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Original Research Article

A Prospective Study of Traumatic Brain Injury in Geriatric Patients

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Abstract:

Introduction and background: The percentage of senior citizens in India has been growing at an increasing rate in recent years and the trend is likely to continue. Traumatic Brain Injury (TBI) still remains a major cause of morbidity and mortality. In developing countries like India where motorized travel is quickly growing without accompanying safety precautions and adherence to traffic rules and regulations, road traffic accidents are more frequent leading to increase in traumatic brain injuries. Elderly patients sustaining TBI have higher morbidity and mortality and prolonged recovery trajectories than younger patients. The current "One size fits all" approach to traumatic brain injury management of adult patients neglects special issues of elderly population.

Aims: Aim of the study is to analyze demographic data, clinico-radiological features, co-morbid illness of elderly patients with traumatic brain injuries treated either with surgical or conservative management, to evaluate the risk factors predicting poor prognosis among elderly TBI patients, to study the outcome and mortality among elderly (65-75) and very elderly (>75 years) age groups and to study the validation of eTBI (Elderly Traumatic Brain Injury) score in predicting mortality.

Methodology: This is a prospective single center study conducted at the Institute of Neurosurgery, Madras Medical College, Chennai, Tamilnadu, India. We enrolled 200 patients of ≥ 65 years of age with Traumatic brain Injury between May 2021 and October 2021. Information about patients, demographic data, clinical and radiological features, co-morbid conditions, in-hospital course of treatment and associated complications as well as outcome was recorded. The collected data were analyzed with IBM-SPSS statistics software 23.0 Version. To find the significance in categorical data Chi-Square test was used. The probability value .05 is considered as significant level.

Results: Mean age among the patients is 70.16 and maximum age enrolled in this study is 88. 144 were males and 38 were females. There were 91 patients with mild injury (50%), 68 patients with moderate injury (37.4%) and 23 patients with severe injury (12.6%). About 127 (69.8%) patients had favorable outcome and 55 (30.2%) patients had unfavorable outcome. Road traffic accidents accounted for most injuries (n=100) followed by ground level falls (n = 74) with other causes accounting for very few numbers. Most common associated comorbidity found was systemic hypertension which was seen in 34.1% followed by diabetes in 25.3%. Statistical analysis showed poor GCS score at admission, absent/effaced basal cisterns and/or presence of midline shift in CT, presence of co-morbidities like diabetes, hypertension and CAD, preinjury intake of antiplatelets or anticoagulants or beta blockers were all associated with unfavorable outcome. Lower eTBI scores were associated with unfavorable outcome. Prolonged ICU stay and prolonged mechanical ventilation both of which was found to adversely affect the clinical outcome in our study.

Keywords: Traumatic Brain Injury, Elderly population, Treatment outcomes.

Introduction

The percentage of senior citizens in India has been growing at an increasing rate in recent years and the trend is likely to continue. According to 2001 census people aged 65 to 79 years constituted4% of the population whereas people aged 80 and above constituted 0.8% [1]. As per United Nations Population Division's World Population Prospects: 2019 revision, 6.378% of total Indian population were aged 65 years and above [2]. It is estimated to increase to nearly 20% in 2050.India also recorded an improvement in life expectancy at birth, which was 47 years in 1969, growing to 60 years in 1994 and 69 years in 2019. It is estimated that by 2050, there

will be more people older than 60 years than those below 15 years.[1]

Traumatic Brain Injury (TBI) still remains a major cause of morbidity and mortality. In developing countries like India where motorized travel is quickly growing without accompanying safety precautions and adherence to traffic rules and regulations, road traffic accidents are more frequent leading to increase in traumatic brain injuries. In most developed nations incidence of TBI is falling in younger age group. This decline has been attributed to successful RTA prevention programs leading to fewer road traffic accidents. This decrease has not been seen in elderly population whose injuries more often occur with falls.

Age is an important prognostic factor in TBI and this has long been recognized. This has been confirmed in many studies including earliest prognostic model of Heiden et al., in 1979 [3] and more recent figures from IMPACT group (Mushkudiani et al., 2007) [4]. Old age is also associated with comorbidities like diabetes, hypertension, coronary artery disease etc which may worsen the outcome. In addition many elderly people use anti-platelet and anti-coagulant medications which may worsen the brain injury. Elderly people also present with alterations in cognition, mood, behavior and memory which can hamper rehabilitation after TBI (Glorioso and Sibille, 2011) [5]. Biologically aged brain is more vulnerable to TBI with less plasticity and repair after injury (Kovacs, 2005) [6]. Unsurprisingly, elderly patients sustaining TBI have higher morbidity and mortality [9] and prolonged recovery trajectories than younger patients[10]. Consequentlythis may induce less aggressive treatment for elderly cases. Some centers even impose age cut-offs for neuro-intensive care admissions and neurosurgicalinterventions [11,12].

In general, TBI outcomes have improved with time (Lu et al.,2005 and Stein et al., 2010) [7,8]. This progress also includes elderly population who has been invariably considered as unsalvageable. The current "One size fits all" approach to traumatic brain injury management of adult patients neglects special issues of elderly population.

