

Original Research Article

Outcome of cerebral contusion in mild traumatic head injury patients

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ABSTRACT

Background: Mild traumatic head injury is classified as one with Glasgow coma scale (GCS) score between 13 and 15. The aim of the study was to find underlying cause of deterioration of mild traumatic brain injury (TBI) and its association with other factors like bony injury.

Methods: A prospective observational study at a tertiary centre with patients admitted for mild TBI with GCS 13-15 were followed up in ward and reviewed after every 6 hours interval, those who deteriorated repeat computed tomography (CT) scan was done for further diagnosis and intervention.

Results: Determining the level of deterioration after 24 hours observation following mild TBI, 268 (63.4%) of the admitted patients were discharged home after 24 hours of observation, 89 (20.7%) needed more observation while 63 (14.8%) deteriorated and 3 (0.7%) died. As the GCS on admission was decreasing; deterioration increased i.e., 12.7%, 16.7% and 33.3% for GCS of 15, 14 and 13 respectively. Likewise, as the GCS increased, recovery improved. Subdural haematoma (39.7%) was the leading cause of deterioration while (13.8%) despite their deterioration, CT brain did not reveal any abnormality.

Conclusions: Patients presenting with mild TBI (i.e., GCS- 13-15) secondary to high velocity/energy trauma i.e., road traffic accidents have to be carefully observed for at least 24-48 hours post-trauma as the chances for neurological deterioration are significant especially in polytrauma patients. The most cause for deterioration is subdural haemorrhage among others, has to be investigated so appropriate management for the same can be instituted as soon as possible.

Keywords: Traumatic brain injury, GCS, Polytrauma

INTRODUCTION

Traumatic brain injury (TBI) is frequently referred to as the silent epidemic because the problems that result from it often are not visible. Mild traumatic brain injury (MTBI) accounts for at least 75 percent of all traumatic brain injuries in India. However, it is clear that consequences of MTBI are not often mild.¹ According to the World Health Organization report 2003, TBI will surpass many diseases as the major cause of death and disability by the year 2020, currently ranking fifth while ischaemic heart diseases being the leading one.²

Furthermore, an estimated 10 million people are affected annually by TBI, the burden of mortality and morbidity that this condition imposes on society, makes TBI a pressing public health and medical problem.

Glasgow coma scale (GCS) was initially used to measure the degree of posttraumatic brain damage severe head injury. Later it was used to define mild traumatic head injury as one with the score between 13 and 15. Independently of difficulties of defining a mild TBI, it should be stressed that patient initially classified as suffering such an injury may develop neurological deterioration. Different studies have shown the

importance of early identification of the factors making neurological deterioration despite these efforts patients labelled as mild TBI are still deteriorating. So, this study was conducted to find underlying cause of deterioration of mild TBI and its association with other factors like Long bone injury. The study enlightens on the magnitude of mild contusion at the institute and also helps to determine the extent of mild traumatic brain injured patients who deteriorated and needed other intervention such as craniotomy or intubation.

The challenge present due to lucid interval in epidural hematoma, slow leaking bridging vein in acute subdural hematoma, gradual increase in intracranial pressure and change of auto regulatory set point due to traumatized brain cells with standard of care which have to meet these challenges will be explained and the inputs will help to improve the standard of care at the institute.

Objectives

The objectives of the study were to determine the outcome of mild cerebral contusion and proportion of mild traumatic brain injuries by demographic distribution by age and sex in patients of traumatic brain injury admitted in tertiary care hospital between June 2015 to November 2017, to determine the level of deterioration of patients admitted for 24 hours observation following mild traumatic brain injury and determining determine the common underlying causes of patient's deterioration and to determine the influence of long bone fractures on neurological outcome of mild traumatic brain injured patients.

