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CASE REPORT

Traumatic bilateral basal ganglia bleed: A report of rare two cases and review of the literature

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ABSTRACT

Traumatic basal ganglia hemorrhage (TBGH) 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. The general incidence of TBGH is reported between 2.4% and 3% of closed head injury. However, the incidence is higher in postmortem studies (9.8%). Bilateral traumatic basal ganglia hematoma is extremely rare. Descriptions are limited to case reports.

Key words: Basal ganglia, bilateral, hemorrhage, trauma

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.[1-3] An incidence of 3–10% of head injuries has been reported.[1,3,4] The incidence is higher in autopsy series when compared with clinical studies.[1,3] Bilateral basal ganglia hematoma after trauma is extremely rare and is limited to case reports.[4,5] The mechanism is unclear though it is proposed to arise from shear strain of the lenticulostriate or anterior choroid vessels caused by acceleration/deceleration forces at the time of injury.[1,6] 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 nontraumatic cause. Thus, the recognition of TBGH is of prime relevance.

Case Reports

Case 1

A 20-year-old male met with a road traffic accident. He was admitted in neurosurgery ward in unconscious state. There is a history of loss of consciousness, and there was no history of ear bleed, nose bleed, convulsion, and cerebrospinal fluid (CSF) leak. There was no history of significant illness, substance abuse, or any history suggestive of any bleeding disorder. The patient was nondiabetic and nonhypertensive. Before the accident, he was not on any medication. On examinations, multiple abrasions over face and right leg were noted. His Glasgow Coma Scale (GCS) was E1V3M5. The pupils were 2 mm bilaterally, reacting to light. Bilateral limb movements were equal. A plain computerized tomogram (CT) showed deep intracerebral bleed [Figure 1] in the basal ganglia involving the corona radiata on the right side (size approximately 11 mm by 14 mm) and left side (size approximately 18 mm by 14 mm). Sulcal spaces and basal cisterns were normal and no midline shift. All blood parameters and radiology including clotting profile were within normal limits. Antibiotic and phenytoin sodium was given. On the 2nd day of admission, the patient became conscious and oriented and GCS was 15/15, and then the patient was discharged on the 3rd day of admission. At the time of discharge, no focal neurological deficit was noted. On three months of follow-up, the patient was alright without any neurological deficit, GCS 15/15, and advised repeat noncontrast CT (NCCT) head but refused for same.

Case 2

A 45-year-old male met with a road traffic accident when he was riding a motor cycle and was hit by another motor cycle. History

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of loss of consciousness was present and nose bleed was present; there was no history of ear bleed, convulsion vomiting, and CSF leak. There was no history of significant illness, substance abuse, or any history suggestive of any bleeding disorder. The patient was nondiabetic and nonhypertensive. Before the accident, he was not on any medication. On examination, lacerated wound of lower lip and right dorsum of foot was noted. His GCS was E2V3M5. The pupils were 2 mm bilaterally, reacting to light. Bilateral limb movements were equal. A plain CT showed intracerebral bleed [Figure 2] in the basal ganglia involving the corona radiate and capsuloganglionic region on the right (approximately 22 mm by 17 mm) and left capsuloganglionic region (approximately 3 mm by 2 mm), and multiple hemorrhagic contusions are seen right temporal lobe. Sulcal spaces and basal cisterns were normal and no midline shift. All blood parameters and radiology including clotting profile were within normal limits. Primary suturing of wound and antibiotic and phenytoin sodium was given. On the 2nd day of admission, the patient became conscious, oriented and GCS was 15/15, and then the patient was discharged on the 5th day of admission. At the time of discharge, no focal neurological deficit was noted. On three months of follow-up, the patient was alright without any neurological deficit, GCS 15/15, and advised repeat NCCT head, but the patient had not come in further follow-up with repeat CT.

Discussion

The general incidence of TBGH is reported between 2.4% and 3%[1,3,7] of closed head injury. However, the incidence is higher in postmortem studies (9.8%).[1,3] Bilateral traumatic basal ganglia hematoma is extremely rare. Descriptions are limited to case reports.[1,2,4,5] However, Kumar *et al*. found one patient with bilateral basal ganglia bleed among 10 cases of TBGH (10%).[8]

It was previously believed that TBGH was small, multiple, sometimes bilateral, and located in the zone of lentiform nucleus and external capsule, whereas spontaneous hemorrhages were large solitary, causing mass effect. A patient suffering from a head injury and having a lesion of the latter type was considered due to a spontaneous bleed causing the event.[9]

Later on, in a patient of fatal severe head injury, Mosberg and Lindenberg[10] at autopsy confirmed a massive hematoma in the palladium and a ruptured twig of the anterior choroidal artery. After histopathology, the arterial tear was confirmed to be traumatic. Maki *et al*.[11] reported TBGH where the mechanism was supposed to be anterior stretch of the lateral branch of the perforator of the middle cerebral artery.