This study addresses the epidemiology of TBI in geriatric patients, factors affecting prognosis and outcome among geriatric age group and analyses management and outcome of such patients.

Review of Literature

Epidemiology- Indian Scenario: Traumatic Brain Injury remains the major cause of morbidity, mortality, disability and socioeconomic loss in India. Around 1.5 to 2 million persons sustain TBI and 1 million succumb to it every year in India. Road Traffic Accidents remain the leading cause of TBIs (60%) followed by falls (20-25%) and assault (10%).

Alcohol intake was present among 15 to 20% of persons with TBIs at the time of admission. Maharashtra reported highest rate of accidental deaths at 16.7% followed by Madhya Pradesh at 9.3% and Tamil Nadu the third highest accidental deaths at 8.4% according to 2010 data.[13,14]

Adolescent and young adults are at highest risk of both fatal and non-fatal traumatic brain injuries due to road traffic accidents. In contrast falls are more common in young children and elderly members of our society although severity profiles of these two groups are different. In children falls are common but not severe. In older age group even a trivial fall can cause fatal traumatic brain injury.

Pathology of TBI: Traumatic Brain Injury is an insult to the brain caused by external physical force resulting in functional disability. TBI has been classified into primary and secondary injury. The primary injury occurs at the time of trauma causing skull fractures, brain contusions, lacerations, diffuse axonal injuries (DAI), vascular tearing and intracranial hemorrhages. Secondary injuries are due to further cellular damage from the effects of primary injuries. Primary injuries trigger a sequence of events which result in alterations in brain metabolism, disruption of cerebral blood flow and cerebral edema. These secondary alterations can produce increased intra-cranial pressure and brain herniation. Secondary changes ultimately lead to cerebral ischemia and cerebral hypoxia.

Classification of Primary Injuries		
Skull Fractures	**Local Injuries**	**Diffuse Injuries**
Linear Basilar Depressed Comminuted Childhood	**Contusions** Coup Contrecoup	**Concussion**
	Lacerations	**Diffuse Axonal Injury**
	Hematomas Epidural Subdural Subarachnoid Intracerebral Intraventricular	**Cerebral Edema**

Skull Fractures: The presence of a skull fracture signifies that traumatic cranial injury has occurred. Skull fractures occur in up to 8 percent of fatal head injuries.[15] Concomitant brain injuries also occur frequently, but they are by no means an invariable consequence of skull fractures.

Linear Fractures: A linear fracture is a simple fracture line that tends to radiate outwards from the point of impact along with path of least resistance. A linear fracture is produced by a broad-based force such as traffic accidents and falls.

Basilar Skull Fracture: Fracture of skull may be limited to the area of impact or may spread into the base from the calvarium. Fractures of the base of skull are not reliably depicted by radiographic examination.[16]

Depressed Fracture: These types of fractures are produced by forceful or heavy impacts striking the skull over a small surface area, resulting in a portion of the bone, being pushed inwards to impinge on the brain. When fractures separate the suture lines, they are called as diastatic fractures.

Comminuted Fracture: These types of fractures result from forceful and heavy impact striking the head over a wider area with bone breaking into multiple pieces.

Contusions

A contusion is a bruise of the cortical surface of the brain which results in a haemorrhage around the blood vessel.

The overlying pia mater always remains intact. Contusions are usually result of mechanical injury and extent depends upon the forcefulness of the impact. Contusions are focal damage and even in severe cases, there may be complete recovery if there is no accompanying DAI.

Contusions tend to be large and more haemorrhagic in hypertensive, alcoholic or who have bleeding diathesis or on anticoagulant medications. Contusions formed at the site of cranial impact are coup contusions and those formed opposite the cranial impact are contrecoup contusions.

Lacerations: A laceration is a mechanical tear or rent in normal tissue. They may or may not be accompanied by skull fracture. Cerebral lacerations usually involve inferior surface of frontal lobe or temporal lobe tips.

Epidural Hematoma: Epidural hematoma follows cranial trauma complicatedby skull fracture and accompanying blood vessel injury, most commonly caused by temporal bone fracture and laceration of middle meningeal artery branches that penetrate the skull in the regions of the pterion. The accumulation of blood between calvarium and endosteal surface of the dura mater is rapid and is accompanied by rapid deterioration of consciousness. If not evacuated promptly, it leads to trans-tentorial herniation and compression of brainstem.

Subdural Hematoma: Normally the space between dura mater and arachnoid is closely opposed. Subdural hematomas result from the dissection of blood into this potential space. Most common location is over the cerebral convexities and it is supposed to be because of rupture of delicate cortical bridging veins that traverse the subdural space enroute to the superior sagittal sinus. Patients on anticoagulants are more prone to subdural hematoma. Subdural hematomas may be acute, subacute or chronic, depending upon the interval of injury and onset of symptoms.

