Analysis of Clinicoradiological Profile and Outcome of Traumatic Basal Ganglia Hematoma: A Perspective from Level 1 Trauma Center

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Abstract

Introduction: This study was conducted to analyze clinico-radiological profile of traumatic basal ganglia hematoma and identify its prognostic factors.

Methods and Materials: A prospective study was conducted in the Department of neurosurgery, Trauma center of Banaras Hindu University from September 2016 to March 2020. All patients with traumatic basal ganglia hematoma based on admission CT scan were enrolled and their demographic, clinical, radiological details were maintained till the time of discharge and subsequent follow up. Follow up period was a maximum of two years.

Results: Out of 41 patients of traumatic basal ganglia hematoma, 68% of cases were males in their third decade. Road traffic accident (76%) was the major etiology. 32% had severe head injury, 78% had hemiparesis at admission and 58% patients required ventilatory support. The mean volume of clot was 15.46 millilitres. Only 24% cases had isolated traumatic basal ganglia hematoma. Advance age, associated intraventricular hemorrhage, ventilator dependence, large hematoma (volume >20 millilitres) and poor Glasgow coma score at admission were significant prognostic factors ($p<0.05$).

Conclusion: Traumatic basal ganglia hematoma is a rare entity. Advance age, large volume of hematoma, associated intraventricular hemorrhage, poor Glasgow coma score at admission and ventilator dependence are poor prognostic factors.

Key words: Prognosis, Traumatic basal ganglia hematoma

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Introduction

Traumatic basal ganglia hematoma (TBGH) is very rare.[1] It includes the area of basal ganglia and the surrounding region of the internal capsule, thalamus.[2] A varying incidence of 3-10% in head injuries is often reported.[1,3] The incidence is higher in autopsy series than clinical. The mechanism is shearing of lenticulostriate arteries or anterior choroidal vessels caused by sudden acceleration deceleration injuries.[2,3] The fact that basal ganglia hematoma can be due to trauma holds importance in medico-legal cases where the cause may be attributed to non-traumatic reasons. Thus, the recognition of TBGH is of prime relevance. This study was done to express and analyze the clinico-radiological profile of TBGH among head injury patients and look for prognostic factors amongst such patients.

Methods and materials

The study was conducted in the Department of Neurosurgery, Trauma center, Banaras Hindu University from September 2016 till March 2020. A total of 41

cases of traumatic basal ganglia hematoma (TBGH) were included based on the CT finding at the time of admission. Their demographic profile, clinical neurological findings, imaging findings, treatment, and follow up neurological status were noted. Treatment was started as per institutional protocol for patients with head injury. They were followed up for a period of maximal 2 years and their outcome was evaluated in Glasgow coma outcome scale. Surgical evacuation was done for patients with large hematoma with midline shift of more than 5 mm or mass effects (Table 1).

For statistical analysis, Statistical Package for the Social Sciences (SPSS) version 25 was used; students t-test was used for analyzing the volume of hematoma risk and for comparing the non-numerical factors logistic regression, ANOVA, and chi square tests were used wherever applicable.

Results

We found that 41 patients out of 1906 presenting with head injury during the study period had traumatic basal ganglia hematoma (2.15%).

The mean age of the patients was 36.3 years. The majority of the patients were in their third decade. 68% (28 out of 41) of the study population were males. The mode of injury was Road traffic accident 76% (31 out of 41) and physical assault 24 % (10 out of 41). Of the demographic profile, advanced age was associated with poor outcome ($p<0.001$).

Of these 41 patients, 5(12%) had GCS of 3, and 3(7%) had GCS 15 (Table 2). The average GCS at admission was 9.3. Severe head injury was observed in 14 patients (32%). The severity of the injury was related to poor outcome, GCS 3 at presentation was associated with a dismal prognosis with three out of five patients succumbing to death. Better GCS at admission was associated with a better outcome (i.e. GOS) but it was found to be statistically insignificant ($p>0.05$).

Seventy eight percent (32/41) patients had hemiparesis and 9.8% (4/41) patients were neurologically intact. No significant correlation of hemiparesis was found with the final outcome (Table 3).

58% (24/41) patients had injuries demanding to be put on a ventilator and 41% (10/24) among them were on a ventilator for more than one month and all of them had some degree of ventilator-associated chest infections. Prolonged ventilator stay was associated with poorer outcomes.

Amongst patients with high clot volume, 19 patients had coagulation disorder in the form of either deranged platelets or elevated PT/INR and it was seen to be associated with high clot volume and worsened outcome ($p<0.05$). The average volume of the clot was around 15.46 cc. A high clot volume of more than 25cc was associated with a grave prognosis (Table 3).

