Bilateral Traumatic Basal Ganglia Hemorrhage Associated with Diffuse Axonal Injury: A Report on Two New Cases and Review of Existing Literature

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Abstract

Traumatic basal ganglia hemorrhage (TBGH) refers to development of intraparenchymal bleed with in the basal ganglia, internal capsule, or thalamus. Though the reported incidence of TBGH is approximately 3% of all closed-head injuries, bilateral TBGH is much rarer, with only 13 cases reported to date. We present two additional cases of bilateral TBGH posttrauma, along with a review of the existing literature.

Keywords

- bilateral basal ganglia hemorrhage
- diffuse axonal injury
- trauma

Introduction

Traumatic basal ganglia hemorrhage (TBGH) is an uncommon manifestation of head injury and is seen in approximately 3% of patients with severe closed-head injury.[1–5] The reported incidence in autopsy series is higher, reaching up to 9 to 12%, likely reflecting a higher associated mortality.[2,4,6,7] In most cases, the hemorrhage is unilateral and cases of bilateral TBGH are extremely rare.[4,8,9] To date, only 13 cases have been reported in the literature (►**Table 1**).[1–4,6–12]

TBGH is considered to be an intermediary contusion, as it involves the deep nuclei and is located between the coup and contrecoup contusions.[2,3,9] It is classified as "large" if the maximal dimension exceeds 2 cm and "small" if the size is less than 2 cm.[13]

TBGH often coexists with other intra- or extra-axial injuries and rarely as an isolated finding. Prognosis depends on the extent and severity of other injuries and varies from good neurologic recovery in isolated cases to severe morbidity, persistent vegetative state, or even death in complex cases.

Case Report 1

A 16-year-old boy was brought to the emergency room after he crashed his car while driving at high speed. As per the paramedic report, the patient was unconscious at the scene and was pulled out of the car. His initial Glasgow coma scale (GCS) score was 4, and he was noted to be in decerebrate posture. The patient was intubated and received mannitol and 3% saline en route.

The patient was immediately brought to Radiology after initial triage in the emergency department. Computed tomography (CT) of the brain was done and revealed bilateral basal ganglia hematomas (►**Fig. 1**). The larger hematoma on the left had a volume of 18 cc whereas the right-sided hematoma measured 3.5 cc. The right-sided hematoma, however, showed intraventricular extension with consequent early obstructive hydrocephalus. In addition, the patient had multiple punctate foci of hemorrhage/diffuse axonal injury (DAI) involving the gray-white junction bilaterally, along the corpus callosum and the ventral midbrain in the midline (►**Fig. 2**). Scattered minor subarachnoid hemorrhage over the bilateral cerebral convexities was also present, along with mild midline shift toward the right side. CT of the cervical, thoracic, and lumbar spine as well as of the chest, abdomen, and pelvis was negative for injury.

The patient immediately underwent ventricular drain placement. However, a CT study obtained 4 hours postoperatively revealed progressive increase in the size of the basal ganglia and intraventricular hematoma. Despite adequate circulatory and respiratory support, the patient

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Table 1 Summary of reported cases of bilateral TBGH

Author/year	Age/sex	Mechanism of trauma	Associated findings	Outcome
Yanaka et al, 1991	17/M	RTA	SAH, DAI	NA
	75/M	Fall from height	None	No residual neurologic deficit
Jang et al, 2007	50/M	Fall from height	SAH, occipital fracture	No residual neurologic deficit
Kumar et al, 2008	35/F	RTA	DAI	Moderate disability
Kushal et al, 2011	42/M	RTA	None	Quadriparesis, worse on left
Aygün et al, 2012	35/M	Rocket explosion	None	No residual neurologic deficit
Bhargava et al, 2012	25/M	RTA	Left temporal bone fracture and hemotympanum, right pneumothorax	Dysphagia, right spastic hemiparesis
	50/M	RTA	Intraventricular extension of hemorrhage	NA
Jain et al, 2013	38/M	Fall from height	Right occipital EDH	Minimal residual deficits
Gupta et al, 2014	50/F	RTA	Bilateral SDH	Left hemiparesis
Calderon-Miranda et al, 2014	28/M	RTA	Right parietal EDH	Dead
Pandey et al, 2014	37/M	RTA	Left SDH, SAH, punctate hemorrhages in corpus callosum	GCS 7
Vega et al, 2015	57/M	Assault	Left temporal hemorrhagic contusions, IVH, SAH, left parietal fracture	Cognitive impairment
Present case 1	16/M	RTA	DAI, IVH, SAH	Died
Present case 2	22/M	RTA	DAI, IVH, SDH	Died