Subarachnoid Haemorrhage: Trauma is the most frequent cause of subarachnoid haemorrhage. Subarachnoid haemorrhage is found overlying contusions, spreading outwards from lacerations. Within a few hours of bleeding into the CSF, fever and meningismus develop. There is polymorphonuclear response within 24 hours and becomes prominent by 48 hours. After 48 hours, lymphocytes and macrophages start replacing them. Macrophages phagocytose RBCs and such lipid laden in phagocytes may persist for years in the arachnoid meninges and Virchow-Robin's spaces. Repeated episodes of subarachnoid haemorrhage may impede the absorption of CSF and may produce hydrocephalus.[17]

Intracerebral Haemorrhage: Intracerebral haemorrhage varies in size from a small petechial haemorrhage of less than 0.5 cm to a large hematoma and may be single or multiple in number. The large intracerebral hematomas are most commonly located in frontal and temporal lobes and are associated with adjacent contusions and lacerations. Large parenchymal haemorrhages may extend into the ventricles, resulting in IVH.[18]

Intraventricular Haemorrhage: Intraventricular haemorrhage is bleeding within the ventricles that may extend into subarachnoid space. Traumatic intraventricular haemorrhage occurs commonly in association with other intracranial injury. The volume of blood varies from few ml to large haemorrhage of 40 to 60 ml.

Burst Lobe: A burst lobe is severely contused frontal or temporal lobe, whose cortical surface is pulped and lacerated with extension of haemorrhage through the arachnoid into subdural space. Burst lobe occurs most commonly in association with contrecoup contusions.

Concussion

A cerebral concussion (commotio cerebri) is a temporary, reversible neurologic deficit caused by trauma, which results in an immediate but tempo-

rary loss of consciousness. Concussion may be accompanied by retrograde and post-traumatic amnesia of variable duration which depends upon the severity of concussion. When concussion lasts more than 24 hours or longer, diffuse brain injury is usually present. Experimental studies of concussion have shown minimal nonspecific neuronal changes. Neuropathology studies of classical cerebral concussion in humans have rarely been available to study.[19,20]

Diffuse Axonal Injury

The diffuse axonal injury was first described by Strich in 1956 as diffuse degeneration of the white matter and was thought to be essential component of posttraumatic dementia. Later on, some authors called it as inner cerebral trauma [21] and shearing injury.[22] The currently accepted name used to describe this entity is diffuse axonal injury (DAI).[23,24]

Axonal injury is a key feature of the post-traumatic encephalopathies and is an important determinant of the outcome in non-missile head injury. DAI is an important cause of coma in the absence of an intracranial expanding lesion, of the vegetative state and of disability after head injury. Patients with moderate to severe diffuse axonal injury are rendered immediately unconscious by their injury and remain unconscious until their death. Those who survive for a longer period are usually in vegetative states. These patients survive longer than severely diffuse head injured patients without axonal injury.[19]

In DAI, grossly brain may appear normal. But in most severe degree of DAI, focal lesions are seen in corpus callosum, one or both dorsolateral quadrants of the rostral brainstem. In the early stage ofinjury the lesions are haemorrhagic and appear as petechial size haemorrhages, streaks of haemorrhages or as small intraparenchymal haemorrhage. Similar haemorrhages occur, in addition to corpus callosum and brainstem, in subcortical white matter, fornix, telachoroidea, walls of third ventricle, basal ganglia and hippocampal regions. In very severe cases larger streaks of haemorrhage are seen in the white matter.

On microscopic examination, the hallmark of DAI is axonal swelling (retraction balls) in the cerebral white matter, corpus callosum and upper brainstem. Axonal swelling becomes apparent within hours of the injury and may persist for a year or more. In patients who survive, microglial stars (clusters of microglial cells) develop at the site of injury and replace the swollen axons.

Diffuse axonal injury can be diagnosed by diffusion-weighted MRI. If survival is from hours to a few days many irregular swellings of the axons and oval or rounded bulbs at the end of the axon are eosinophilic in H and E stain sections. Axons may appear tortuous or varicosed and show irregular thickening. These findings are better brought out in silver staining. Identification of axonal swelling can be augmented by staining for ubiquitin or amyloid precursor protein (APP). If patient survives for many months in vegetative state then Wallerian degeneration appear.

Possession of e4, allele of the apolipoprotein E gene (APOE e4) is associated with higher incidence of moderate/severe contusional injury and severe ischemic brain damage in fatal cases of TB1. This may also be associated with poor outcome.[25]

Adams et al [26] proposed a grading system based on the severity of injury.

1. In grade I DAI, the histological evidence of axonal damage is found in the white matter of the cerebral hemispheres;
2. In grade II DAI, in addition there is also focal lesion in the corpus callosum
3. In Grade III DAI, there is diffuse damage to axons and focal lesions in both corpus callosum and dorsolateral quadrant or quadrants of the rostral brainstem. It represents the most severe spectrum of DAI.