METHODS

A prospective observation with consecutive patients with mild traumatic brain injury admitted and followed on their progress in the wards. The study was conducted at tertiary trauma centre and Orthopaedic, Trauma and Neurosurgical services. Patients enrolled in the study were recruited from the emergency department during admission, after meeting the inclusion criteria and thereafter followed in the wards. All consecutive patients admitted with mild traumatic brain injury through emergency medical services from June 2015 to November 2017 were followed up in ward and being reviewed after every 6 hours interval, and based on Glasgow coma scale, those who deteriorated repeat CT scan was requested for further diagnosis and intervention.

Inclusion criteria

All patients age 13 years onward. This is the age in which patient is able to explain his condition and evaluate himself or herself on the progress. Mild traumatic brain injured patient admitted at the institute, arriving within 24 hours post injury, Glasgow coma score of 13 and 14 and Glasgow coma score 15 with the following features as history of LOC, post traumatic amnesia, persistent and

worsening headache, vomiting, seizures and presence of lateralizing sign were included.

Exclusion criteria

Age below 13 years, patients arriving 24 hours post injury with serious medical condition i.e., in stroke, patients with known epileptic disorders, patient in alcohol intoxication and non-consenting patients were excluded.

Data collection was done through a structured questionnaire at Emergency Department and in the ward in the course of 24 hours. After admission, these patients were reviewed at 6 hours interval to assess progress. This was done with assistance by pre-trained admitting staff, and assisted by other neuro surgery firm doctors in the ward. The fully filled questionnaires were entered in the data base prepared in the SPSS programme version 17 by the researcher for analysis. Data were summarized in form of proportions, frequency tables, pie charts bar charts and two by two tables.

RESULTS

An overview of the demographic profile of the participants in relation to age and sex have been highlighted. Level of neurological deterioration and their underlying causes are presented while statistical significance was determined through the Chi-square and p value. Tabulations and pie chart were used to present the characteristic features observed.

Table 1: Socio-demographic characteristics of patients admitted with mild traumatic brain injury.

Characteristics	No. of patients	%
Age (in years)		
<15	4	0.9
15-24	127	29.9
25-34	140	33
35-44	71	16.7
45-54	34	8.0
55-64	21	4.9
>65	22	5.2
Mean 33.6 and SDV 14.7		
Sex		
Male	334	78.8
Female	90	21.2

Male: female 3.7: 1.

On proportion of mild traumatic brain injuries by their demographic distributions the findings revealed that, males were more affected 334 (78.8%) than female 90 (21.2%) by ratio of 3.7: 1. The age group 25-34 years were at high risk of sustaining mild traumatic brain injury i.e., 140 (33.0%) while age group <15 years was least group to be affected 4 (0.9%). Mean age found to be 33.6 and SD 14.7.

Cause of traumatic brain injury

Road traffic accidents were the leading cause of mild traumatic brain injuries comprising 77.8% of the total admission followed by assault 15.30%.

Determining the level of deterioration after 24 hours observation following mild traumatic brain injury, two hundred and sixty-eight (63.4%) of the admitted patients were discharged home after 24 hours of observation, eighty-nine (20.7%) needed more observation while 63 (14.8%) deteriorated and 3 (0.7%) died. Level of consciousness (GCS) has a direct impact towards patients' recovery or deterioration. These findings were found to be statistically significant ($p=0.000$).

Table 3: Outcomes of mild traumatic brain injury after 24 hours of observation.

Level of consciousness (GCS)	N	Discharged within 24 hours	More observations after 24 hours	Deteriorated	Dead	P value
		N (%)	N (%)	N (%)	N (%)	
15	309	239 (77.6)	29 (9.4)	39 (12.7)	2 (0.6)	
14	85	30 (35.3)	41 (48.2)	14 (16.5)	0 (0.0)	
13	30	0 (0.0)	19 (60.0)	10 (33.3)	1 (0.3)	
Total	424	269 (63.4)	89 (20.7)	63 (14.8)	3 (0.7)	0.000

Table 4: Association of symptoms and outcome.

