Outcome Of Traumatic Basal Ganglia Hemorrhage

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Citation

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Abstract

Traumatic basal ganglia hematoma (TBGH), seen rarely, has been associated with dismal prognosis. We evaluated ten patients (8 males, 2 females; average age 30 years) of TBGH and present their outcome. Average GCS at admission was 10 including 6 patients with severe head injury (GCS≤8). Brain lesions other than BGH were: diffuse axonal injury (DAI) (n=3), intraventricular bleed (n=1) and focal contusions in addition to BGH (n=4) in a total of 6 patients. Average volume of the BGH was 13.2 ml. None of the patients had a coagulation disorder. Surgical evacuation of focal hematoma other than BGH was done in 2 patients. Average GCS at discharge was 13. Average follow up was 30.4 months. Outcomes were excellent (GOS= 5) (n=3) or fair (GOS=4) (n=7) with no mortality. Outcome of TBGH appears favorable unless it is large and associated with coagulation disorders; however, DAI is an important factor governing the outcome in head injury including TBGH.

INTRODUCTION

Traumatic basal ganglion hematoma is defined as intracerebral hemorrhagic lesion located in basal ganglion (caudate nucleus, putamen and globus pallidus) and neighbouring structures like thalamus and internal capsule[1]. Basal ganglia hematomas were infrequently described before the scan era[2]. In post computerized scan era its incidence is approximately 3% of closed head injured patients; however autopsy series indicate a higher incidence ranging between 10%-12%[3456]. TBGH is thought to be due to shearing of lenticulostriate or anterior choroidal blood vessels caused by rapid acceleration and deceleration forces at the time of trauma leading to parenchymal coup and countercoup contusions[237]. DAI and delayed (within 48 hours) increase in hematoma have been implicated for poor prognosis in these patients[18] .

Earlier studies show poor outcome in these patients with high mortality and morbidity, their likelihood to increase in initial 48 hours and frequent association with DAI.[136 7910] .We present our experience of ten successive patients of TBGH seen at our centre.

MATERIAL AND METHODS

Ten successive patients of TBGH, shown in initial CT head, admitted in our institute from May 2005 to November 2008, formed the study group. There were 8 males and 2 females; age ranging from 8 to 50 years (average 30 years). Patients with penetrating injury, BGH volume of less than 2 ml and doubtful history of trauma or unknown mode of injury were excluded from the study. All patients had sustained road traffic accidents. Patients were subjected to CT head after initial resuscitation. GCS at admission were 6 to 15 (mean 10), which included mild (n=2), moderate (n=2) and severe (n=6) head injuries. ICP monitoring (n=4) and ventilatory support (n=3) for 24-48 hours were instituted in patients with severe head injury. Clinical characteristics and radiological findings are summarized in the table (Table 1).

Figure 1

Table 1: Patient characteristics, radiological findings and outcome

CN	Age (yrs)/Sex	CT		Associated injury	GCS at admission	Focal deficit	GCS at discharge	FU (Mon)	Outcome
		BGH	Add. Brain injury						
1.	8 M	R	-	-	E2V3M6	L hp (III)	E4V5M5	11	Fair
2.	50 M	R	IV extension of BGH	MF, L knee #	E4V5M6	No weakness	E4V5M6	6	Excellent
3.	35 M	R	-	L black eye	E4V5M6	L hp (LL-U, LL-II)	E4V5M6	9	Fair
4.	30 M	R	BL F, R T contusion and acute SDH	-	E2V2M5	No weakness	E4V5M6	6	Excellent
5.	32 M	L	-	# R Zygoma	E1V2M5	R Hp (II)	E3V1M5	4	Fair
6.	17 M	L	L F contusion, DAI	# L foot	E1V1M4	R Hp (I)	E4V2M5	10	Fair
7.	35 M	R	DAI	-	E1V1M4	L hp (I)	E4V3M6	5	Fair
8.	25 F	L	L FT contusion	-	E1V1M5	R Hp (III)	E4V3M6	4	Fair
9.	35 F	BL	L F contusion, DAI	-	E1V1M2	L Hp (II)	E4V1M5	3	Fair
10.	36M	L	-	-	E1V2M5	R Hemiparesis	E4V3M6	4	Excellent

CN- case number, yrs- years, M- male, F- female, CT- Computed tomography, Add.- additional, IV- intraventricular, BGH- basal ganglion hematoma, BL- bilateral, R- right, L- left, F- frontal, T- temporal, SDH- subdural hematoma, DAI- diffuse axonal injury, MF-mandibular fracture, #- fracture, Hp- hemiparesis, FU- follow up, Mon- months

Basal ganglia bleeds were located on the right in (n=5), left in (n=3) and bilateral in the remaining one case. Six patients had additional brain parenchymal injuries in the form of focal contusions (n=4), intraventricular bleed (n=1) and diffuse axonal injury (n=3) (Figures 1 & 2).

Figure 2

Figure 1: CT showing right basal ganglion hematoma with surrounding edema producing mass effect shown by midline shift and effacement of ipsilateral frontal horn. Left small fronto-parietal contusion with midline small contusions suggestive of DAI.

