DOI: http://dx.doi.org/10.24327/ijcmes2017.07090018

Available online at http://journalijcmes.com

INTERNATIONAL JOURNAL OF CURRENT MEDICAL SCIENCES

Research Article

ISSN: 2320- 8147

International Journal of Current Medical Sciences- Vol. 7, Issue, 09, pp. 286-290, September, 2017

CLINICO-EPIDEMIOLOGICAL PROFILE AND FACTORS PREDICTING THE OUTCOME OF PEDIATRIC ISOLATED TRAUMATIC BRAIN INJURY - A BICENTRIC STUDY IN EASTERN INDIA

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A R T I C L E I N F O

Article History:

Received 20th June, 2017
Received in revised form 3rd July, 2017
Accepted 27th August, 2017
Published online 28th September, 2017

Key words:

Traumatic brain injury, head injury, Glasgow coma scale, Glasgow outcome score.

A B S T R A C T

Introduction: Traumatic brain injury (TBI) in infancy and childhood is documented as the single most common cause of death and also leads to functional disability and psychosocial maladjustments in the survivors.

Methods: In this retrospective, bi-centric study on clinico- epidemiological profile, outcome and prognostic factors of traumatic brain injury in pediatric patients, all the case records of 220 children aged <12 years were reviewed and pertinent data (basic demographic, clinical, biochemical, and radiological data on admission and during ICU stay) were collected. They were followed up on outpatient basis for 1 year. Categorized data were analyzed by Chi-square test. Continuous variable were evaluated by Student's t-test. Risk factors were evaluated by multivariate analysis by a multiple logistic stepwise regression procedure. Odds ratios and risk ratios were estimated with their 95% confidence intervals (95%CI).

Results: Among 220 patients, 53.2%, 30.9% and15.9% suffered mild, moderate and severe head injury. 50% patients developed secondary systemic insults (SSIs). Neurosurgical procedures were needed in 25% cases. Mortality was 20%, brain herniation being the leading cause. Univariate analysis showed need for mechanical ventilation, anisocoria, SSIs, and low Glasgow Coma Scale scores to be the strongest predictors of mortality (p values<0.0001). Multivariate analysis showed that moderate [RR 1.7 (95% CI 1.3-2.1), p<0.0001] and severe head injury [RR 2.0 (95% CI 1.6-2.5), p<0.0001], hyponatremia [RR 1.4 (95% CI 1.2-1.8), p=0.005], nosocomial infections [RR 1.5 (95% CI 1.3-1.9), p=0.002] and presence of midline shift in CT brain [RR 1.7 (95% CI 1.3-2.1), p=0.004] were the independent risk factors for development of poor outcome. 49% had good outcome with low disability (Glasgow outcome score 5) and 7.2% had post-traumatic seizure disorder.

Conclusion: Head injury is commonest in 8-12 years age group, RTA being the commonest etiology, and mild injuries being the commonest type. Secondary systemic insults negatively affect outcome. GCS at admission can predict mortality, but it is not a significant predictor of long term outcome among survivors.

INTRODUCTION

The silent epidemic of traumatic brain injury (TBI) has taken hold in low and middle income countries, especially those undergoing socioeconomic changes. An estimated 1.5-2 million persons are injured and 1 million succumb to death every year in India [1]. Children<15 years constitute 35% of the total population and contribute to 20-30% of all head injuries [2]. The maturing brain passes through maximum synaptogenesis, increased levels of glucose metabolism, and higher neurotropic factors. Post-traumatic depression of neuronal activities often results in increased apoptosis and lost developmental potentials [3]. 35% of children have profound disturbances of cognitive abilities, physical, emotional and behavioural functioning after TBI, which result in functional disability and psychosocial maladjustment [4]. In children, mortality rate varies from 10-60% [5,6] and TBI in infancy and childhood has been documented as single most common cause of death [7]. Apart from the primary insult, the

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underlying pathophysiology also highlights the importance of the secondary processes of cerebral hypoxia and ischemia- which is often the leading cause of in-hospital deaths [8]. In this study we aim to analyze the clinic-epidemiological spectrum, outcome of pediatric TBI and evaluate the different predictors of mortality and long-term outcome.

