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Original Article

Clinical outcomes and prognostic factors of traumatic basal ganglia hematomas: A 4-year single-center study

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ABSTRACT

Background: Traumatic basal ganglia hematomas (TBGH) are rare entities. They are situated in the deep cerebral parenchyma and have also been termed as intermediate coup contusions. Available literature is sparse with regards to the characteristics and prognosis of TBGH. We aim to share our experience in the management, outcomes, and prognostic factors of TBGH.

Methods: A 4-year retrospective study which included all cases of TBGH, except dot contusions (<2 mL) and those with coagulopathies. Admission variables were correlated with Glasgow Outcome Scale score at discharge and 12 months.

Results: Thirty-two patients were analyzed. The mean age was 39.2 years. Two-thirds were due to road traffic accidents. Around 60% were severe head injuries. The mean Glasgow coma scale (GCS) score at presentation was 8.5. Twenty patients had moderate-to-severe hemiparesis. The mean hematoma volume was 18.1 mL. Associated traumatic intracranial lesions were seen in 28 cases. Only 7 patients (22%) underwent surgery. The mean follow-up was 17.4 months (range 14–34 months). The mortality rate was 12.5% (n = 4). Among the survivors, only 39% (n = 11) had good outcomes at discharge which showed modest improvement to 54% (n = 15) at 12 months.

Conclusion: Our study noted that poor admission GCS scores, poor motor response, presence of significant hemiparesis, and larger hematoma volumes (>20 mL) correlated with poor outcomes at 12 months. The overall outcomes have been mostly unfavorable as observed in majority of studies due to deeper location of these hematomas, high proportion of severe head injuries, and high proportion of residual weakness in survivors.

Keywords: Basal ganglia hematomas, Glasgow outcome scale, Intermediate contusions, Outcome and prognosis, Traumatic

INTRODUCTION

In the subset of trauma patients, traumatic brain injury (TBI) is the leading cause of death and disability and the majority of this burden is observed in low- and middle-income countries.[14,18] Among the various morphological TBI lesions such as extradural hematomas (EDH), subdural hematomas (SDH), diffuse axonal injury (DAI) and contusions, and traumatic basal ganglia hematomas (TBGH) are very rare entities. They form a distinct subgroup of traumatic lesions, which were described in detail by Adams *et al.* in 1986.[1] TBGH are defined as intracerebral

hemorrhages located in the region of basal ganglia (caudate nucleus, putamen and globus pallidus) and neighboring structures like thalamus and internal capsule.[1,3] Since they are situated in the deep brain parenchyma between coup and contrecoup contusions, they have also been termed as intermediate coup contusions or intermediary contusions. They account for <2% of post traumatic intracerebral contusions and are mostly encountered in high-velocity trauma such as road traffic accidents (RTA).[3] Literature is sparse with very few large case series available, with regards to the characteristics, mechanisms of injury and the overall prognosis of these traumatic lesions. In this study, we aim to analyze the clinicoradiological characteristics and outcomes of TBGH who were managed at our tertiary care hospital.

MATERIALS AND METHODS

This was a single-center 4-year retrospective study conducted between January 2015 and December 2018 at Kasturba Medical College, Manipal, India.

Inclusion criteria

Patients with basal ganglia region hemorrhage noted on computed tomography scan and an unequivocal history of trauma (RTA, fall from height, and assault) were included in the study.

Exclusion criteria

The following criteria were excluded from the study:

1) Patients with spontaneous basal ganglia hematomas, such as due to hypertension or coagulation abnormalities
2) Absent brainstem reflexes on arrival
3) Dot-like contusions with volume <2 mL.

