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TRAUMATIC BASAL GANGLIA BLEED

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ABSTRACT

Traumatic Basal ganglia bleed is not a frequent hemorrhagic lesion of basal ganglia or adjacent structures. The lesion determines clinical signs related to particular sub cortical structures involved, with incidence of 3% in close head injuries and 10% to 12% in autopsy cases.[1, 2, 3, 4] It is compatible with a favorable outcome, especially when occurring in isolation. We evaluated 19 cases to define its clinical status, cause and prognosis in duration of 2 years in department of neurosurgery LUMHS. we admitted 10 patients at GCS ≤ 8(severe head injury), 7 with moderate head injury at GCS between 9/12 and 2 patients were at GCS between13/15 with mild head injury. Seventeen patients had Contra- lateral hemiparesis, which recovered to varying extents, apparently related to lesion. Intraventricular, tentorial bleed, pneumocephalus and EDH were the associated findings in the selected subjects of study. Overall cognitive impairment and speed and quality of recovery are more related to associate cerebral damage with varying degree of prognosis.

KEYWORDS: head injury, basal ganglia, hematoma.

INTRODUCTION

The head injury is a very devastating condition in this mechanized world, affecting young people and it is the third most common cause of death[5], has a very worse impact on the economy of a country like Pakistan. Besides dealing routine head trauma, where a neurosurgeon faces skull fractures, traumatic intracranial hematomas and contusions, the traumatic Basal ganglia bleed is a rare but most distressing lesion to be dealt especially when associated with diffuse axonal injury.

Traumatic Basal ganglia bleed is an infrequent hemorrhagic lesion of basal ganglia (caudate nucleus, putamen, globus pellidus, substantia nigra) or adjacent structures (internal capsule, thalamus or sub thalamic nuclei), due to shearing and tearing of lenticulostriate or anterior choroidal vessels by aggressive acceleration or deceleration forces at the time of insult.[1, 6, 7]

With the advent of modern techniques of investigations like CT scan and MRI, traumatic basal ganglia bleed can diagnose early but difficult to differentiate from diffuse axonal injuries.[1,2,3,4,7] Traumatic Basal ganglia hemorrhage may present as an isolated lesion with good prognosis. When associated with cerebral contusion, subdural or extradural hematomas and with diffuse axonal injury, prognosis becomes worst depending upon severity of the injury.[3]

When isolated traumatic basal ganglia bleed present with poor GCS, it is difficult to interpret weather this is an isolated traumatic basal ganglia bleed or diffuse axonal injury which is the main culprit. Sometimes it is difficult to separate two pathologies. We are presenting the type, presentation and the recovery time after the isolated traumatic basal ganglia bleed in patients admitted in our ward the during period of two years.

MATERIAL AND METHODS

This study was conducted in department of neurosurgery LUMHS, during the period of 2 years from June 2012 to may 2014. Nineteen cases of traumatic basal ganglia bleed were separated after their imaging and presentation. All patients were admitted through causality where they were managed on primary trauma care protocol and after CT scan, patients were admitted in the department of neurosurgery. A Proforma of study was filled with complete bio data, mode of injury, time of injury, first aid and time between injury and hospital arrival, GCS at arrival and recovery. All patients were treated conservatively. Patients with GCS below 08 or those not maintaining airway were shifted to intensive care unit.

Associated injuries were managed simultaneously. Enteral feeding was started along with physiotherapy of limbs depending upon the conscious level and paresis of patient on the second day of admission.

Patients were discharged when they had developed neurological stability, normal electrolyte values and were kept on oral or nasogastric feed.

Patients were called for follow up in out patient department fortnightly till they return back to their work.

RESULTS

Out of 19 patients 16 were male and only 3 patients were female, age ranging from 04 to 45 years, (average 18 years). Majority of patients belongs to young age group. Most of the patients had suffered road traffic accidents followed by fall from height. Patients reached our department within 1 to 12 hours of injury (average time7 hours), without initial resuscitation due to lack of facilities of basic health units, trained staff at the scene of accident and transportation.

Most of the patients received in moderate to severe head injury. There were10 patients of severe head injury, 7 patients of moderate head injury and 2 patients of mild head injury. Seventeen patients had contra lateral hemiparesis grading 1 to 4 (medical research council scale), one patient was quadreparetic due to associated cervical trauma and one patient had no motor weakness.

There was nearly equal distribution of traumatic basal ganglia bleed on both sides of cerebral hemispheres. Traumatic basal ganglia bleed were located on right side in 8 patients and 9 patients had lesion on left side, while remaining 2 had bilateral basal ganglia bleed. 3 patients had associated intraventricular bleed while two cases had non surgical EDH one on the right and other one the left side of parietal region and one had tentorial bleed. Pneumocephalus was seen in 2 patients.

Four patients died, two patients with associated diffuse axonal injury and two patients with associated intraventricular bleed also having chest problem.