METHODS AND MATERIALS

This retrospective study was conducted at the pediatric wards and intensive care units (PICU) of Dr B.C. Roy Post Graduate Institute of Pediatric Sciences, Kolkata and Institute of Post Graduate Medical Education & Research, Kolkata from July 2016 to June 2017. All the case records were reviewed and pertinent data [clinical history, age, sex, mode of injury, clinical findings (vitals, motor deficits, pupillary response, and convulsions), computed tomography (CT) findings, interventions (use of mechanical ventilation and inotropes, need of cardiopulmonary resuscitation for cardiac arrest), morbidity and mortality] were analyzed. Initially all the cases were scored according to GCS score and resuscitated at the pediatric emergency ward and then shifted to PICU, if needed. Treatment was individualized as per neurological status and CT findings. We assessed the outcome at discharge and at 12 months post discharge follow-up on outpatient basis using Glasgow Outcome Score (GOS). In the PICU, the head end was kept elevated for all patients. For clinical features of raised intracranial tension (ICT) and CT findings of herniation or cerebral edema, mannitol and 3% hypertonic saline (for hypotensive patients) were used. All patients requiring ventilation underwent hyperventilation ($PCO_{2=}$30-35 mm of Hg) and received sedation using fentanyl and midazolam combination. Appropriate neurosurgical procedures (decompression craniotomy, evacuation of hematomas) were done as necessary. Anticonvulsants were given if children developed seizures. Repeat CT scans were done when deemed necessary and therapies were re-directed. For each patient we recorded the development of secondary systemic insults (SSI) on admission and during ICU stay. SSI were divided into subgroups of circulatory (hypo/hypertension), metabolic (hypo/hypernatremia, hypo/hyperglycemia), respiratory (hypoxemia, hypo/hypercapnea), nosocomial infections (pneumonia, urinary tract infections, meningitis and septicaemia) and haematological (anaemia). We categorised the cases into mild, moderate and severe injury. Mild TBI results in loss of consciousness (LOC) of <30 minutes, an initial GCS of 13-15 and post-traumatic amnesia (PTA) lasting <24 hours. Moderate TBI results in a LOC of 30 minutes to 24 hours, initial GCS of 9-12 and PTA of 24 hours to 7 days. Severe TBI results in a LOC of >24 hours, an initial GCS of 3-8 and PTA of > 7 days.

Statistical analysis- Categorized data were expressed in proportions and analyzed by Chi-square test. Continuous variable were expressed as means (± standard deviations or SD) and sub-groups evaluated by Student's t-test. Risk factors were evaluated by multivariate analysis by a multiple logistic stepwise regression procedure. Odds ratios and risk ratios were estimated with their 95% confidence intervals (95%CI). For comparable data, p value<0.05 was considered statistically significant.

RESULTS

During the study period, 220 children aged <12 years were admitted in our PICU with isolated traumatic head injury and were all included in our study. 80 (36.4%) cases were brought to the hospital by traffic police, and 140 (63.6%) were brought by their family members. 96 (43.6%) were from Kolkata city and 124 (56.4%) were from neighbouring districts.

The demographic parameters are shown in **Table 1**

Parameters	Age < 4 years	4-8years	8-12 years
Sex : Male	22	46	82
Female	26	21	23
Mode of injury:			
RTA	12	39	70
Fall from height	19	20	24
Fall of heavy object over head	8	3	7
Fall while playing	4	4	3
Assault	5	1	2

Mean age of the cases was 8.3±4.5 years, with a male:female ratio of 2.14:1. Head trauma in road traffic accidents was commoner in older children and home/playground accidents were common in younger children. 117 (53.2%), 63 (28.6%), 40 (18.2%) children had mild, moderate and severe brain injury respectively.