Materials and Methods

Study Design & Patient Characteristics: This is a prospective observational study and this work has been compared and contrasted with related works available in the literature. The period of this study was six months. This prospective single center study enrolled 200 patients ≥ 65 years of age with Traumatic brain Injury between May 2021 and October 2021. Information about patients, demographic data, clinical and radiological features, their comorbid conditions, in-hospital course of treatment and associated complications as well as outcome was recorded. Patients were followed up for 3 months after discharge. For the purpose of analysis patients were divided into two age groups (65-74 years and ≥ 75 years) and two outcome groups (Favorable – GOS scores 4 or 5 and Unfavorable – GOS score 1,2,3).

Study Centre: This study was conducted at the Institute of Neurosurgery, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai, Tamilnadu, India from May 2021 to October 2021after obtaining proper clearance from the Institutional Ethics Committee.

Inclusion Criteria: All patients aged 65 years and above with evidence of traumatic brain injury in CT or MRI brain were included in this study.

Exclusion Criteria: The following patients were excluded from the study

1. Patients with age less than 65 years were excluded from this study.
2. Patients declared dead within 6 hours of admission were not included.
3. Patients with concomitant severe injuries (blunt injury abdomen / spine injuries) were excluded.

Statistical Analysis: The collected data were analyzed with IBM-SPSS statistics software 23.0 Version. To describe the data descriptive statistics frequency analysis was used, percentage analysis was used for categorical variables and the mean & S.D were used for continuous variables.

To find the significance in categorical data Chi-Square test was used. The probability value <0.05 is considered as significant level.

Two hundred patients were enrolled in our study. 8 patients went against medical advice and another 10 patients were lost in follow up. Hence, 182 patients were finally included in our study and analysis.

Results

Age Distribution

Table 1 A: Descriptive Analysis of Age Distribution

Mean Age	70.16
Standard Deviation	5.156
Minimum Age	65
Maximum Age	88
Range	23

Table 1 B: Frequency Distribution among Age Groups

Age Group	Frequency	Percent
65 to 74 Years	139	76.4%
≥ 75 Years	43	23.6%

Gender Distribution

Table 2 A: Gender distribution among Age Groups

Gender	Age Group		Total	Percent
	65 to 74 Years	≥ 75 Years		
Male	113	31	144	79.1%
Female	26	12	38	20.9%

Among the total number of patients (n=182), 144 were males and 38 were females (Table 2-A). Most of the patients admitted fell under the age group 65 to <75 (n=139) with males comprising 113 and females comprising 26. In the ≥ 75 Years (n=43) age group, there were31 males and 12 females. Mean age among the patients was 70.16 and maximum age enrolled in this study was 88 years.

Outcome Analysis

Table 3 A: Frequency Distribution among outcome groups

Outcome	Frequency	Percent
Favourable	127	69.8%
Unfavourable	55	30.2%

Glasgow Outcome Scale (GOS)

Table 3 B: Frequency distribution of GOS at discharge and GOS at 3 months

Glasgow Outcome Scale	At Discharge		At 3 Months Followup	
	Frequency	Percent	Frequency	Percent
Death	34	18.7%	34	18.7%
Persistant Vegetative State	3	1.6%	0	0%
Severe Disability	17	9.3%	13	7.1%
Moderate Disability	64	35.2%	47	25.8%
Good Recovery	64	35.2%	88	48.4%

We measured the clinical outcome with Glasgow outcome score at discharge and at 3 months after discharge. The frequency distribution was as follows: Patients who expired during the study period were Grade 1 (n=34). A few patients who were in the Persistent vegetative state Grade 2 (n=3) at discharge improved during follow-up and at the end of 3 months none were in Grade 2(n= 0). This was

followed by Severe Disability Grade 3 (n=17) and Moderate Disability Grade 4 (n=64) at discharge. Good recovery (Grade 5) was seen in 35% patients (n = 64) at the time of discharge and 48.4% patients (n=88) at the end of 3 months. (Table 3-B). Patients were also dichotomized into Favorable (GOS 4 and GOS 5) and Unfavorable (GOS 1, GOS 2 and GOS 3) outcome groups for the purpose of statistical analysis: About 127 (69.8%) patients were in favorable outcome group and 55 (30.2%) patients had unfavorable outcome. (Table 3-A)

GCS/ Clinical Severity vs GOS

The Glasgow Coma Score of the patients was calculated on admission and patients were classified into three categories based on their GCS, Mild (14-15), Moderate (9-13) and Severe (3-12) injuries and its relation to the outcome was analyzed. There were 91 patients with mild injury (50%), 68 patients with moderate injury (37.4%) and 23 patients with severe injury (12.6%). (Table 4-A) Statistical analysis was then made to compare the clinical severity with the GOS and it showed p-value of 0.000 ($p < 0.05$), which meant a significant correlation between the clinical severity and the Glasgow outcome scale. (Table 4-B, Table 4-C).