Signs and symptoms	N	Discharge home	More observation	Deterioration	P-value
		N (%)	N (%)	N (%)	
LOC					
+	359	219 (61.0)	85 (23.7)	55 (15.3)	0.024
-	62	50 (80.6)	4 (6.45)	8 (12.9)	
Vomiting					
+	73	13 (17.8%)	35 (47.9)	25 (34.2)	0.000
-	348	256 (73.6)	54 (15.5)	38 (11.0)	
Headache					
Normal	145	103 (71.0)	8 (5.5)	34 (23.4)	0.707
Mild headache	245	164 (66.9)	68 (27.8)	13 (5.3)	
Severe headache	51	2 (3.9)	13 (25.5)	36 (70.6)	
Seizure					
+	39	14 (35.9)	15 (38.5)	10 (25.6)	0.001
-	382	255 (66.8)	74 (19.4)	53 (13.9)	
Dilated pupils					
+	27	4 (14.8)	19 (70.4)	4 (14.8)	0.001
-	394	265 (67.3)	70 (17.8)	59 (15.0)	
Ear, nose, mouth discharge					
+	89	40 (44.9)	31 (34.8)	18 (20.2)	0.001
-	332	229 (69.0)	58 (17.5)	45 (13.6)	
Skull fracture					
+	70	35 (50.0)	20 (28.6)	15 (21.4)	0.015
-	351	234 (66.7)	69 (19.7)	48 (13.7)	
Total	421	269 (63.9)	89 (21.1)	63 (14.9)	

Cause of deterioration

Subdural haematoma (39.7%) was the leading cause of deterioration while (13.8%) despite their deterioration,

In Table 4, the findings revealed that, initial loss of consciousness, vomiting, seizures, dilated pupils, skull fracture, ear, nose and mouth discharge had statistically significant value on determining the outcome of mild TBI while headache showed no significant role.

Table 2: The underlying causes of injuries among patients admitted with mild traumatic brain injury.

Cause of injuries	Proportion (%)
Road traffic accidents	77.80
Fall	4.20
Assault	15.30
Others	2.60

CT brain did not reveal any abnormality. On association of long bone fractures on neurological outcomes, a high proportion of deterioration was observed in isolated TBI group 51 (17.4%) than TBI with long bone fractures 12 (9.3%).

Table 5: The diagnosed underlying cause of patients' deterioration after 24 hours of observation.

Diagnosis following deterioration	Proportion (%)
Subdural haematoma	39.70
Epidural haematoma	19.00
Brain oedema	22.40
Normal CT brain	13.80
Subarachnoid haematoma	5.20

Table 6: Association of long bone fractures and neurological outcome of mild traumatic brain injury

Patterns of TBI	N	Deteriorated	Not deteriorated	P value
		N (%)	N (%)	
TBI+Long bone fractures	129	12 (9.3)	117 (90.7)	0.043
Isolated TBI	292	51 (17.4)	241 (82.5)	
Total	421	63 (14.9)	358 (80.0)	

DISCUSSION

In this study the male- female ratio was 3.7: 1. This is a rapid increase in a gap between male and female for the past 5 years where a ratio of 2:1 was observed by Mwanga on his pattern of traumatic brain injury at MOI.¹ Many studies show men suffer twice as many TBIs as women do and have a fourfold risk of fatal head injury.² The age of the study population ranged from 13 years to 80 years. Age group between 25-34 years being most affected 140 (33%) with mean age 33.6 years and standard deviation 14.5.

Nearly similar findings were observed by Bruns et al who concluded that male gender and young adults are at increased risk of succumbing traumatic brain injury compared to female and other age groups.^{3,4} The study findings revealed that 63 (14.8%) of the study population deteriorated within 24 hours of observation and 3 (0.7%) died. As the level of consciousness on admission was decreasing; deterioration increased i.e., 12.7%, 16.7% and 33.3% with GCS of 15, 14 and 13 respectively. Likewise, as the level of consciousness increased, recovery improved. These findings were statistically significant ($p < 0.001$).