Abbreviations: DAI, diffuse axonal injury; EDH, extradural hemorrhage; GCS, Glasgow coma scale; IVH, intraventricular hemorrhage; RTA, road traffic accident; SAH, subarachnoid hemorrhage; SDH, subdural hemorrhage; TBGH, traumatic basal ganglia hemorrhage.

continued to deteriorate clinically with the GCS ranging between 3 and 6 and the intracranial pressure between 20 and 100 cm of water. On day 4, the sedation was withdrawn without any change in the clinical status. Bedside electroencephalogram (EEG) revealed diffuse background abnormalities consistent with severe encephalopathy. Neurologic examination was consistent with brain death, which was subsequently confirmed with technetium-99m (^{99m}Tc) HMPAO perfusion scan. The patient died on day 6 of admission.

Case Report 2

A 22-year-old man was brought to the emergency department after being involved in a high-speed motor vehicle injury. GCS at the scene was 3 and the patient required intubation. CT of the brain was performed and revealed foci of hemorrhage in the right lentiform nucleus, measuring less than 2 cm (►**Fig. 3**). There were multiple punctate foci of hemorrhage/DAI involving the gray-white junction over the bilateral cerebral hemispheres and right cerebellar hemisphere, intraventricular hemorrhage, minor subarachnoid hemorrhage over the right frontal convexity, and a tiny tentorial subdural hemorrhage. In addition, the patient had multiple extracranial injuries including bilateral pulmonary contusions and pneumothoraces, a three-column fracture of the T-10 vertebral body, and bilateral femoral fractures.

The patient was managed with bilateral chest tubes and external fixation of the femoral fractures. An intracranial pressure monitor was placed. He also required respiratory and cardiovascular support but failed to improve neurologically. Magnetic resonance imaging (MRI) of the brain on day 4 of admission confirmed extensive DAI, with involvement of the cerebral hemispheres, corpus callosum, and brainstem (►**Fig. 4**). Mild increase in the right TBGH was noted along with multiple punctate foci of hemorrhage in the left basal ganglia, thus resulting in the diagnosis of bilateral TBGH (►**Fig. 5**). Given the poor neurologic status, failure of clinical improvement, and extensive injury on imaging, the patient was placed on palliative care after discussions with the family and died on day 6 of admission.

Discussion

TBGH is an uncommon manifestation of traumatic brain injury (TBI) but is being more frequently recognized in the post scan era.[6,7,9] It is usually unilateral, with bilateral TBGH being extremely rare. In the limited number of reported studies, the most common etiology is road traffic accident (RTA), followed by fall from a height and assault. On imaging, a cutoff of 2 cm in maximal dimension is arbitrarily used to differentiate small from large lesions. Smaller lesions appear to be more commonly associated with DAI.[1,9] Clinical presentation is

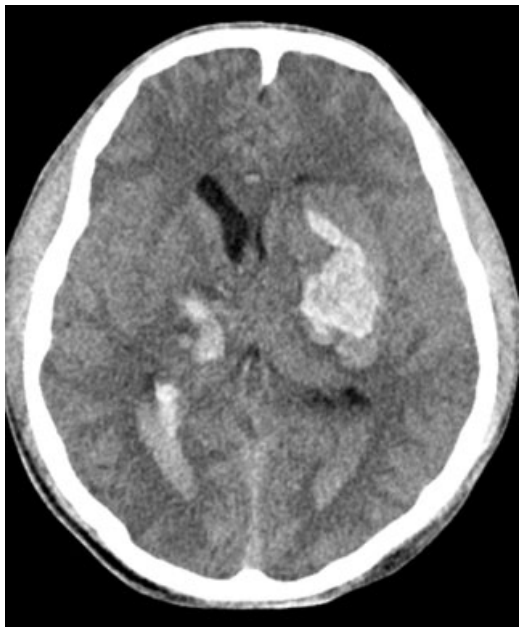

Fig. 1 Axial CT image at the level of basal ganglia reveals bilateral parenchymal hematomas and intraventricular hemorrhage. There is mass effect on the left lateral ventricle.