Table 4 A: Frequency Distribution of Clinical Severity / GCS

Severity	GCS	Frequency	Percent
Mild	13-15	91	50%
Moderate	9-12	68	37.4%
Severe	3-8	23	12.6%

Table 4 B: Statistical analysis for correlation between GCS and outcome

		Outcome		Total
		Favourable	Unfavourable	
Severity	Mild GCS 13-15	90	1	91
	Moderate GCS 9-12	37	31	68
	Severe GCS 3-8	0	23	23
Total		127	55	182

Table 4 C: Chi-Square Tests: GCS * Outcome

	Value	Df	Asymp. Sig. (2- sided)
Pearson Chi-Square	97.321	2	.000
Likelihood ratio	118.280	2	.000
N of valid cases	182		

It can be seen that the clinical severity, measured by the GCS score on admission correlated well with the outcome of the patient and it had a statistical significance - the more the initial GCS, the better was the clinical outcome and vice-versa.

Mode of Injury Vs Outcome

Road traffic accidents accounted for most injuries (n=100) followed by ground level falls (n = 74) with other causes accounting for very few numbers (n=8) including 3 case of assault, 3 cases of fall from height and 2 unknown modes of injury (Table-5).

Table 5: Frequency distribution of Mode of Injury

Mode of Injury	Frequency	Percent
Road Traffic Accidents	100	54.9
Ground level fall	74	40.7
Fall from height	3	1.6
Assault	3	1.6
Unknown	2	1.1

Co-Morbidities Distribution

Table 6 A: Frequency distribution of co-morbidities:

Co-Morbidities	Frequency	Percent
Diabetes	46	25.3%
Hypertension	62	34.1%
CAD	35	19.5%
CVA	14	7.7%
Renal	4	2.2%
Malignancy	3	1.6%
Pulmonary	3	1.6%

Most common associated comorbidity was systemic hypertension which was seen in 34.1% (n=62) followed by diabetes in 25.3%(n=46) and coronary artery disease in 19.5% (n=35) patients. 14 patients had previous CVA, 4 patients have chronic kidney disease, 3 patients have systemic malignancies (2- Lung Ca,1-Breast Ca) and 3 patients have COPD. (Table 6-A)

Table 6 B: Statistical analysis for correlation between co-morbidities and outcome

		Outcome		TOTAL
Co-Morbidities		Favourable	Unfavourable	
Diabetes	Present	24	22	46
	Absent	103	33	136
Hypertension	Present	27	35	62
	Absent	100	20	120
CAD	Present	19	16	35
	Absent	108	39	147
CVA	Present	8	6	14
	Absent	119	49	168
Renal	Present	3	1	4
	Absent	124	54	178
Malignancy	Present	3	0	3
	Absent	124	55	179
Pulmonary	Present	3	0	3
	Absent	124	55	179

Table 6 C: Chi-Square Tests: Co-Morbidities * Outcome

Co-Morbidities		Value	df	Sig.(2-sided)
Diabetes	Pearson Chi-Square	9.049	1	.003
	Likelihood Ratio	8.627	1	.003
Hypertension	Pearson Chi-Square	30.684	1	.000
	Likelihood Ratio	29.978	1	.000
CAD	Pearson Chi-Square	4.933	1	.026
	Likelihood Ratio	4.676	1	.031
CVA	Pearson Chi-Square	1.149	1	.284
	Likelihood Ratio	1.085	1	.298
Renal	Pearson Chi-Square	.053	1	.818
	Likelihood Ratio	.055	1	.815
Malignancy	Pearson Chi-Square	1.321	1	.250
	Likelihood Ratio	2.181	1	.140
Pulmonary	Pearson Chi-Square	1.321	1	.250
	Likelihood Ratio	2.181	1	.140

In statistical analysis very significant association was found between outcome and the following co-morbidities: Diabetes (p-value=0.003), Hypertension (p-value=0.000) and CAD (p-value=0.026). Presence of any of these co-morbidities is strongly associated with unfavourable outcome. There is no statistically significant association between other co-morbidities (CVA, CKD, COPD, Malignancies) and outcome.

CT Findings

Table 7: Frequency distribution of CT findings

	Present		Absent	
	Frequency	Percent	Frequency	Percent
EDH	6	3.3%	176	96.7%
IVH/SAH	49	26.9%	133	73.1%
Acute SDH	53	29.1%	129	70.9%
Chronic SDH	25	13.7%	157	86.3%
Skull Fractures	70	38.5%	112	61.5%
Contusion	55	30.2%	127	69.8%
ICH	11	6%	171	94%
Normal	26	14.3%	156	85.7%

Out of the 182 cases, CT brain was normal in 14.3% (n=26) patients. Skull vault fractures were most common finding noted in 38.5% (n=70) patients followed by cerebral contusion in 30.2% (n=55) and acute SDH in 29.1% (n=53). IVH/SAH noted in 26.9% (n=49) and chronic SDH in 13.7% (n=25) patients. (Table 7)

Status of Basal Cisterns

Table 8 A: Frequency distribution of Basal Cisterns Status

Basal Cisterns	Frequency	Percent
Open	115	63.2%
Compressed	54	29.7%
Absent	13	7.1%

Midline Shift

Table 8 B: Frequency distribution of Midline Shift

Midline Shift	Frequency	Percent
Present	52	28.6%
Absent	130	71.4%

Table 8 C: Statistical analysis of Basal cisterns / Midline shift vs Outcome

		Outcome		Total
		Favourable	Unfavourable	
Basal Cisterns	Open	104	11	115
	Compressed	23	31	54
	Absent	0	13	13
Total		127	55	182
Midline Shift	Present	24	28	52
	Absent	103	27	130
Total		127	55	182

Out of the total 182 cases, basal cisterns were absent / completely obliterated in 13 cases (7.1%) and all of them eventually had unfavourable outcome. Basal cisterns were partially compressed in 29.7% (n= 54) and open in 63.2% (n=115). Midline shift was present in 28.6% (n=52) patients and 53.8% among them (n=28) had unfavorable outcome. (Table 8-C, Table 8-D).