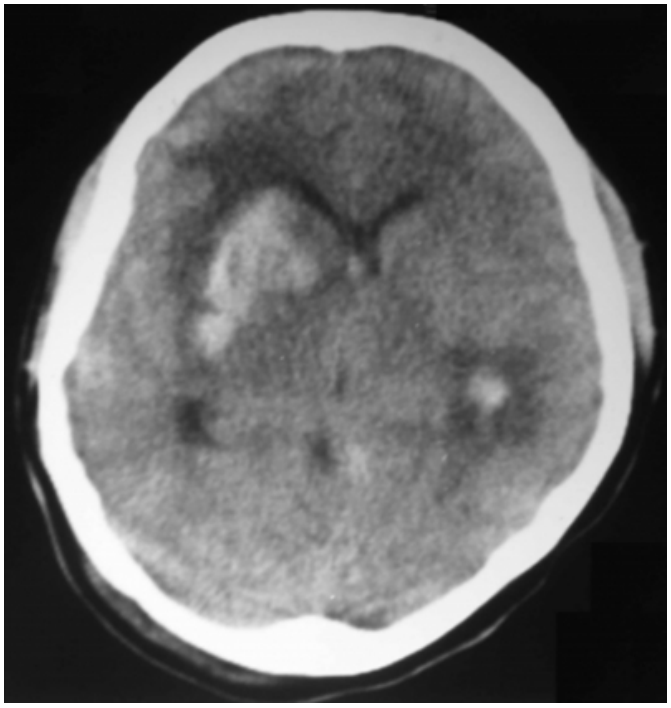
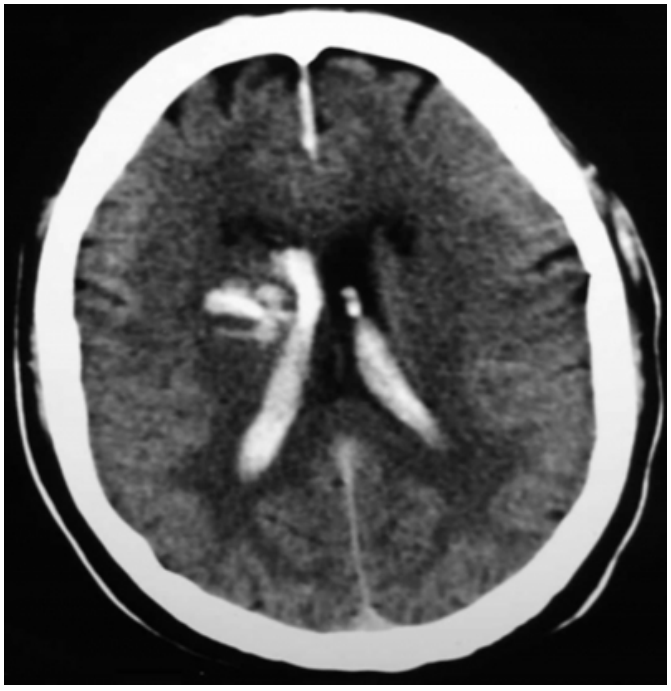


Figure 3

Figure 2: CT showing right basal ganglion hematoma with intraventricular extension.



Bilateral frontal and right temporal (case 4) and left fronto temporal contusion hematomas (case 8) in 2 patients were evacuated due to progressive neurological

deterioration and increasing edema on repeat CT. Contralateral limb weakness was noticed in 8 patients.

Volume of basal ganglion hematoma (V) was calculated by using formula: V= length × width × height × 0.5_9. Length, width and height of the hematoma were obtained from CT/MRI. 'V' ranged from 2.8 ml to 23 ml (average 13.2 ml).

PATIENT MANAGEMENT

Patients were managed as per their clinical condition and radiological (CT/MRI) findings.

After initial resuscitation and radiological examinations (CT head, X ray of cervical spine and extremities and USG of abdomen as and when needed), all but fully conscious patients were shifted to neurosurgical ICU. Orthopedic injuries were managed simultaneously; initially conservatively and once the patient became neurologically stable, operatively (n=2), whenever required. Dehydrants, antiepileptics, ICP monitoring (n=4) and mechanical ventilation (n=3) were given as and when needed. Repeat CT/MRI was done after 24 hours and as and when required in patients with neurological deterioration. MRI was done within 72 hours for assessment of DAI as and when clinical condition permitted. Enteral feeding (oral or through Ryle's tube) and active and/or passive (in unconscious or hemiplegic patients) physiotherapy of limbs and chest were started after first 24 hours. Focal hematomas with progressive neurological deterioration, increasing ICP with radiological evidence of increasing mass effect were evacuated by craniotomies (n=2).

Patients were discharged, once they were on oral or nasogastric tube feeding and medications, with stable neurological status and electrolyte values and attendants were ready to care for them at home.

Patients were followed up at 6 weeks, 3 and 6 months and then at yearly intervals. Outcomes were analyzed at 3 months follow up visit using Glasgow Outcome Score (GOS). Outcomes were labeled as excellent (GOS- 5), fair (GOS- 4) and poor (GOS 1-3).