Patients' data were retrieved through case sheets, operative records, discharge summaries, outpatient clinic notes, and telephonic conversations. The following variables were noted for analysis: age, sex, admission Glasgow Coma Scale (GCS) score (mild: 13–15, moderate: 9–12, and severe: <8), mechanism of injury, radiological findings (EDH/SDH/DAI/Intraventricular haemorrhage [IVH]), type of management, follow-up, and outcomes. Patients of severe head injury, with signs of raised intracranial pressure (ICP), and not responding to initial conservative measures underwent surgical evacuation. All patients were regularly followed up on an outpatient basis, at 1 month, 3–6 months, and 12 months after discharge. Outcomes were assessed using the Glasgow Outcome Scale (GOS) score at discharge and follow-up. We classified the GOS scores into good (good recovery and moderate disability) and poor outcomes (severe disability, vegetative state, or death).

Statistical analysis

Statistical analyses were carried out using SPSS software version 21.0. Qualitative variables were presented as frequency and percentages. Quantitative variables were presented as mean (standard deviation) or median (range) depending on the distribution of data. All admission variables were correlated with GOS score at discharge and at 12-month follow-up. Age and hematoma volumes were dichotomized based on the mean values. Chi-square test was used to perform the statistical analysis. $P < 0.05$ was considered statistically significant.

RESULTS

After exclusion, 32 patients were analyzed. The mean and median ages were 39.2 years (standard deviation [SD] ± 18.7) and 39 years (range 7–79 years), respectively. Half of them were below 40 years. The male: female ratio was 9.6:1 (29:3). Two-thirds were due to RTA ($n = 24$). There were 19 severe, seve moderate, and six mild head injuries. The mean GCS at presentation was 8.5 (±3.5 SD) and the median GCS was 7.5. Eight patients had presented with nasal/ear bleed and three patients had seizures at presentation. Asymmetrical and nonreactive pupils were noted in 9 (28%) cases. Twenty patients (62%) had moderate-to-severe hemiparesis.

The mean hematoma volume was 18.1 mL and the median volume was 8.6 mL (range, 2.5–70 mL). There were 21 (65%) left-sided, 9 (28%) right-sided, and two bilateral hematomas. IVH was present in 20 cases (62%). Associated traumatic intracranial lesions (EDH, SDH, contusions, DAI, and subarachnoid hemorrhage [SAH]) were seen in 28 cases (87%). Most of the patients ($n = 25$) (78%) were managed conservatively and only 7 patients (22%) underwent surgery.

Table 1 summarizes the demographical data of the cohort.

Follow-up and outcomes

The mean follow-up was 17.4 months (range 14–34 months) and all patients had a minimum of 12-month FU. The mortality rate was 12.5% ($n = 4$) and all were severe head injuries. Among the survivors, 61% ($n = 17$) patients had poor outcomes and 39% had good outcomes at discharge. At 1-year follow-up, there was a modest increase in the proportion of good outcomes (GOS 4 and 5), from 39% ($n = 11$) to 54% ($n = 15$). Of the 20 patients with hemiparesis, 12 had residual weakness at 1-year follow-up.

We tried to correlate various variables with patient outcomes, at discharge and 12 months. At discharge, the following variables had statistically significant association with poor outcomes, namely, poor admission GCS score ($P < 0.001$), poor motor response component of GCS ($P < 0.001$), pupil nonreactivity ($P = 0.015$), presence of hemiparesis

(P = 0.024), and hematoma volume >20 mL (P = 0.016). At 12 months, except pupillary asymmetry, all the other variables were significantly associated with poor outcome namely, admission GCS score (P = 0.003), GCS motor component (P = 0.01), hematoma volume (P = 0.01), and paresis (P = 0.028). The remaining variables such as age, gender, presence of ENT bleed, laterality, associated CT findings, presence of IVH, and mode of management did not show statistical association with outcomes.

Table 2 summarizes the statistical results.

Table 1: The characteristics of the study cohort.