The clinical parameters are shown in **Table 2**

Parameters	Age <4 years	4-8 years	8-12 years
1. Severity of head injury			
Minor	32	45	40
Moderate	10	16	37
Severe	15	10	15
2. Shock	9	15	26
3. Cardiac arrest	13	10	11
4. Hypertension	4	12	18
5. Hyperthermia	4	6	12
6. Hypoglycemia	6	8	11
7. Hyponatremia	15	21	34
8. Anemia needing PRBC transfusion	11	12	19
9. Hypoxemia	11	12	12
10. Hypercapnea	12	18	22
11. Hypocapnea	7	9	9
12. Hypernatremia	25	41	45
13. Infections and sepsis	12	25	31

Inotropes were used in 45 (20.5%) patients; dopamine in 45(20.5%), epinephrine in 25 (11.4%) and nor-epinephrine in 15 (6.8%). 108 (49.0%) developed nosocomial infections [46 (20.5%) developed pneumonia, 5 (2.2%) developed UTI, 7 (3.2%) developed meningitis and 10 (4.5%) developed septicaemia]. 44 (20%) required mechanical ventilation due to hypoxemia (paO2<80) and hypercapnea (paCO2>60). Mean duration of ventilation was 9±2.2 days. Mean duration of ICU stay was 16±2.8 days.

Neurosurgical procedures were needed in 55 (25%) cases craniotomy and evacuation of extra-dural bleed 25 (11.36%) , burr hole and evacuation of sub-dural bleed in 15 (6.82%), elevation of depressed fracture 8 (3.6%) and decompression craniotomy 7 (3.1%).

Table 3 shows the Computed Tomography findings among the patients

CT Scan findings	No. (%)
Normal	48 (21.9)
Diffuse cerebral edema	20 (9)
Cerebral contusion	28 (12.7)
Extradural hemorrhage	56 (25.4)
Subdural hemorrhage	26 (11.8)
Intraparenchymal/intraventricular hemorrhage	4 (1.8)
Pneumocephalus	1 (0.45)
Skull fracture	37 (16.8)
Midline shift due to mass effect	25 (11.4)

Table 4 shows the outcome of TBI

Parameters	Age <4 years	4-8 years	8-12 years
Mortality	3	20	21
GOS at 1 year after discharge			
Score 2	4	4	12
Score 3	6	8	8
Score 4	5	9	12
Score 5	30	26	52

Among 44 (20%) patients who died, 18 (8.1%) were within first 48 hours, 10 (4.5%) within 3-7 days and 16 (7.2%) thereafter. The leading causes of death was brain herniation in 26 (11.8%) and sepsis with multi-organ failure 16 (7.2%). Among 176 (80%) survivors, 48 (21.8%) had functional motor deficits and 16 (7.2%) had post traumatic seizure disorder.

Table 5 showing the factors responsible for mortality in univariate analysis

Parameters	Survivors	Death	p value
Age in years (mean±SD)	7.2±2.3	7.5±1.8	0.421
GCS score (mean±SD)	12±2.5	7±1.9	<0.0001
Shock requiring inotropes	32	13	0.02
Need for mechanical ventilation	0	44	<0.0001
Anisocoria	45	35	<0.0001
Intraparenchymal/intraventricular bleed	1	3	0.02
Midline shift	15	10	0.014
Secondary systemic insults	70	40	<0.0001

Thus the univariate analysis shows that low GCS scores, shock needing inotropes, need for mechanical ventilation, anisocoria, intraparenchymal/intraventricular bleeds, presence of midline shift and secondary systemic insults were associated with higher mortality.

DISCUSSION

In India, the incidence, mortality and case fatality rate due to TBI were 100/1lakh, 20/1lakh and 10% respectively [6]. In our study, the male:female ratio was 2.14:1. Although Sambasivam [9] reported an equal incidence of males and females in his series on the pediatric TBI, Chiaretti [10] hypothesized that the higher incidence of TBI in boys might be due to larger head circumference and more physical activities in comparison to girls.

Fall from height (cot, staircase, unprotected roof or balcony) was the most common mode (39.58%) of TBI in younger children <4 years, whereas RTA (road traffic accident) was the most common (66.67%) in 8-12 years children. Several studies have reported falls followed by RTA as the commonest cause of TBI in children [2,11]. However Ovalle [12] reported RTA to have higher incidence than falls (38.8% versus 26.7%). Teenage passion for bicycling and vulnerability for physical assaults during fights with peer groups may be responsible for TBI in 8-12 years age group. In our study, out of 121 cases of RTA, 65 (53.7%) were pedestrians which was comparable to other studies (51.25% as reported by Satpathy [2]). Assault was responsible for 8 (3.6%) cases, compared to 7.48% in the study of Satpathy [2]. Mild injury was the commonest type as described in other studies [3,13,14]. However, Udoh [15] found severe head injury commonest among the 127 studied patients. In their study, they excluded all mild head injuries that were neuro-radiologically normal, thus excluding a large number of mild head injuries, and reducing their relative percentage.