31 patients had associated injuries other than basal ganglia hematoma in the form of contusions (17.1%), EDH (7.3%), or multiple injuries to the brain (19.5%) (Figure 1, 2 and 3). Only 10 patients had pure basal ganglia hematoma. None of the patients in our study had bilateral TBGH. 22% (9/41) had associated IVH and 9.7% had extracranial injuries in the form of chest injury and blunt trauma injury. Though associated injuries had a bearing on immediate general condition but did not affect the final outcome of patients except in the case of IVH and associated parenchymal injuries.

Surgeries were done for 44% of patients mainly for associated contusion and EDH. For two patients we had to do clot evacuation of TBGH due to raised ICP and midline shift. Of these patients, both had hemiparesis which persisted in the post-operative period. Immediately after the surgery, GCS either improved or remained the same for the majority of patients but did not deteriorate for any of the patients. Four patients (10%) expired in our study, three were due to severe head injury, and one due to ventilator-associated pneumonia and associated respiratory failure.

38% (16/41) patients had favorable final outcomes with GOS >3. Co-morbidities like diabetes, heart disease had a bearing on the poorer outcome in the patients.

CT findings	GCS	Management	
Evacuable mass lesion with mass effect / midline shift	<8	Surgery	Neurosurgical ICU care
	>8	Surgery	Neurosurgical ICU care
Mass lesion with no midline shift or mass effect	>8	Conservative	Neurosurgical ICU care
	<8	Conservative	HDU care
No lesion	<8	Conservative	Neurosurgical ICU care
	>8	Conservative	HDU care

Table 1. Showing the indications for surgical and nonsurgical indications in our study.

** ICP monitoring was performed as per Brain Trauma Foundation guidelines*

		1	2	3	4	5	Total
GCS at presentation	3	3	2	0	0	0	5
	6	0	0	1	0	0	1
	7	0	0	1	1	0	2
	8	1	1	3	0	1	6
	9	0	1	4	0	0	5
	10	0	0	3	0	0	3
	11	0	0	0	1	0	1
	12	0	0	5	1	1	7
	13	0	0	0	3	0	3
	14	0	0	0	4	1	5
	15	0	0	0	1	2	3
Total		4	4	17	11	5	41

Table 2: Effect of GCS at presentation on the outcome in GOS (Glasgow outcome scale)

Good prognostic factors	Poor prognostic factors (p<0.05)	Factors having no effect on outcome (p>0.05)
-	Deranged coagulation factor in large hematoma volume	Surgery done not
-	Associated IVH	Associated injuries except IVH
Smaller clot volume	Large size of the clot (>20 cc)	Gender
-	Prolonged ventilator support	Hemiparesis
Younger age	Advanced age	Mode of injury (Assault or RTA)
-	Poor GCS at presentation (<8)	-

Table 3. Good and poor prognostic factors related to traumatic basal ganglia hematoma (TBGH) and the factors which had no association with outcome

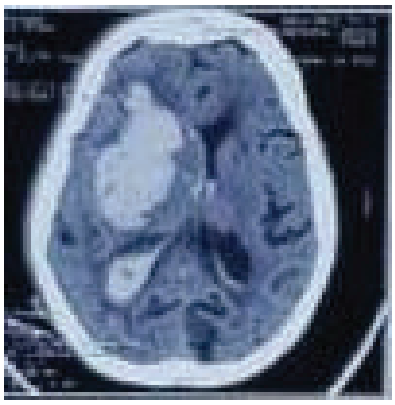

Figure 1: Plain CT scan showing large clot with IVH

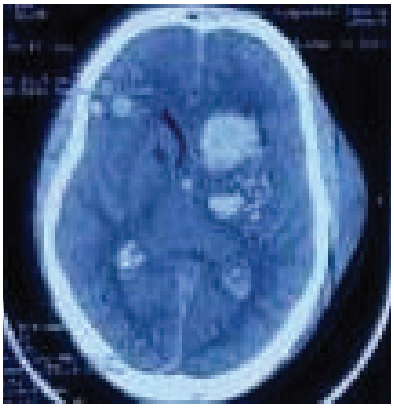

Figure 2: Plain CT scan showing hematoma with IVH extending into both sides

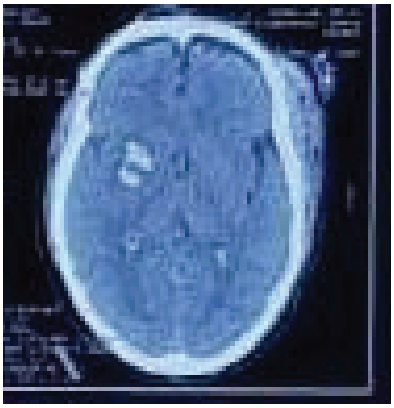

Figure 3: Plain CT scan showing a small hematoma with no IVH

Discussion

Traumatic basal ganglia hematoma (TBGH) is an extremely rare entity, constituting roughly 2-3% [1,4] however the incidence from autopsy series is 9.8%.[1,2] Bilateral hematomas are even rarer such that only description is limited to case reports.[5,6,7] Kumar et al reported one case of bilateral TBGH.[8] We had an incidence of 2.15 % corroborating with earlier studies.