Characteristic/variable	*n* (%)
Total number	32
M: F	29:3 (9.7:1)
Mean age±SD	39.2±18.7 years
Median age	39.5 years
ENT bleed	
Yes	8
No	24
GCS score	
3–8	19
9–12	7
13–15	6
Mean GCS score ±SD	8.5±3.9
Median GCS score	7.5
Mode of injury	
RTA	24
Fall	8
Laterality	
Right	10
Left	21
Bilateral	1
Anisocoria	
Yes	9
No	23
Hemiparesis	
Yes	20
No	12
Volume of hematoma	
<20 mL	19
>20 mL	13
IVH	
Yes	18
No	14
Other CT findings	
Yes	28
No	4
Management	
Conservative	25
Surgery	7
GOS discharge	
Good	11 (39)
Poor	17 (61)
Dead	4 (12.5)
GOS 12 months*	
Good	15 (54)
Poor	13 (46)

*GOS at 12 months was calculated for 28 survivors (excluded four patients who died). M: Male, F: Female, ENT: Ear nose throat, GCS: Glasgow coma scale, RTA: Road traffic accident, IVH: Intraventricular hemorrhage, CT: Computed tomography, GOS: Glasgow outcome scale, SD: Standard deviation, *n*: Number

DISCUSSION

In clinical studies, the incidence of TBGH has been observed to be around 2–3% of closed head-injured patients; however, autopsy series indicate a higher incidence, ranging between 10% and 12%.[3,8,11,15] They have been defined as hemorrhages involving the striatum, pallidum, and thalamus.[1,3] This was earlier included in the spectrum of DAI; however, Adams *et al*. described it as a distinct entity with well-defined etiopathogenesis.[1] They are considered as both hemorrhagic contusions and hematomas with both the terminologies often being used interchangeably. Very few studies have been reported in the literature with regards to the clinicoradiological features and outcomes of TBGH.[3,5,7,10-12,15,19]

Pathophysiology of injury

Multiple theories have been proposed to elucidate the pathophysiology of such lesions. Mosberg and Linberg observed that shearing forces are produced when sufficient impact directed toward the tentorium is delivered to the vertex/forehead/occipital area when the head is in motion, thus causing stretching and tearing of pallidal branches of the anterior choroidal artery and striatal branches of MCA which in turn produce hemorrhage in the basal ganglia

Table 2: Summarizing the statistical data: Correlation of variables with outcome at discharge and 12-month follow-up.

Variable	At discharge: *P*-value	At 12 months: *P*-value
Age	0.54	0.66
Gender	0.29	0.48
ENT bleed	0.46	1
GCS score	**<0.001**	**0.03**
GCS M score	**<0.001**	**0.01**
Pupil asymmetry	**0.015**	0.16
Hemiparesis	**0.024**	**0.028**
Laterality	0.62	0.48
Volume	**0.016**	**0.01**
IVH	0.43	0.18
Other CT findings	0.98	0.75
Management	0.39	0.25

Chi-square test was used for statistical analysis. P<0.05-significant. ENT: Ear nose throat, GCS: Glasgow coma scale, IVH: Intraventricular hemorrhage, CT: Computed tomography, Values in bold indicate significance

region.[16] Boto *et al*. stated that inertial phenomenon was responsible for most of the TBGH.[3] McPherson *et al*. observed that TBGH share many features of severe diffuse white matter injury (as described by Zimmerman *et al*.) and proposed that TBGH occur due to acceleration/deceleration forces resulting in shearing of white matter blood vessels.[15,21] In 1980, Maki *et al*. hypothesized that anterior stretch on the lateral branches of the MCA perforators secondary to opposite direction of skull rotation may play a role in traumatic TBGH.[13] Recently, Fujioka *et al*. proposed that infarction and hemorrhage in basal ganglia region are produced by traumatic MCA dissection.[4]

