction

Traumatic Basal ganglia hemorrhage (TBGH) is defined as a hemorrhagic lesion located in the basal ganglia or neighboring structures such as internal capsule or thalamus. TBGH is relatively uncommon.^[1] An incidence of 3–10% of head injuries has been reported.^[1,2] The incidence is higher in autopsy series when compared with clinical studies.^[1] Bilateral basal ganglia hematoma after trauma is extremely rare and is limited to case reports.^[3,4] The mechanism is unclear though it is proposed to arise from shear strain of the lenticulostriate or anterior choroidal vessels caused by acceleration/deceleration forces at the time of injury.^[2] The fact that Basal ganglia hematoma can be due to trauma per se takes importance in the medico legal cases where bleeding may be attributed to a non traumatic cause. Thus, the recognition of TBGH is of prime relevance.

Case Reports

Case 1

A 25 year old male, farmer by occupation, met with an accident when he was riding a bicycle and was hit by a fast moving truck. He was brought to the emergency in an unconscious state. Left ear bleed was present. There was no history of nose bleed, seizure, or cerebrospinal fluid leak.

There was no history of significant illness, substance abuse, or any history suggestive of any bleeding disorder. Patient was a non-diabetic non-hypertensive. Prior to the accident, he was not on any medication. On examination, his Glasgow Coma Scale (GCS) was E1V1M2 Pupils were 2 mm bilaterally, reacting to light. There was right hemiplegia. Right sided pneumothorax was present for which a tube thoracostomy was done. The pulse and blood pressure was normal. Patient was intubated and put on mechanical ventilation. A plain Computerized Tomogram (CT) showed Intracerebral bleed [Figure 1a] in the basal ganglia involving the corona radiata on the right (approximately 18 mm by 11 mm) and left (approximately 30 mm by 21 mm). There was small amount of blood in the right frontoparietal cortex. Sulcal spaces and basal cisterns were effaced. There was fracture of left temporal bone [Figure 1b] and blood in the left mastoid, middle ear, sphenoid and bilateral ethmoidal sinus. All blood parameters and radiology including clotting profile were within normal limits. Patient was shifted to neurosurgical ICU and decongestants in the form of frusemide and 20% mannitol were given. Antibiotics and phenytoin sodium was also given. He was continued on ventilatory support and subsequently tracheostomized on day 5. Nasogastric feed was also started gradually. A Gadolinium enhanced magnetic resonance imaging study (MRI) done on day 7 of injury did not reveal any vascular lesion [Figure 1a-c]. The hematomas were resolving and edema was decreasing. Non-contrast CT done after 35 days showed complete resolution of the hematoma [Figure 1d]. His motor score gradually improved to withdrawal response (M4) and eye opening was spontaneous (E4) by the 8th day of injury. Phenytoin was continued. The chest tube was removed after confirming lung expansion on chest radiograph. Supportive care was given and patient was weaned off the ventilator. He had spontaneous eye opening and was maintaining eye contact. On follow-up, patient had regained consciousness

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Address for correspondence:

Dr. Pranshu Bhargava, Department of Neurosurgery, Christian Medical College, Brown Road, Ludhiana, Punjab - 14 008, India.
E-mail: bhargava_pranshu@rediffmail.com