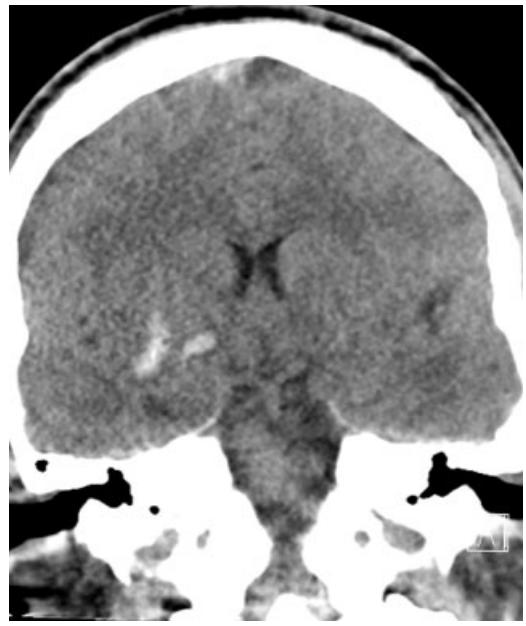

Fig. 3 Coronal MPR image from case 2 reveals foci of basal ganglia hemorrhage on the right. Small subarachnoid hemorrhage is also seen in the right para-falcine region. Note that the left basal ganglion appears unremarkable on CT.

dependent on the severity of TBI, with most patients either comatose or with low GCS.[1] In some cases, patients may have headache or hemiparesis. Rarely, the patient may be asymptomatic.

Pathophysiologically, TBGH is thought to result from shear injury to the lenticulostriate perforators or the anterior choroidal artery (AChA).[3,8,10] In fact, Mosberg and Lindenberg showed evidence of vascular laceration involving the palladial branch of AChA in their patient on autopsy.[14] Hemorrhage within the putamen may also result from traumatic injury to the middle cerebral artery, as shown by Fujioka et al.[15] It has also been shown on biomechanical studies that a direct impact to the vertex, forehead, or occiput in the direction of the tentorium results in displacement of the brain through the tentorial notch, resulting in stretching or tear of the palladial branches of AChA.[1–3,8] The hemorrhage often begins in the putamen or external capsule and may be bilateral.[1,7,9] This is in contrast to the hypertensive

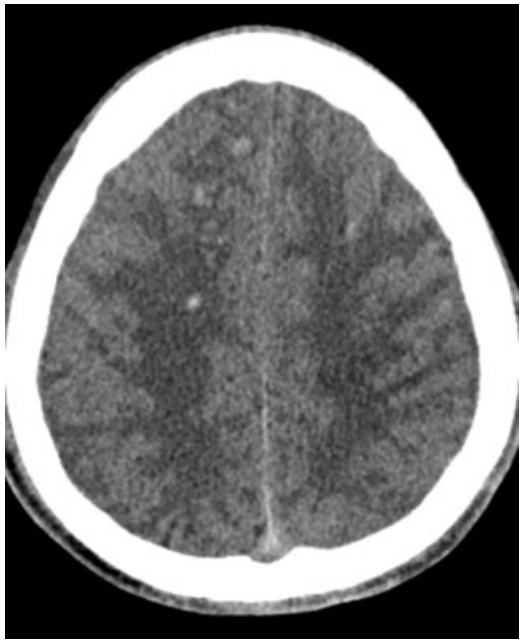

Fig. 2 Axial CT image at the level of centrum semiovale reveals multiple punctate foci of hemorrhage involving the gray-white junction in the frontal regions bilaterally, consistent with DAI.

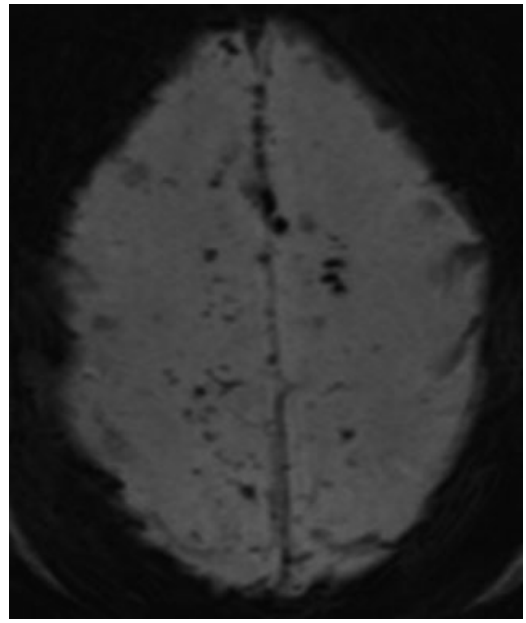

Fig. 4 Axial SWI images at the level of the centrum semiovale shows extensive foci of hemorrhage involving the gray-white junction in frontal and parietal lobes bilaterally.