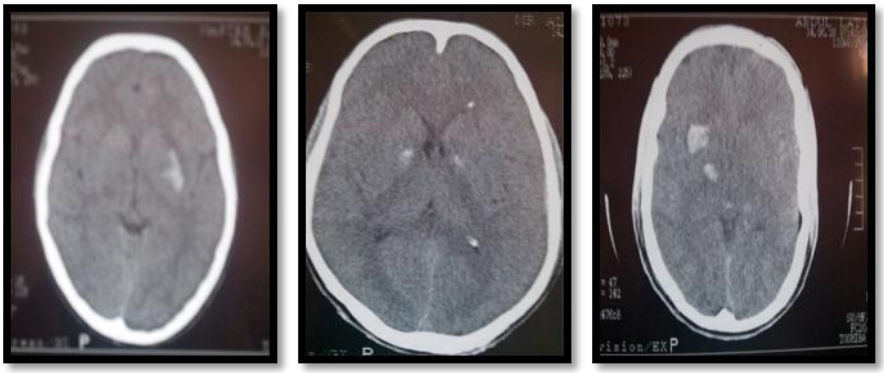
All other patients showed good prognosis and improvement to varying degree ranging from 2 to 5.

Clinical and radiological findings were as under:

S#	AGE	MOI	Time to reach Hosp:	GCS at Adm:	CT scan finding		Volume of blood	Associated Injury	Focal DEFECTS	Prognosis
					Side Of BGB	Associated brain injury				
1	14y	RTA	8hrs	6/15	Right		24 ml	Right femur #	Left hemiparesis	Expired
2	18y	RTA	12hrs	8/15	Left		12 ml	Nasal bone #	Right hemiparesis	
3	27y	RTA	3hrs	10/15	Left	Tentorial bleed	9 ml		Right hemiparesis Right Facial palsy	
4	22y	FALL	10hrs	14/15	Right		4 ml		Left hemiparesis	
5	22y	RTA	6hrs	15/15	Right		1.5 ml		Left hemiparesis	
6	25y	RTA	4hrs	11/15	Left		6.25 ml		Right hemiparesis Left Facial palsy	
7	07y	RTA	10hrs	8/15	Left		9 ml		Right hemiparesis	
8	45y	RTA	6hrs	6/15	Right	Intraventricular bleed	18.37 ml	Left wrist #	Left hemiparesis	Expired
9	30y	RTA	7hrs	7/15	Right		13.87 ml		Left hemiparesis	
10	22y	RTA	12hrs	8/15	Bilateral	Right parietal EDH	11.25 ml	Right knee#	Left hemiparesis	
11	32y	Fall	4hrs	11/15	Right	Intraventricular bleed	7.5 ml		Left hemiparesis	

S#	AGE	MOI	Time to reach Hosp:	GCS at Adm:	CT scan finding		Volume of Blood	Associated Injury	Focal DEFECTS	Prognosis
					Side Of BGB	Associated brain injury				
12	19y	RTA	8hrs	7/15	Left	Pneumocephalus	13.12 ml	Mastoid#	Right hemiparesis	
13	6y	RTA	4hrs	6/15	Right	Intraventricular bleed	18 ml	Left femur#	Left hemiparesis	Expired
14	8y	FALL	6hrs	10/15	Right		7.5 ml		Left hemiparesis	
15	25y	RTA	10hrs	7/15	Left	Pneumocephalus	16. 08 ml	C3 fracture dislocation	Quadreparesis	Expired
16	9y	RTA	12hrs	9/15	Bilateral	Left parietal EDH	10.08 ml	Right tibia#	Right sided hemiparesis	
17	7y	RTA	4hrs	8/15	Left		15.3 ml		No motor weakness	
18	07y	RTA	4hrs	11/15	Left		7.92 ml		Speech deficit	

19	4y	RTA	1hrs	12/15	Left		7.02 ml	De-gloving injury of face and scalp	Right hemiparesis	



DISCUSSION

Though the TBGH is not very common but still it is a serious complication of head injury[8], though the volume of blood in TBGH is never so high that needs evacuation but still poses challenges to a neurosurgeon.

Main victims of TBGH in our society are young males because they are the bread and butter winner for their families.[1, 4, 6, 7, 9,10]

Out of 19 patients 10 patients have fair, 5 have good out come and 4 patients died.

Out come largely dependent on two main factors that is diffuse axonal injury (DAI) and volume of blood and in certain cases time to reach hospital is also very important.

Prognosis is better with small volume of blood as compare to large volume of blood. Maximum volume of hematoma in our study 24ml minimum was 1.5 ml, so an average volume in our study was 11.14 ml and this almost equal to study by Boto etal.[6]

In our study patient who on average have about 20 ml 0f blood suffered poor prognosis as compare to a study S Kumar and D Jha et al, where 25 ml of blood was associated with poor prognosis.[11]

The second important factor was DAI. We encounter four pt. and these were associated with worst prognosis. This was compatible with various other studies.[1, 3, 4, 6,12,13,14]

In contrast to study by Boto, Lobeto, Okada et al, who reported enlargement of hematoma in subsequent CT scan of brain, was not found in our study.[6,15]

Time period to reach hospital from the scene of accident is also an important factor. We found that those patients, who reach hospital early, relatively have good chance of early recovery and survival. The minimum and maximum time to reach hospital was 1 and 12 hour respectively. The average time was 7 hours. We did not find any other study who mentions the time interval from the scene of accident to hospital.

Surgical evacuation was not carried out in any of our patient. All of our patients were managed conservatively with the help of medicine and ventilator support as per management protocol and guideline and our results are compatible with different studies who also showed good or favorable outcome[3,4,11,16,17] but are incompatible with study by Boto GR, Loboto RD etal.[6]

CONCLUSION

TBGH if managed according to proper guideline has favorable outcome. Isolated TBGH has good outcome if not associated with DAI and other serious associated injuries. Time to reach hospital is another factor that has some effect on patient outcome. Size of hematoma has definite role, greater the size poor will be the outcome.

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