RESULTS

Follow-up and clinical outcomes of the patients are summarized in the table (Table 1). Increase in the sizes of focal hematomas and surrounding edema other than BGH was noted in 2 patients resulting in increased ICP, midline

shift and neurological deterioration and both of these patients were subjected to craniotomies and evacuation of their hematomas. None of the patients showed increase in their basal ganglia hematoma size. One patient with intraventricular extension of hematoma was managed conservatively as he showed progressive clinical improvement and repeat CT never showed hydrocephalus. One patient each of mandibular fracture and fractured zygoma required open reduction and fixation. Remaining 2 patients of long bone fractures were managed conservatively by closed reduction and immobilization in plaster casts.

At admission average GCS of the patients was 10 (6 to 15); however average GCS at the time of discharge was 13 (9 to 15). Radiological features of DAI were seen in 3 patients (cases 6, 7 and 9). Two patients had no contralateral hemiparesis; however remaining 7 patients had contralateral hemiparesis with power ranging from grade 1 to grade 3. Only 2 patients had raised blood pressures during the course of admission and needed pharmacological support, which could be discontinued over a period of 3-6 weeks.

Follow up periods ranged from 27 to 35 months and 7 patients showed fair outcomes; however remaining 3 patients showed excellent outcomes.

DISCUSSION

TBGH is a rare but serious complication of head injury[2]. TBGH are usually small and located in the zone of lenticular nucleus and external capsule on one or both sides. In contrast, spontaneous hemorrhages are solitary and located in the region of thalamus and internal capsule. TBGH occurs mainly in young patients and is associated with high morbidity and mortality [1367910]. Out of 10 patients, 7 had fair and 3 had excellent outcomes and none of our patients was dependent with regard to carrying out their daily activities. Although a hematoma in the region of basal ganglia is rarely large enough to have appreciable mass effect, it is evidence of significant primary brain injury and carries worse prognosis than other posttraumatic intracranial hematomas. Enlargement of hematomas in sequential scans, observed in high percentage of patients in earlier studies by Boto and Okada et al, were not seen in any of our patients.[111]. DAI too was observed radiologically in only 3 patients unlike earlier reports where it was very common and was associated with poorer prognosis [1356121314]. Though DAI was associated with a relatively poorer GCS at admission in our study, further improvement was no different than the patients not having DAI. There is increasing evidence from human and experimental studies that the most important factor governing the outcome in head injury is the severity of DAI; however, after increasing use of MRI in head injury cases, DAI is detected more often than previously assumed (even in mild and moderate head injuries).

Hematoma volume of 23 ml was the maximum in our study group; however mean hematoma volume (13.2 ml) was comparable to the initial hematoma volumes in the study by Boto et al [1]. 65% of the TBGH enlarged during the acute posttraumatic period and 86% of these exhibited some type of coagulation disorder that might have contributed to the development of delayed hematomas or the enlargement of preexisting one [1]. Relatively poorer prognosis in patients with hematoma volumes larger than 25 ml and frequent association of coagulation disorder were not seen in our series. None of our patients was a chronic alcoholic or had coagulopathy.

Surgical evacuation of TBGH was not required in any of the cases in our study group as ICP could be controlled under the safe limit (less than 25 mm of H2O) by pharmacological means, ventilatory support, and evacuation of larger focal contusions other than BGH in 2 patients. Management of patients with TBGH is as per established lines of management of traumatic brain injury; however basal ganglia hematoma per se rarely needs surgical evacuation. Various surgical options have been used for these patients including open surgery, CT guided stereotactic aspiration and ultrasonography guided aspiration [15610151617]. Except few case reports[516171819], most of them experienced poor outcomes in these surgically treated patients [31416]. However, we do feel that patients with increasing size of hematoma with deteriorating neurological condition should be subjected to surgery without delay. In addition, medical and supportive treatments including judicious use of ICP monitoring and ventilatory support are extremely important for better outcomes in these patients.

Earlier reports have shown poor outcomes in TBGH patients with regards to mortality and morbidity[1235671020]. The poor prognosis in patients with traumatic basal ganglia hematoma reflects the global nature of the injury to brain, though hemorrhage is localised on computed tomography to a small part of brain [9]. Superior outcomes in our study could be due to smaller volumes of TBGH, lesser incidence of DAI and no coagulation disorder in our patients. TBGH with coagulation disorder should be taken as a separate entity and it is associated with dismal prognosis [1].

Prompt surgical interventions in selected patients leading to improved survival have been shown by few studies [5][16][17]. However, morbidity still remains a matter of concern in these patients. Favorable and good outcome have been reported by Katz et al [5] and we too feel that, outcome of these patients, in current situation with better trauma care with regards to transport, diagnosis, monitoring facilities, ICU care and management appears better than experienced in the past [5][21][22]. A larger prospective study is required to substantiate these findings.

CONCLUSION

Prognosis of TBGH patients appears favorable if not associated with coagulation disorders and/or large hematoma. DAI, per se, is an important factor governing the outcome in any head injury patient including TBGH.

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