The CT scan reflects the seriousness of head injuries and predicts the clinical course. Eisenburg [16] found a good relationship between lesions resulting in increased intracranial pressure and death. According to 'Traumatic Coma Data Bank' classification mortality rates were significantly higher in type 6 (unoperated mass lesions) [17]. Extradural bleed occurs from focal impact injuries, whereas subdural bleed occurs from angular decelaration of the head, in which the brain continues to rotate relative to the more stationary skull and dura [18].

Table 6 showing multivariate analysis of the prognostic factors predicting adverse outcome

Parameters	GOS -1-4	GOS- 5	Odd's ratio (95%CI)	Risk ratio (95%CI)	p value
1. Severity of head injury:					
Mild	32	85	0.1(0.005-0.2)	0.3(0.2-0.4)	3.6
Moderate	45	18	3.3(0.7-2.5)	1.7 (1.3-2.1)	<0.0001
Severe	35	5	9.4(3.5-25)	2.0 (1.6-2.5)	<0.0001
2. Shock	30	20	1.6 (0.8-3.4)	1.24 (0.95-1.64)	0.09
3. Mechanical ventilation	44	0	-	-	<0.0001
4. Hypoglycemia	15	10	1.5 (0.6-3.5)	1.2 (0.8-1.7)	0.2
5. Hyperglycemia	28	24	1.7 (0.6-2.2)	1.1 (0.8-1.4)	0.63
6. Hyponatremia	45	25	2.2 (1.2-4)	1.4 (1.2-1.8)	0.005
7. Hypernatremia	6	5	1.2 (0.35-3.9)	1.0 (0.6-1.8)	0.5
8. Nosocomial infections	66	42	2.25 (1.3-3.8)	1.5 (1.3-1.9)	0.002
9. Skull fracture	20	17	1.2 (0.5-2.3)	1.1 (0.7-1.4)	0.4
10. Midline shift in CT brain	20	5	4.4(1.6-12.4)	1.7(1.3-2.1)	0.004

Thus multivariate analysis shows that moderate and severe head injury, need for mechanical ventilation, hyponatremia, nosocomial infection and midline shift were independent predictors of poor prognosis.

In the study of Debnath [19], 3 (25%) out of 12 patients required decompression craniectomy, 1(8%) required craniotomy and extradural hemorrhage evacuation and 1(8%) required burr hole evacuation of chronic subdural hemorrhage, compared to 3.1%, 11.36%, 6.82% in our study.

Table 7 shows comparison of CT findings with other studies

Parameters	Index study (%)	Bahloul [8] (%)	Satapathy [2] (%)	Bhargava [6] (%)
Normal	21.9	14.5		46
Cerebral edema	9	27	8.8	7.5
Extradural hemorrhage	25.4	13.4	29.9	16.5
Subdural hemorrhage	11.8	16.3	12.2	2.5
Pneumocephalus	0.4	11.2		
Depressed skull fracture	16.8	17	19.5	16.5
Intraventricular hemorrhage	1.8		1.4	2.5

Bahloul [8] reported 25% patients required craniotomy, 2.2% required subdural hematoma evacuation, 0.36% required decompressive craniectomy and 1.1% required lobectomy.