Table 8 D: Chi-Square tests: Basal Cisterns / Midline Shift Vs Outcome

Basal Cisterns / Midline Shift		Value	df	Sig.(2-sided)
Basal Cisterns	Pearson Chi-Square	72.212	2	.000
	Likelihood Ratio	76.811	2	.000
Midline Shift	Pearson Chi-Square	19.271	1	.000
	Likelihood Ratio	18.419	1	.000

On statistical analysis, both the status of basal cisterns (p-value=0.000) and midline shift (p-value=0.000) has statistically significant association with patient outcome. Presence of midline shift or absence of basal cisterns has very strong association with unfavorable outcome. (Table 8-C, Table 8-D)

Duration of Hospital Stay / ICU STAY / Ventilator Support

Table 9 A: Descriptive analysis of duration of hospital stay/ ICU days/ Ventilator days

	Length of Hospital Stay	Number of ICU Days	Number of Days in Mechanical Ventilator Support
Mean	7.62	2.73	1.30
Standard Deviation	4.370	2.999	2.334
Minimum	2	0	0
Maximum	29	13	11
Range	27	13	11

Table 9 B: Statistical analysis of duration of hospital stay/ ICU days/ Ventilator days vs outcome

Variable		Value	Df	Sig.(2-Sided)
Length of Hospital Stay	Pearson Chi-Square	28.361	19	.077
	Likelihood Ratio	32.991	19	.024
Number of ICU Days	Pearson Chi-Square	92.916	13	.000
	Likelihood Ratio	113.764	13	.000
Number of Days in Ventilator Support	Pearson Chi-Square	124.067	10	.000
	Likelihood Ratio	132.838	10	.000

As evident from table 9-A, mean duration of hospital stay was 7.62 and maximum hospital stay was 29 days. Mean duration of ICU stay was 2.73 and maximum ICU stay was 13 days. Statistical analysis showed strong association between number of ICU days (p-value=0.000), number of days in mechanical ventilation (p-value=0.000) with outcome. More the number of days in ICU or ventilator more is the likelihood of unfavorable outcome. There was no association between length of hospital stay (p-value=0.077) and outcome.

Medications

Table 10 A: Frequency distribution of medication intake

	Present		Absent	
	Frequency	Percent	Frequency	Percent
Antiplatelets &/Or Anticoagulants	41	22.5%	141	77.5%
Beta Blockers	33	18.1%	149	81.9%
Statins	39	21.4%	143	78.6%

In our study 22.5% (n=41) patients were on either antiplatelets or anticoagulants or both out of which 18 patients had unfavorable outcome. Out of 21.4% (n-33) patients who were on statins, 15 had unfavorable outcome and among 18.1% (n=39) patients who were on beta blockers, 20 had unfavorable outcome. (Table 10-A, Table 10-B).

Statistical analysis showed significant association between intake of all three medications and unfavorable outcome with p-values < 0.005. {Antiplatelets/Anticoagulants (p-value= 0.030) and Beta Blockers (p-value= 0.000)}. In our study we found no association between statin intake (p-value=0.206) and outcome. (Table 10-C)

Table 10 B: Statistical analysis for correlation between medications and outcome

		Outcome		Total
Medications		Favourable	Unfavourable	
Antiplatelets &/Or Anticoagulants	Yes	23	18	41
	No	104	37	141
Beta Blockers	Yes	13	20	33
	No	114	35	149
Statins	Yes	24	15	39
	No	103	40	143

Table 10 C: Chi-Square test: Medication Intake vs Outcome

Medications		Value	df	Sig.(2-sided)
Antiplatelets &/Or Anticoagulants	Pearson Chi-Square	4.698	1	.030
	Likelihood Ratio	4.492	1	.034
Beta Blockers	Pearson Chi-Square	17.649	1	.000
	Likelihood Ratio	16.328	1	.000
Statins	Pearson Chi-Square	1.559	1	.206
	Likelihood Ratio	1.550	1	.213

Marshall CT Classification

Table 11 A: Frequency distribution of Marshall CT classification

Category	Frequency	Percent
I	26	14.3%
II	86	47.3%
III	24	13.2%
IV	8	4.4%
V	35	19.2%
VI	3	1.6%

Patient's Marshall scores were calculated from the CT brains and then their correlation to the clinical outcome was then statistically analyzed. Most of the patients had a score of 2 (n=86), followed by scores of 5 (n=35), 1 (n=26) and 3 (n=24).