Clinicoradiological features

They are predominantly seen in young adults (3rd and 4th decades) and uncommonly reported in pediatric age group.[11,12] Clinical manifestations depend on the structures involved by the hematoma which include pyramidal weakness, extrapyramidal symptoms, sensory impairments, and prolonged unconsciousness due to involvement of internal capsule, substantia nigra, thalamus, and reticular activating system, respectively.[3,9] Majority (more than two-thirds) of the TBGH are due to RTA involving high-velocity trauma and are mostly seen in severe TBI patients.[3,7,11,12,15] Bilateral TBGH are very rare and few studies have reported significant proportion of cases occurring contralateral to the impact of trauma.[2,3,6,15,16,20] In our study, we had two patients with bilateral hematomas. They are frequently associated with DAI, ranging from 30% to 70%.[1,3,11,15] It is, however, to be noted that TBGH can occur in the absence of DAI and vice-versa.[3,15] Surprisingly, in our study, only 8 cases (25%) had features of DAI which is slightly lesser as compared to previous studies. Although some studies have observed skull fractures to be relatively infrequent co-occurrence, they have been observed in at least one-third to half of cases in majority of studies.[3,7,11,12,15,17,19] SDH, focal contusions, and IVH are other common findings associated with TBGH.[3,11,12,15,19] Associated CT findings were seen in around 90% of our cases (n = 28).

Boto *et al*. retrospectively analyzed 37 cases of severe TBI with TBGH. About 94% were due to RTA. Skull fracture was present in 10 (43%) of the 23 patients in whom skull X-rays were obtained. Coagulation disorders were present in 32 (86%) cases. CT findings included DAI in 27 patients (73%), IVH in 22 patients (59%), and SAH in 16 patients (43%). Four patients (11%) underwent surgical evacuation.[3]

Takeuchi retrospectively analyzed 20 cases of TBGH. About 60% were due to RTA (n = 12) and rest due to FFH (n = 8). The mean GCS score was 7.5. Skull fractures were seen in 5 (25%) cases. They noted a high frequency of putaminal involvement (n = 15). The mean hematoma volume was 10.7 mL. Associated lesions were as follows: SAH (n = 10), focal contusions (n = 9), SDH (n = 5), IVH (n = 4), and DAI (n = 5). Six patients (30%) underwent surgery and rest were conservatively managed.[19]

Kurwale *et al*. analyzed 21 pediatric cases of TBGH. High velocity RTA (52%) and falls (38%) were the injury mechanisms. The mean GCS score was 6 and 4.8 (overall and in severe HI subgroup). Majority (76%) were severe HI. Ten (47%) had associated injuries and skull fractures were noted in seven cases. Only 3 (14%) cases underwent surgical evacuation.[11]

With regards to clinicoradiological features, our study observations are similar to most of the previously published studies.

Management and outcomes

Treatment options for patients with TBGH include conservative management, craniotomy, and stereotactic/ultrasound-guided aspiration. In all the previous studies, majority of the cases were managed conservatively. In general, the authors have recommended surgical evacuation for hematoma volume > 25 mL.[3] The proportion of cases who underwent surgical evacuation ranges from 11% to 30%.[3,11,19] In our study, 7 (22%) patients underwent surgery.

Although there have been mixed observations regarding the outcomes of TBGH, majority of the authors have noted poor outcomes in their patients.[3,10-12,19] Boto *et al*. reported an overall poor outcome for TBGH. There were 22 (59%) deaths. Nine patients had unfavorable outcomes (2 (5%) vegetative and 7 (19%) with severe disabilities) and only 6 patients (16%) attained favorable recovery. They observed that patients with hematoma volume >25 mL and cases in whom there was hematoma volume enlargement or raised ICP had the worst outcomes. However, one confounding factor in this study was the presence of coagulation abnormalities which were seen in close to 90% of their cases.[3] As it is an independent factor for poor outcome for intracerebral hemorrhage, all other studies, including ours have excluded presence of coagulation problems while analyzing data related to TBGH. On the other hand, dot contusions (<2 mL volume) too should not be included in the category of TBGH as it may falsely provide high proportion of good outcomes.