Available data suggests that although between 5% and 13% of patients with a GCS score of 15 evaluated in the emergency department will have a traumatic lesion of some type on CT scan <1% of patients will require neurosurgical intervention. These findings concur with previously established studies by Kiboi et al at Kenyatta hospital who found a statistically significant difference in the proportion of patients who achieved functional recovery with 65.2% in mild TBI versus 19.7% and 15.1% in moderate and severe head injury respectively.^{5,6} Moreover in this study, thirty nine patients 61.8% who deteriorated were admitted with GCS 15. These are the patients whom regardless of good score, talking during admission rapidly deteriorate and even die as a short term outcome i.e., talk and die syndrome.⁷ 57.1% of the patients that deteriorated had severe headache at final

evaluation while 164 (60.7%) of the patients who recovered well had mild headache at discharge, similar findings were observed by Kitumka at Mulago hospital in Uganda in their study of similar cases.⁸

In the present study, subdural haematoma was a leading cause of deterioration with about 39.7% rate. Similar findings were observed by Adeleye et al where subdural hematoma was the leading cause of mortality and disability.⁹ This was explained by its mechanism of injury which involves tearing of bridging veins which are easily torn even with minor injury, sudden acceleration and deceleration. Brain CT-scan was found to be normal in 8 (13.8%) despite of their deterioration. Shackford et al reported the same rate of normal brain CT among TBI group who deteriorated.^{10,11} Cause of deterioration can be explained by the presence of diffuse brain injuries due to second hit impact e.g., hypoxia due to decreased cerebral perfusion pressure, diffuse axonal injuries etc. These kinds of injuries are not evident in routine brain CT scan.

On impact of long bone fractures on neurological outcomes of mild traumatic brain injury, the findings revealed that, the proportion of patients with head injury and long bone fractures who deteriorated was 12 (9.3%) compared to 51 (17.4%) of isolated mild traumatic brain injury. This was contrary to what was expected, because of blood loss due to fracture, increased metabolic and hormonal response due to trauma and the resultant inflammatory response which has direct negative impact on brain physiology. These complex inflammatory events render the traumatized brain highly susceptible to secondary brain injury hence it was expected bring poor outcomes. These injuries probably did not have direct effect on brain physiology. Statistically the results were statistically significant ($p = 0.043$). Lining with these findings, Stahel et al concluded that, during the first 24 hours, any unnecessary surgical interventions, including intramedullary fracture fixation, may negatively alter the patient's MAP, resulting in deterioration of the CPP and aggravation of secondary brain injury, although in this study all patients with long bone fractures were included

regardless whether earlier treated surgically or conservatively.¹² On the contrary, McKee et al revealed different findings from the present study and concluded that, femoral fracture in a patient with a concomitant head injury does not increase mortality or neurologic disability, and supports the continued early intramedullary nailing of femoral fractures for these patients.^{13,14} The exact GCS indicated for neuro-imaging is still under debate at the institute. The protocol cut off point of GCS 12 and below as an indication for brain CT is not valid due to a number of constraints which arises on the possible deterioration despite of good score.

In this study of the 85 patients admitted with GCS of 14, only 30 (35.3%) recovered well after 24 hours and needed discharge, the remaining 55 (64.7%) either needed more observation or deteriorated and brain CT scan were required for all of them. This proportion is higher and statistically significant enough to warrant new cut off point of GCS 14 and below as an indicator of neuro imaging straight forward plus GCS of 15 with any of following features; loss of consciousness for more than 5 minutes, depressed or decreasing level of consciousness, focal neurological findings, seizure, failure of the mental status to improve over time in an alcohol-intoxicated patient, penetrating skull injuries, signs of a basal or depressed skull fracture, confusion or aggression on examination, severe headache, more than two episodes of vomiting and ≥ 65 years.^{15,16} These symptoms were assessed and found to be statistically significant in predictive role on neurological outcomes. Therefore, the established neuroimaging guidelines on traumatic brain injury prove superior and warrant adoption by the institute.