Very few patients were in the scores 4 (n=8) and 6 (n=3). The statistical analysis showed p – value of 0.000 and there was significant statistical correlation between Marshall score and the clinical outcome. (Tables 11-A and 11-B)

Table 11 B: Statistical analysis for correlation between Marshall CT classification and outcome

		Outcome		Total
		Favourable	Unfavourable	
Marshall CT Category	I	22	4	26
	II	78	8	86
	III	3	21	24
	IV	2	6	8
	V	22	13	35
	VI	0	3	3
Total		127	55	182

Table 11 C: Chi-Square Tests: Marshall CT Classification * Outcome

	Value	df	Asymp. Sig. (2- sided)
Pearson Chi-Square	73.230	5	.000
Likelihood ratio	74.211	5	.000
N of valid cases	182		

Rotterdam CT Score

Table 12 A: Frequency distribution of Rotterdam Score

Rotterdam CT Score	Frequency	Percent
1	0	0
2	75	41.2
3	52	28.6
4	39	21.4
5	16	8.8
6	0	0

Similar to Marshall score, Rotterdam score was also calculated from the patients CT findings and its correlation to clinical outcome was analyzed. Most of the patients had score of 2 (n=75), followed by 3 (n=52), 4 (n=39) and 5 (n=16). (Table 12-A)

Statistical analysis showed p – value of 0.000 and there was significant statistical correlation between Rotterdam score and the clinical outcome. (Table 12-B, Table 12-C)

Table 12 B: Statistical analysis for correlation between Rotterdam CT score and Outcome

		Outcome		Total
		Favourable	Unfavourable	
Rotterdam CT Score	1	0	0	0
	2	71	4	75
	3	39	13	52
	4	16	23	39
	5	1	15	16
	6	0	0	0
Total		127	55	182

Table 12 C: Chi-Square Tests: Rotterdam CT Score * Outcome

	Value	df	Asymp. Sig. (2- sided)
Pearson Chi-Square	68.615	3	.000
Likelihood ratio	73.030	3	.000
N of valid cases	182		

Elderly Traumatic Brain Injury Score

Table 13 A: Frequency distribution of Rotterdam Score

eTBI Score	Frequency	Percent
-2	1	0.5%
-1	2	1.1%
0	3	1.6%
1	6	3.3%
2	9	4.9%
3	20	11.0%
4	42	23.1%
5	55	30.2%
6	44	24.2%

Elderly Traumatic Brain Injury (eTBI Score) was calculated for all patients based on clinical and laboratory values. Most of the patients had a score of 5 (n=55), followed by scores of 6 (n=44), 4 (n=42) and 3 (n=20). Very few patients had scores of -2, -1, 0, 1 and 2 as shown in table 13-A.

Statistical analysis showed p-value of 0.000 showing significant correlation between eTBI scores and outcome. More the scores the better is the outcome among elderly patients with traumatic brain injury. (Table 13-b, Table 13-c)

Table 13 B: Statistical analysis for correlation between elderly traumatic brain injury score and outcome

		Outcome		Total
		Favourable	Unfavourable	
eTBI-Score	-2	0	1	1
	-1	0	2	2
	0	0	3	3
	1	0	6	6
	2	0	9	9
	3	5	15	20
	4	24	18	42
	5	54	1	55
	6	44	0	44
Total		127	55	182

Table 13 C: Chi-Square Tests: eTBI Score * Outcome

	Value	Df	Asymp. Sig. (2- sided)
Pearson Chi-Square	110.784	8	.000
Likelihood ratio	133.174	8	.000
N of valid cases	182		

Management Distribution

Table 14 A: Frequency distribution of Management Modality

Management	Frequency	Percent
Conservative	144	79.1%
Surgery	38	20.9%

Out of the 182 patients, 79.1% (n=144) were managed conservatively and the rest 20.9% patients (n=38) were managed by surgical intervention. (Table 14-A) Statistical analysis comparing management modality with outcome showed no significant correlation with p-value of 0.163. (Tables 14-B and 14-C)

Table 14 B: Statistical analysis for correlation between management and outcome

		Outcome		Total
		Favourable	Unfavourable	
Management	Conservative	104	40	144
	Surgery	23	15	38
Total		127	55	182

Table 14 C: Chi-Square Tests: Management * Outcome

	Value	Df	Asymp. Sig. (2- sided)
Pearson Chi-Square	1.950	1	.163
Likelihood ratio	1.883	1	.170
N of valid cases	182		

Discussion

The results of our study showed similar results to various other studies found in the literature, being comparable for both geriatric patients and traumatic brain injuries as a whole. We had identified 200 patients aged 65 years and above with traumatic brain injury in a 6-month period from May 2021 to October 2021. As we could not follow-up 18 patients, study was conducted on 182 patients as sample size.