Similar results (high rates of poor outcomes and mortality rates) were reported by other authors as well.[11,15,19] Out of the 20 cases reported by Takeuchi *et al*., 7 patients (35 %) died, one had persistent vegetative state, four had severe disabilities with 8 patients (40%) achieving favorable outcome. They observed that low GCS score and presence of midline shift were important prognostic factors.[19] In the study by Kurwale *et al*., the mortality rate was 52% (n = 11) and five were primarily due to raised ICP. Among

the initial nine cases with poor nonfunctional outcomes, five improved neurologically with overall 6 patients (28%) becoming independent and functional at a mean FU of 11.6 months. They concluded that overall outcome was unfavorable and GCS score was the only factor associated with outcome.[11] McPherson *et al*. observed that two-thirds of their patients were either dead, vegetative or severely disabled at 6 months.[15]

However, one study by Kumar *et al*. reported good outcomes (GOS 4 or 5) in all their patients with zero mortality. This was because of the favorable admission characteristics in their study, namely, mean GCS score of 10 and mean volume of 13.2 mL.[10] Barring this, across all other studies, the mortality rates range from 23% to 59%, proportion of cases achieving good/functional outcomes range from 16% to 53% and those achieving unfavorable outcomes range from 46% to 84%.[3,11,12,15,19]

In our study, the mortality was rather low as compared to other studies. We feel that one of the main reasons for this is that we had a lesser proportion of severe head injuries in comparison with other studies. In the study by Boto *et al*., all 100% were severe TBI while it was 72% as noted by Kurwale *et al*. In terms of GCS scores, the mean GCS score was 7.5 and 6, respectively, in the studies by Takeuchi *et al*. and Kurwale *et al*., while it was 8.5 in our study. Furthermore, more than half of the patients in the study by McPherson *et al*. had features of raised ICP (effacement of basal cisterns and >5 mm midline shift) and Kurwale *et al*. reported that five patients died primarily due to raised ICP. These would have also contributed to the high mortality in these studies. The exact mortality rate has not been mentioned by McPherson *et al*., however, they observed that two-thirds were either dead or severely disabled at 6 months. Apart from the mortality rates, the proportion of poor outcomes (severely disabled and vegetative state) in our study was similar to the findings of other studies.[3,11,15,19]

With regards to prognostic factors, poor admission GCS scores, pupillary asymmetry, presence of midline shift, larger hematoma volume, enlarging hematomas, and hematomas with raised ICP were the common factors associated with poor outcomes in all of the previous studies.[3,11,12,17,19] In our study, admission GCS scores, motor component of GCS, focal deficits, and hematoma volumes were poor prognostic factors at 12 months. Residual hemiparesis is often noted in a significant proportion of the survived cases.[5,11,13] In our study, 12 patients had varying degrees of residual/persistent limb paresis. In their study, Maki *et al*. reported hemiparesis as a common disability in all survivors with TBGH.[13] Similarly, Kurwale *et al*. stated that five out of 10 survivors had varying degrees of residual weakness.[11]

Merits and limitations

Merits

We analyzed 32 patients with strict inclusion and exclusion criteria (excluding dot contusions) and all patients were followed for a minimum of 12 months which is not commonly reported in other studies.

Limitations

The limitations of the study are retrospective nature of the study, single-center study, and the relatively smaller number of subjects for statistical analysis.

CONCLUSION

TBGHs are rare lesions encountered in clinical practice. Majority are due to high velocity trauma and most of them have severe head injuries. They are mostly managed conservatively with surgery reserved for larger hematoma volumes. Our study noted poor admission GCS scores, poor motor response component of GCS score, presence of notable hemiparesis, and larger hematoma volumes (>20 mL) to correlate with poor outcomes at 12 months. The overall outcomes have been mostly unfavorable as observed in majority of studies due to deeper location of these hematomas, high proportion of severe head injuries, and high proportion of residual weakness in survivors.