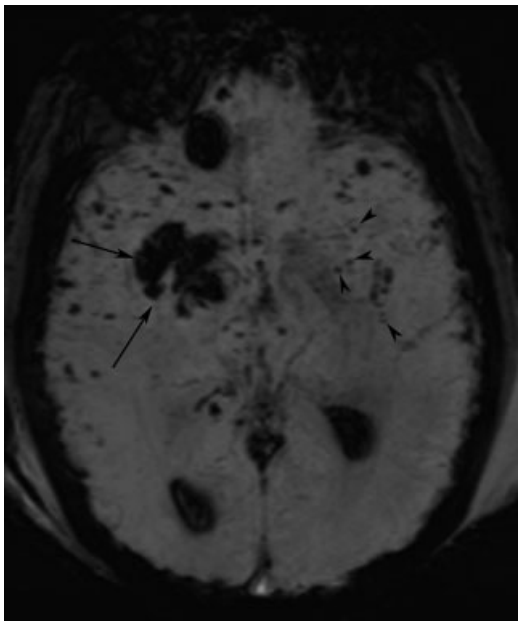

Fig. 5 SWI image at the level of basal ganglia shows right basal ganglia hemorrhage (arrows). In addition, there are multiple punctate foci of hemorrhage involving the left basal ganglia (arrowheads), consistent with DAI.

hemorrhage that often starts in the internal capsule or thalamus.

On imaging, TBGH is hyperdense on CT in the acute phase, an expected appearance of acute intraparenchymal hematoma. The parenchymal hematoma varies in size and may dissect in to the ventricular system. Other manifestations of TBI may be present, such as subarachnoid, subdural, or epidural hemorrhage, DAI, or fractures.

In both these cases, the patients had imaging findings consistent with DAI, with hemorrhagic lesions involving the bilateral gray-white junction, corpus callosum, and brainstem. In both patients, however, punctate hemorrhage was present in the midline ventral midbrain, in contrast to the involvement of the dorsolateral midbrain, which is typically described in DAI.

Including the present cases, DAI has been reported in 5 of the 15 cases of bilateral TBGH, with an incidence of approximately 33%. Interestingly, patients with coexisting DAI were younger (mean age of 25 years) as compared with those without DAI (45 years). This is similar to the relative DAI frequency of 30% reported by Kumar et al in their series of 10 patients where all but one patient had unilateral TBGH.[6] The previously reported high incidence of DAI described by Katz et al may have been an overestimation since they diagnosed DAI based on clinical criteria of loss of consciousness on impact and most of the patient did not have imaging till a few weeks later.[5] Similarly, Boto et al reported a DAI prevalence of 73% on imaging in their series on TBGH.[16] However, in their study patients with a GCS of more than 8 were excluded and the sample size therefore only consisted of patients with severe TBI, thereby inducing sampling bias and overestimating the true prevalence of DAI in TBGH.

Nevertheless, a 33% incidence of DAI in patients with TBGH is still relatively high and is likely a reflection of the similar pathophysiologic mechanism of injury, since both are felt to be secondary to shear strain from rapid acceleration or deceleration. Adams et al showed a statistically higher incidence of basal ganglia hemorrhage in patients who had DAI as compared with those without DAI.[13,17] Shimura et al described autopsy findings in five cases of DAI and felt that TBGH was likely a form of DAI.[18] This is in fact supported by our second case that showed TBGH on the right side and multiple punctate foci of hemorrhage in the left basal ganglia, consistent with DAI. Ours is the first case of TBGH where susceptibility-weighted imaging (SWI) was performed. SWI is known to be much more sensitive to hemorrhage as compared with CT or even conventional MRI. In our case, the SWI provides great insight into the relationship between DAI and TBGH, further strengthening the argument that TBGH is possibly a part of the DAI spectrum.

Interestingly, studies in nonhuman primate models of TBI have previously shown that acceleration in the coronal plane produces the most severe DAI, when compared with a similar force in other anatomical planes.[17] This is coincidentally similar to the pathomechanism of TBGH that involves a force to the vertex or occiput, directed toward the tentorium. It is therefore conceivable that the basal ganglion is only involved in the most severe cases of DAI, reflecting injury severe enough to damage both the axons and blood vessels. These patients would therefore be expected to have poor prognosis, a finding also noted in the reported literature. Of the five reported patients with bilateral TBGH and DAI on CT, two died (present cases), one had moderate disability, and another had a GCS of 7 while the final outcome of the last case was unclear.