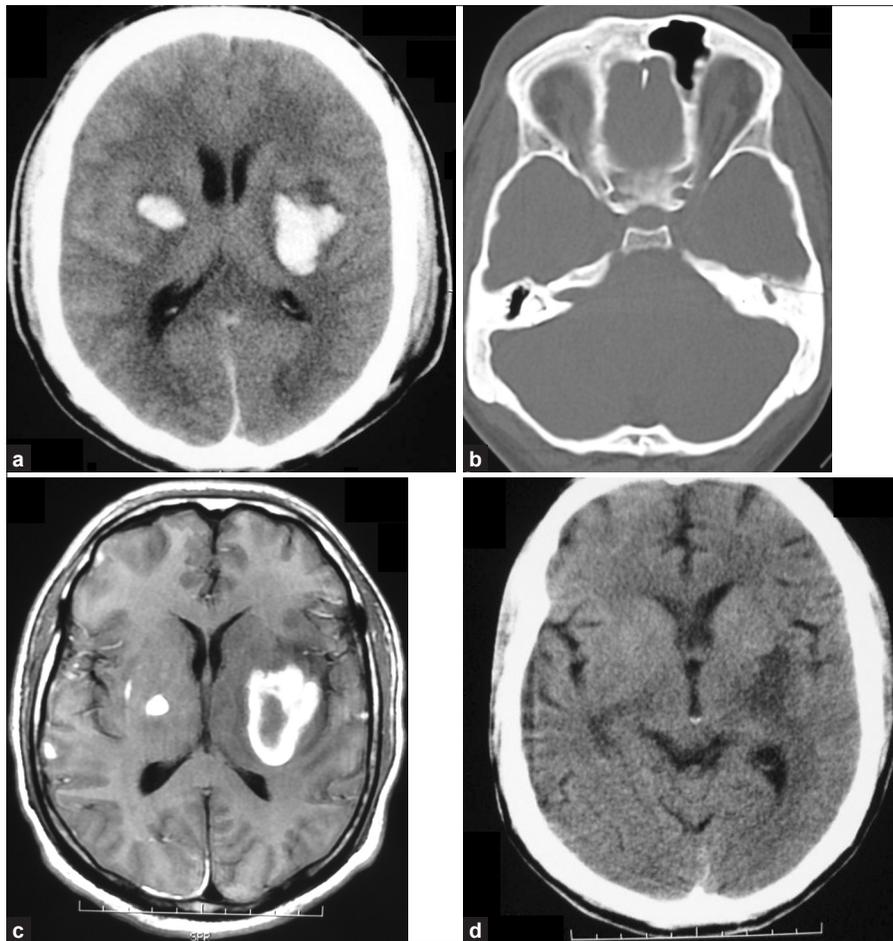


Figure 1: (a) Patient 1 initial plain CT showing bilateral basal ganglia bleed; (b) Patient 1 bone window showing fracture left temporal bone; (c) Axial T1 MRI showing resolving hematoma; (d) Patient 1 plain CT at 1 month

and obeyed verbal commands. He still had dysphasia with right spastic hemi paresis.

Case 2

A 50-year-old male, farmer by occupation, met with an accident while driving a two wheeler, being hit by a fast moving four wheeler. He lost consciousness immediately. There was no history of any substance abuse, previous illness, or bleeding diathesis. He was not on any regular medication. Patient was non-diabetic and non-hypertensive prior to the accident, he was not on any medication. On examination, his GCS was E1V1M2. Pupils were 4 mm bilaterally, reacting to light. Bilateral limb movements were equal. There were no apparent long bone fractures. Chest compression test was negative. All other parameters were normal. The pulse and blood pressure were normal. Patient was intubated and put on mechanical ventilation. A plain CT scan revealed basal ganglia bleed bilaterally [Figure 2], involving the temporo parietal regions, 4.2 by 1.8 cm on the right and 3.2 by 3.7 on the left. There was intraventricular hemorrhage also. Basal cisterns were effaced. Subsequent scan after 3 days showed that the hemorrhage was resolving. All blood and



Figure 2: Patient 2 plain CT head

radiological parameters including clotting profile were within normal limits. Patient was shifted to neurosurgical ICU and decongestants in the form of frusemide and 20% mannitol were given. Antibiotics and phenytoin sodium was also given. He was continued on ventilatory support, and subsequently tracheostomized on day 6. Nasogastric feed was

also started gradually. His motor score gradually improved to withdrawal response (M4) and eye opening to pain (E2) by the 4th day of injury. Supportive care was given. Patient started to localize by day 9, and was referred to district hospital for supportive care.