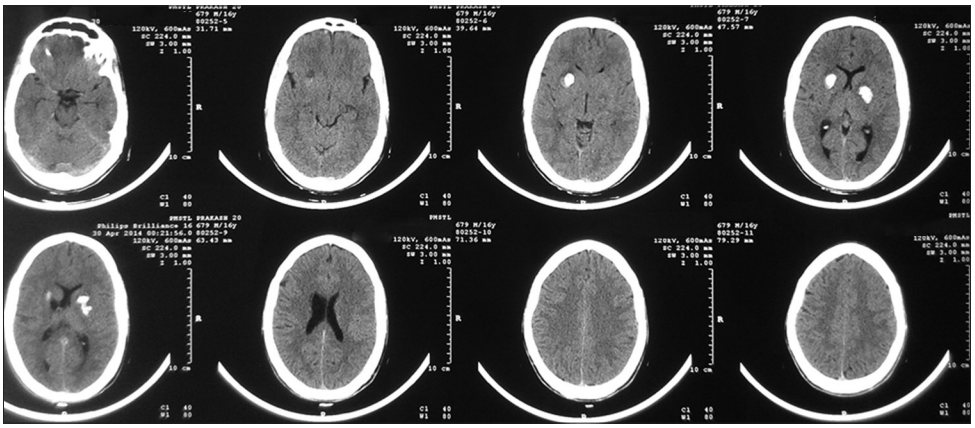

Figure 1: Noncontrast computerized tomogram head showed deep intracerebral bleed in bilateral basal ganglia involving coranaradiata in right and left

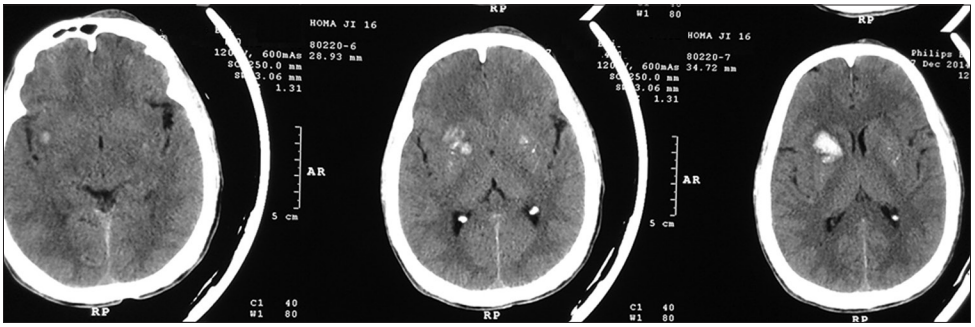

Figure 2: Noncontrast computerized tomogram head showed intracerebral bleed in bilateral basal ganglia involving corona radiate and capsuloganglionic region on the right and left capsuloganglionic region

Kinoshita *et al.*[12] have also postulated anterior choroidal artery causing basal ganglia bleed. Fujioka *et al.*[13] 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.[13]

TBGH may be classified as large if more than 2 cm in diameter or small if <2 cm in diameter.[1,3] Small lesions in the basal ganglia have also been described as a part of diffuse axonal injury (DAI). However, the mechanism is shearing strain on the axons, and it also involves the cerebral white mater, corpus callosum, and upper brainstem.[14,15]

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 intracranial pressure. Treatment options for TBGH include conservative, open surgery, CT guided stereotactic or ultrasound guided aspiration.[6] Boto *et al.*[6] recommend surgical evacuation for all lesions having a volume >25 ml. In the series by Kumar *et al.*[8] all the patients were managed conservatively (average volume 13.2 ml). However, all patients had a volume <25 ml (maximum 23 ml). In the case of bilateral TBGH by Jang *et al.*[4] the patient had a GCS of 15 and hence was conservatively managed.

The outcome of TBGH has been found to be variable. Amongst 37 patients studied by Boto *et al.*[6] 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.*[8] all patients had a favorable outcome (Glasgow Outcome Score of 4 or 5) and mortality was nil. Katz *et al.*[7] Kimura *et al.*,[16] Lee and Wang[17] Jang *et al.*[4] have also reported good prognosis for TBGH. Large size, associated coagulation disorders, DAI, presence of other bleeds like intraventricular of brain stem hemorrhage, age >60, abnormal pupillary response, abnormal motor response to pain, and severe head injury are reported to be indicators for poor prognosis.[3,7,8,17]

Conclusion

TBGH is rarely reported and bilateral BGH is very rare entity only few cases reported in world's literature. It is compatible with a favorable recovery if not associated with damage to other cortical and subcortical structures and present in isolation. BGHs seem to be hemorrhagic contusions as a result of a shearing injury due to high-velocity impact. It can be managed conservatively. Prognosis is variable and dependent on many factors.

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Conflicts of interest
There are no conflicts of interest.

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