Treatment options in patients with TBGH vary from conservative management in isolated cases to image-guided aspiration or craniotomy with hematoma evacuation.[8,9] Not unexpectedly, the final prognosis in TBGH is strongly correlated to presence of DAI, even when the bleed is unilateral.[1,2,9] Besides absence of DAI, good prognostic factors in TBGH include young age (< 60 years), an isolated small lesion (< 2 cm), and a GCS of greater than 9 at admission. Poor prognostic factors include a GCS of less than 7, longer duration of coma, volume of TBGH greater than 25 mL, persistently raised intracranial pressure, old age, larger lesions, delayed hematoma expansion, and coexisting injuries.[1,3,8,9,11]

Conclusion

Bilateral TGBH is an extremely rare occurrence in the setting of TBI, and it likely reflects severe injury, especially when associated with DAI on imaging. The relatively high incidence of DAI in TBGH possibly stems from a shared pathophysiology, an argument supported by our cases. However, the exact relationship between the two entities is unclear. It is conceivable that TBGH likely reflects a part of the DAI spectrum, being seen only in cases with severe injury and a specific injury mechanism.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

1 Vega MB, Hamamoto Filho PT, Machado CJ, Zanini MA. Traumatic brain injury presenting with bilateral basal ganglia hemorrhage. Neurol Neurochir Pol 2015;49(6):456–459
2 Jang KJ, Jwa CS, Kim KH, Kang JK. Bilateral traumatic hemorrhage of the basal ganglia. J Korean Neurosurg Soc 2007;41(4):272–274
3 Pandey N, Mahapatra A, Singh PK. Bilateral large traumatic hemorrhage of the basal ganglion. Asian J Neurosurg 2014;9(4): 240
4 Calderon-Miranda WG, Alvis-Miranda HR, Alcala-Cerra G, Rubiano AM, Moscote-Salazar LR. Bilateral traumatic basal ganglia hemorrhage associated with epidural hematoma: case report and literature review. Bull Emerg Trauma 2014;2(3): 130–132
5 Katz DI, Alexander MP, Seliger GM, Bellas DN. Traumatic basal ganglia hemorrhage: clinicopathologic features and outcome. Neurology 1989;39(7):897–904
6 Kumar S, Jha D, Abbey P, Mishra V, Handa A. Outcome of traumatic basal ganglia hemorrhage. Internet J Neurosurg 2008;6:1
7 Kaushal R, Kataria R, Gupta A, Sinha VD. Traumatic bilateral (mirror image) basal ganglia bleed. Indian J Neurotrauma 2011; 8:109–110
8 Bhargava P, Grewal SS, Gupta B, Jain V, Sobti H. Traumatic bilateral basal ganglia hematoma: a report of two cases. Asian J Neurosurg 2012;7(3):147–150
9 Jain SK, Sundar IV, Sharma V, Prasanna KL, Kulwal G, Tiwari RN. Bilateral large traumatic basal ganglia haemorrhage in a conscious adult: a rare case report. Brain Inj 2013;27(4):500–503
10 Aygün FM, Aygün MS, Onal MB, Demirci OL. Isolated basal ganglia hemorrhage due to blast injury. Ulus Travma Acil Cerrahi Derg 2012;18(5):461–462
11 Gupta P, Mittal RS, Purohit D, Shekhawat JS, Meena US. Bilateral basal ganglia hemorrhage following motor vehicle accident: a case report. Romanian Neurosurg 2014;XXI 2:224–226
12 Yanaka K, Egashira T, Maki Y, et al. [Bilateral traumatic hemorrhage in the basal ganglia: report of two cases]. No Shinkei Geka 1991;19(4):369–373
13 Adams JH, Doyle D, Graham DI, Lawrence AE, McLellan DR. Deep intracerebral (basal ganglia) haematomas in fatal non-missile head injury in man. J Neurol Neurosurg Psychiatry 1986;49(9): 1039–1043
14 Mosberg WH Jr, Lindenberg R. Traumatic hemorrhage from the anterior choroidal artery. J Neurosurg 1959;16(2):209–221
15 Fujioka M, Maeda Y, Okuchi K, Kagoshima T, Taoka T. Secondary change in the substantia nigra induced by incomplete infarct and minor hemorrhage in the basal ganglia due to traumatic middle cerebral arterial dissection. Stroke 1999;30(9):1975–1977
16 Boto GR, Lobato RD, Rivas JJ, Gomez PA, de la Lama A, Lagares A. Basal ganglia hematomas in severely head injured patients: clinicoradiological analysis of 37 cases. J Neurosurg 2001;94(2): 224–232
17 Adams JH, Doyle D, Ford I, Gennarelli TA, Graham DI, McLellan DR. Diffuse axonal injury in head injury: definition, diagnosis and grading. Histopathology 1989;15(1):49–59
18 Shimura T, Nakazawa S, Kobayashi S, Yokota H, Otsuka T, Nakamura T. [Clinicopathological studies of diffuse axonal injury—five autopsy cases]. No Shinkei Geka 1988;16(5, Suppl) 647–653