Head injury occasionally leads to parenchymal contusions and the pathophysiology of such a lesion is poorly understood. TBGH is considered as an intermediary contusion, as deep-seated hemorrhages in basal ganglia develop in neural parenchyma between coup and contrecoup contusions, and they occur as a result of shearing injury to vasculature in and around basal ganglia.[9] The proposed mechanism of injury is proposed to be the

one with accelerating decelerating injury, motion injuries, and impact severe enough to deform the skull or force vector making the brain to shift through tentorium due to inertia make cause shearing of vessels leading to TBGH.[10]

It was previously believed that the presentation of TBGH was small, multiple, often bilateral, and located in the zone of a lentiform nucleus and external capsule, whereas spontaneous hemorrhages usually present with large solitary, in the region of the thalamus and internal capsule. causing mass effect. However, a patient suffering from a head injury and having a lesion with characteristics of the latter lesion was considered to be due to a spontaneous bleed causing the event.[11]

On the contrary, Mosberg et al in a patient of a fatal severe head injury at autopsy found a massive hematoma in the palladium and a ruptured twig of the anterior choroidal artery.[12] Histopathological analysis of the report showed trauma to be the cause of the rupture. In 1980, Maki et al proposed pathophysiology for TBGH to be the anterior stretch of middle cerebral artery perforator.[13] A study by Kinoshita et al in 2008 also stated anterior choroidal artery injury or rupture to be the cause of basal ganglia bleed.[14] Fujioka et al also observed and stated traumatic dissection of the middle cerebral artery as a cause for TBGH.[10]

TBGH may be classified as large TBGH if they are more than 2 cm. and small if less than 2 cm which may also be a part of diffuse axonal injury (DAI). In our study we had patients with associated parenchymal injuries in 19.5% but no correlation could be sought with outcome on analysis. It is rare for a basal ganglia hematoma to be large enough to cause mass effect, but if it does it will carry a worse prognosis compared to other intracranial hematomas. A sequential increase in the size of the hematoma on subsequent CT was reported by Boto and Okada et al but was not seen in our study.[3]

The mean volume of Hematoma in our study was 15.46 ml. which was similar to observe from the previous study by Boto et al[3] and Kumar et al.[8] Treatment protocol focuses on the hematoma volume as large hematoma >25cc needs evacuation (as per Boto et al).[3] We also performed surgical evacuation for more than 25cc volume and it was associated with poorer outcome in form of persistent hemiparesis and dependence.

In all our patients there was a clear history of trauma with both clinical and radiological parameters pointing to be a traumatic etiology. The majority of the patients were in their 3rd decade and young males, also making road traffic accidents to be leading cause of head injury in our population possibly because of increasing urbanization, transportation, and industrialization[15]

A wide array of abnormalities was seen in patients with TBGH in post-traumatic acute phase namely coagulation disorders,[3,4,8,16] which in our patients could be seen in 46% cases.

The literature reporting on outcome of TBGH is not uniform. In a study by Boto et al on 37 patients reported only 16% percent made a favorable recovery, 5% were severely disable and 5% died.[3] On the contrary Kumar et al,[8] Katz et al,[4] Kimura et al,[17] Lee et al,[16] and Jang et al[5] found favorable outcome in all patients (of GOS 4 or 5). In our study, we had a mixed picture with 38% patients having a favorable prognosis and 10% mortality (Table 2). We found advanced, post-resuscitation GCS of less than 7, large hematoma volume (>20cc), associated parenchymal injuries, and intraventricular hemorrhage were poor prognostic indicators.[2,3,4,8,16]

Conclusion

Patients when adequately and timely managed carry a favorable prognosis. Large evacuable hematoma causing mass effect, associated conditions of DAI and IVH should be considered while managing such patients as they carry a dismal prognosis and attendants can be prognosticated based on such parameters. Because of rarity, the literature is filled with case reports, this is our sincere effort and possibly the first prospective study to analyze such patients. Further research is still needed to more clearly focus on the disease entity.

Conflicts of Interest: None
Source(s) of support: None

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