CONCLUSION

The current study and the associated review of literature underlines and emphasises the need for a close follow up of mild TBI patients owing to possible deterioration secondary to variety of causes. The current guidelines suggesting a more conservative approach to mild TBI patients should be viewed critically and tailored to the individual patient. Patients presenting with mild TBI (i.e., GCS- 13-15) secondary to high velocity/energy trauma i.e., road traffic accidents have to be carefully observed for at least 24-48 hours post-trauma as the chances for neurological deterioration are significant especially in polytrauma patients i.e., those associated with long bone injuries. The causes for deterioration most commonly subdural haemorrhage among others, have to be investigated and appropriate management for the same can be instituted as soon as possible. Thus, a close follow up and with a special focus on certain 'tell-tale' signs is prudent and necessary to optimize outcomes on managing these patients. The category of mild TBI patients offer excellent prognostic outcomes and a definitive protocol need to be framed for realising the same.

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REFERENCES

1. Raymond K. Mwangi on pattern of traumatic brain injury at MOI (not published).
2. Rao V, Lyketsos C. Neuropsychiatric sequelae of traumatic brain injury. *Psychosomatics*. 2000;41(2):95-103.
3. Bruns J, Hauser WA. The epidemiology of traumatic brain injury: a review. *Epilepsia*. 2003;44(10):2-10.
4. Adeleye AO, Olowookere KG, Olayemi OO. Clinico epidemiological profiles and outcomes during first hospital admission of head injury patients in Ikeja, Nigeria. *Neuroepidemiol*. 2009;32:136-41.
5. Ong L, Selladurai BM et al. The prognostic value of the Glasgow Coma Scale, hypoxia and computerised tomography in outcome prediction of pediatric head injury. *Pediatr Neurosurg*. 1996;24(6):285-91.
6. Kiboi GJ, Kitunguu KP. Predictors of functional recovery in african patients with traumatic intracranial hematomas. *World Neurosurg*. 2011;75(5):586-91.
7. Reilly PL, Graham DI, Adams JH, Jennett B. Patients with head injury who talk and die. *Lancet*. 1975;2(7931):375-7.
8. Kitumka O. Mulago hospital on the short term outcomes of head injuries in Uganda (not published).
9. Shackford SR, Wald SL, Ross SE, Cogbill TH, Hoyt DB, Morris JA, et al. The clinical utility of computed tomographic scanning and neurological examination in the management of patients with minor head injuries. *J Trauma*. 1992;33:385-94.
10. Iverson GL, Lovell MR, Smith S, Franzen MD. Prevalence of abnormal CT scans following mild head injury. *Brain Inj*. 2000;14:1057-61.
11. Stahel PF, Ertel W, Heyde CE. Traumatic brain injury: impact on timing and modality of fracture care. *Orthopade*. 2005;34:852-64.
12. McKee MD, Schemitsch EH, Vincent LO, Sullivan I, Yoo D. The effect of a femoral fracture on concomitant closed head injury in patients with multiple injuries. *J Trauma*. 1997;42(6):1041-5.
13. Seibel R, LaDuca J, Hassett JM, Babikian G, Mills B, Border DO, et al. Blunt multiple trauma (ISS 36), femur traction, and the pulmonary failure-septic state. *Ann Surg*. 1985;202:283-95.
14. Jeret JS, Mandell M, Anziska B, Lipitz M, Vilceus AP, Ware JA, et al. Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. *Neurosurg*. 1993;32:9-15.

15. Moran SG, McCarthy MC, Uddin DE, Poelstra RJ. Predictors of positive CT scans in the trauma patient with minor head injury. *Am Surg.* 1994;60:533-5.
16. Verwe BH, Muizelaar JP, Vinas FC, Peterson PL, Xiong Y, Lee CP. Impaired cerebral mitochondrial function after traumatic brain injury in humans. *J Neurosurg.* 2000;93(5):815-20.

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