Univariate analysis showed that although presence of extra/subdural bleeds did not significantly affect mortality, however, intraparenchymal and intraventricular bleed and midline shift due to mass effect was significantly associated with mortality. Bahloul [8] described meningeal hemorrhage to be associated with mortality in univariate analysis. However Ovalle [12] found subarachnoid, subdural and extradural bleeds to be significantly associated with mortality, but other unspecified intracranial hemorrhage and cerebral laceration were not significantly associated. Satpathy [2] described that individual mortality risk for diffuse axonal injury, EDH, SDH and contusions were 28.5%, 11.6%, 5.56%, and 4.76%. GCS score is widely used as a guide to the severity of brain injury [8]. In our study the preintubation GCS was found to be associated with mortality in univariate analysis. However multivariate analysis did not show any correlation between GCS score and long term outcome. Several studies have shown good correlation between GCS score and neurological outcome [8,12,19,20,21,22]. Debnath [19] reported a statistical improvement in GCS score at the time of discharge when compared with the values during admission. A key component of neurologic assessment is the pupillary examination, and bilateral equal sized pupils with recovery of reflexes are a good predictor after brain trauma. If an abnormal pupil or pupillary response is present on the initial examination, it should be clearly documented. Fearnside [23] showed a significant difference in mortality according to whether both pupils reacted or not and bilateral mydriasis was clearly associated with mortality in the study of Bahluol [8]. In fact pupil reactivity is related to cerebral blood flow [24]. In our study, anisocoria was clearly associated ($p < 0.0001$) with mortality in the univariate analysis. Anisocoria may be an early sign of an impending neurologic emergency and suggestive of tumour compression, intracranial hypertension with impending uncal herniation, expanding intracranial aneurysm, or haemorrhage [25].

Very few pediatric studies evaluated the effect of secondary systemic insults on the outcome of head injury.

Table 8 compare the systemic secondary insults incidence with the study of Bahloul [8].

Parameters	Index study (%)	Bahloul [8] (%)
Hyponatremia	31.8	27.5
Hypernatremia	5	6.9
Hypotension	27.3	26.4
Hyperglycemia	23.6	17.4
Hypoglycaemia	11.4	2.2
Hypoxemia	15.9	10.5
Hypercapnea	23.6	8.7
Anemia	19.1	11.6
Sepsis	4.5	2.1
Pneumonia	20.9	9.7
Urinary tract infection	2.2	3.2
Meningitis	3.2	2.1

Both the studies demonstrate the negative influence of secondary systemic insults. Univariate and multivariate analysis showed that secondary systemic insults were associated with poor GOS score and mortality. Multivariate analysis showed that moderate [RR 1.7 (95% CI 1.3-2.1), $p<0.0001$] and severe head injury [RR 2.0 (95% CI 1.6-2.5), $p<0.0001$], hyponatremia [RR 1.4 (95% CI 1.2-1.8), $p=0.005$], nosocomial infections [RR 1.5 (95% CI 1.3-1.9), $p=0.002$] and midline shift [RR 1.7 (95% CI 1.3-2.1), $p=0.004$] were the independent risk factors for development of poor outcome. Although Bahloul [8] described hyperglycemia as important risk factor, our study did not find it to be significant [RR 1.1 (95% CI 0.8-1.4) $p=0.63$].

Table 9 compares the outcome of index study and other studies

Parameters	Index study (%)	Bahloul [8] (%)	Debnath [15] (%)	Nnadi [3] (%)	Satpathy [2] (%)	Smits [26]
Death	20	17.4	8	7.9	7.48	4.2
Persistent vegetative state	9.1	0.7	0		5.45	
Severe disability	10		42		8.85	3
Moderate disability	11.8		33	5.26	9.52	30
Good recovery	49	63.8	16	92.1	68.7	63
Post traumatic seizure disorder	7.2			15.9	8.85	

Thus, in our study the mortality rate are higher, possibly because of the higher incidence of secondary systemic insults in our study. However, marginally higher mortality (20.50%) than in our study has also been reported [18]. Mortality in developing countries is often attributed to late arrival, unpreparedness for surgery and low socio-economic status.

Our study has few limitations. Firstly, bring a retrospective study; it suffers from incomplete and inconsistent information. Secondly, in our ICU invasive intracranial pressure monitoring was not done and we relied on clinical examination (pupillary response, Cushing's triad, motor deficits, and convulsions).

In conclusion, our study adds data that head injury is commonest in 8-12 years age group, RTA being the commonest etiology, and mild injuries being the commonest type. It emphasizes the negative impact of secondary systemic insults on outcome; nosocomial infections and hyponatremia being the most significant among them. The study also establishes the fact that although the GCS at admission can predict mortality, but

it is not a significant predictor of long term outcome among survivors. Severe TBI has poor outcome; but the outcome may be improved if patients are resuscitated and referred early to equipped centres and managed on individualized basis with an organized team approach.

Funding-none, Conflict of interest- none.

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