Discussion

The general incidence of TBGH is reported between 2.4-3%^[1,5] of closed head injury. However, the incidence is higher in post mortem studies (9.8%).^[1] Bilateral traumatic basal ganglia hematoma is extremely rare. Descriptions are limited to case reports.^[3,4] However, Kumar *et al.* found one patient with bilateral basal ganglia bleed amongst 10 cases of TBGH (10%).^[6]

It was previously believed that TBGH were small, multiple, sometimes bilateral and located in the zone of lentiform nucleus and external capsule, whereas spontaneous hemorrhages were large solitary, large causing mass effect and in the region of thalamus and internal capsule. A patient suffering from a head injury and having a lesion of the latter type was considered to be due to a spontaneous bleed causing the event.^[7]

Later on, in a patient of fatal severe head injury, Mosberg *et al.*^[8] at autopsy confirmed a massive hematoma in the pallidum and a ruptured twig of the anterior choroidal artery. After histopathology, the arterial tear was confirmed to be traumatic. Maki *et al.*^[9] (1980), reported TBGH, where the mechanism was supposed to be anterior stretch of the lateral branch of the perforator of the middle cerebral artery. Kinoshita *et al.*^[10] (2008) have also postulated anterior choroidal artery causing basal ganglia bleed. Fujioka *et al.*^[11] have demonstrated that the traumatic dissection of the middle cerebral artery causing hemorrhage and infraction in the basal ganglia and thalamus.

In both our patients, there was a clear cut history of trauma, and no evidence, historically, clinically, and radiologically to suggest any other cause of this lesion, thereby confirming it to be TBGH. However, in the second case, the age may favor hypertensive bleed, but his blood pressure had been normal during his inpatient stay.

It has been postulated that if trauma occurs while the head is in motion, and the impact sufficient to deform the skull is applied to the vertex, forehead or occipital area and directed to the tentorium, it shifts the brain through the tentorial notch, producing shearing forces leading to the damage to the vessels described above. Both coup and countercoup injuries can cause this and this may cause bilateral lesions.^[11]

TBGH may be classified as large if more than 2 cm in diameter or small if less than 2 cm in diameter.^[1] Small lesions in the basal ganglia have also been described as a part of diffuse axonal injury. However, the mechanism is shearing strain on

the axons and it also involves the cerebral white matter, corpus callosum, and upper brainstem.^[12,13]

Treatment is based on protocol as for intracranial hematoma taking into account the neurological status, presence of mass effect and response to other means of controlling ICP. Treatment options for TBGH include conservative, open surgery, CT guided stereotactic or ultrasound guided aspiration.^[2] Boto *et al.*^[2] recommend surgical evacuation for all lesions having a volume greater than 25 ml. In the series by Kumar *et al.*^[6] all the patients were managed conservatively (average volume 13.2 ml). However, all patients had a volume less than 25 ml (maximum 23ml). In the case of bilateral TBGH by Jang *et al.*^[3] the patient had a GCS of 15 and hence was conservatively managed.

Outcome of TBGH has been found to be variable. Amongst 37 patients studied by Boto *et al.*^[2] 59% died, 5 % were vegetative, 19% experienced severe disabilities, and 16% made a favorable recovery. In contrast, in a study of 10 patients by Kumar *et al.*^[6] all patients had a favorable outcome (Glasgow Outcome Score of 4 or 5) and mortality was nil. Katz *et al.*^[14] Kimura *et al.*^[15] Lee *et al.*^[16] Jang *et al.*^[3] have also reported good prognosis for TBGH. Large size, associated coagulation disorders, DAI, presence of other bleeds like intraventricular of brain stem hemorrhage, age greater than 60, abnormal pupillary response, abnormal motor response to pain, and severe head injury are reported to be indicators for poor prognosis.^[2,5,6,16]

In both our patients, the post resuscitation GCS was 4 indicating severe head injury. The first patient did not regain consciousness at 45 days post injury. The patient improved on conservative management; hence surgery was not contemplated. In the second patient also, GCS improved to localizing (M5) on conservative management; however, he did not regain consciousness.

Conclusion

Bilateral traumatic basal ganglia bleed is rare in clinical literature. However, basal ganglia bleed should be recognized as a definitive entity. It can be managed conservatively as evacuation involves approaching through the internal capsule which may be further detrimental. Prognosis is variable and dependant on many factors.

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