Declaration of patient consent

Patients' consent not required as patients' identities were not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Adams G, Doyle D, Graham DI, Lawrence AE, McClellan DR. Deep intracerebral (basal ganglion) hematomas in fatal non-missile injury in man. J Neurol Neurosurg Psychiatry 1986;49:1039-43.
2. Bhargava P, Grewal SS, Gupta B, Jain V, Sobti H. Traumatic bilateral basal ganglia hematoma: A report of two cases. Asian J Neurosurg 2012;7:147-50.
3. 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;94:224-32.

4. Fujioka M, Maeda Y, Okuchi K, Taoka TK. 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;30:1975-7.
5. Gupta N, Kankane VK, Gupta TK. Outcome of Traumatic bilateral basal ganglia Hemorrhage: Rarest entity: Prospective study of five cases: Single institutional Experience. Roman Neurosurg 2018;32:322-31.
6. Jang KJ, Jwa CS, Kim KH, Kang JK. Bilateral traumatic hemorrhage of the basal ganglia. J Korean Neurosurg Soc 2007;41:272-4.
7. Jayakumar PN, Kolluri VR, Basavakumar DG, Arya BY, Das BS. Prognosis in traumatic basal ganglia hematoma. Acta Neurochir 1989;97:114-6.
8. Jellinger K. Traumatic basal ganglia hemorrhage. Neurology 1940;40:862-3.
9. Katz DI, Alexander MP, Seliger GM, Bellas DN. Traumatic basal ganglia hemorrhage: Clinicopathologic features and outcome. Neurology 1989;39:897-904.
10. Kumar S, Jha D, Abbey P, Mishra V, Handa A. Outcome of traumatic basal ganglia hemorrhage. Internet J Neurosurg 2008;6:1-5.
11. Kurwale NS, Gupta DK, Mahapatra AK. Outcome of pediatric patients with traumatic basal ganglia hematoma: Analysis of 21 cases. Pediatr Neurosurg 2010;46:267-71.
12. Lee JP, Wang AD. Post-traumatic basal ganglia hemorrhage: Analysis of 52 patients with emphasis on the final outcome. J Trauma 1991;31:376-80.
13. Maki Y, Akimoto H, Enomoto T. Injuries of basal ganglia following head trauma in children. Childs Brain 1980;7:113-23.
14. Massenburg BB, Veetil DK, Raykar NP, Agrawal A, Roy N, Gerdin M. A systematic review of quantitative research on traumatic brain injury in India. Neurol India 2017;65:305-14.
15. McPherson P, Teasdale E, Dharkar S, Allerdyce E, Galbraith S. The significance of traumatic hematoma in the region of basal ganglia. J Neurol Neurosurg Psychiatry 1986;49:29-34.
16. Mosberg WH Jr., Lindenberg R. Traumatic hemorrhage from the anterior choroidal artery. J Neurosurg 1959;16:209-21.
17. Munemoto S, Komai T, Aizumi S. Traumatic hemorrhage in the basal ganglia in the child: Five cases. No Shinkei Geka 1985;13:1027-33.
18. Rubiano AM, Carney N, Chesnut R, Puyana JC. Global neurotrauma research challenges and opportunities. Nature 2015;527:S193-7.
19. Takeuchi S, Takasato Y, Masaoka H, Hayakawa T, Yatsushige H, Shigeta K, *et al*. Traumatic basal ganglia hematomas: An analysis of 20 cases. Acta Neurochir Suppl 2013;118:139-42.
20. Yanaka K, Egashira T, Maki Y, Takano S, Okazaki M, Matsumaru Y, *et al*. Bilateral traumatic haemorrhage in the basal ganglia: report of two cases. No Shinkei Geka 1991;19:369-73.
21. Zimmerman RA, Bilaniuk LT, Genneralli T. Computed tomography of shearing injuries of the cerebral white matter. Radiology 1978;127:393-6.

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