Epidemiological data from around the world reported a greater number of male patients compared to females and this was the case in our study also. We had about 144 (79.1%) males and only 38 (20.9%) females.76.4% of the patients were in the age group 65-74 years which was more than what we could find in the literature. This may be explained by the increased incidence of RTA in this age group in contrast to falls which are more common in very elderly patients aged ≥ 75 years. RTA is the most common mode of injury in Indian literature [13,14] whereas in developed countries falls still remain the common mode of TBI. Indeed, the most common mode of injury in our study was by road traffic accidents (n=100, 54.9%) followed by ground level falls (n=74, 40.7%). We could establish statistically significant correlation between mode of injury and clinical outcome in our study.
Neurological status assessment was done using the Glasgow Coma Score and patients were periodically followed-up. Based on the GCS, patients were categorized into mild (with GCS scores 14-15), moderate (with GCS scores 9-13) and severe (with GCS scores 3-8) grades. Only 12.6% (n=23) of the patients had severe injury, while 37.4% (n=68) had moderate injury, whereas majority 50% (n=91) had mild injury. Even though GCS is the commonly used score for rapid assessment of patient in primary survey, in elderly patients GCS may be impacted by comorbidities that affects cognitive function like dementia, delirium etc. These symptoms are especially unmasked in unfamiliar environment like hospital. But our statistical analysis revealed a significant correlation between admitting GCS and outcome. We also endeavored to find out whether length of hospital stay, length of ICU stay, number of days in mechanical ventilation had any impact on clinical outcome. Our study established significant association between duration of ICU stay and number of ventilator days with unfavorable outcome. But we could not establish a significant correlation with the total duration of hospital stay and clinical outcome. Like Canadian CT head rule, our institution protocol also mandates CT brain for all geriatric patients immediately upon arrival in TAEI (Trauma And Emergency Initiative) ward. As the geriatric patients often have cerebral atrophy, any haemorrhage may progress without a significant change in neurologic exam. Additionally, in patients who are on anticoagulants, even if the initial imaging is normal, there is minimal risk of subsequent haemorrhage. Itshayek et al. 27 reported delayed SDH and ICH among elderly patients on anticoagulants. We recommend further observation and additional imaging in such cases.

The most common CT finding in our study was skull fractures (n=70, 38.5%) followed by cerebral contusions (n=55, 30.2%) and acute SDH (n=53, 29.1%). The association between these variables to the outcome was not studied in our patients. Instead, we used standard prognosticating CT models using Rotterdam and Marshall scores.

In our study, most patients had a Marshall score of 2 (n=86, 47.3%) and 5 (n=35, 19.2%). Only 3 patients had a score of 6 and none of them survived. Most of our patients had a Rotterdam score of 2 (n=75, 41.2%) and 3 (n=52, 28.6%). Numerous other studies in the literature have evaluated these scores and validated their use in TBIs. We could establish a significant correlation and statistical association of both Rotterdam score(p-0.000) and the Marshall score(p-0.000) to the clinical outcome after 3 months. We also found significant association between effacement of basal cisterns and presence of midline shift with unfavorable outcome.

Medical comorbidities are quite common among elderly population and in our study most common comorbidity found was diabetes in 34.1% (n=62) followed by hypertension (n=46, 25.3%) and CAD (n=35, 19.5%). We established a statistically significant correlation between presence of any the above

comorbidity and outcome. Other comorbidities which were present in our study population without statistical significance were CVA, CKD, COPD and malignancies.

In our study, about one-fifth of the patients (n=41, 22.5%) were on either antiplatelets or anticoagulants or both. Pre-injury antiplatelets is associated with three times higher mortality in patients over 50 years.28 Interestingly, Fortuna et al. [29] reported a similar higher mortality for patients with preinjury antiplatelets who received platelet transfusion compared with those who were not transfused. Franko et al. [30] in a retrospective analysis of 1493 patients with TBI reported that INR > 4.0 and age > 70 years were associated with higher mortality. Karni et al. [31] reported that 30day mortality among warfarinized patients with mean INR 3.0 was 50% whereas in non-warfarinized group mortality was 20%. In our study we could establish strong association between antiplatelet/anticoagulant intake (p-value=0.030) and clinical outcome.

The effect of beta blockers on clinical outcome after TBI is controversial. Some retrospective studies suggest that elderly patients on beta blockers have lower mortality after severe TBI. Probable postulated mechanism is that beta blockers may attenuate post traumatic catecholamine induced vasospasm thereby reducing local ischemia.[32] But in patients with severe systemic injuries beta blockers may mask the systemic response to trauma leading to increased mortality.[33] We were able to establish a significant correlation between preinjury beta blocker intake and clinical outcome.

Schneider et al. [34] reported that preinjury statin use in elderly patients with TBI is associated with decreased mortality and good functional recovery at 12 months post injury. This effect is possibly related to the anti-inflammatory and immunomodulatory effects of statins. But in our study, we could not establish a significant correlation between statin intake (p-value=0.206) and clinical outcome.

The overall mortality for geriatric patients aged 65 years and above with TBI reported in literature varies between 14% to 55%.[35,36,37] Despite the fact that elderly patients sustain less severe injuries old age is associated with higher mortality and worse functional outcome at discharge.[38] Ramanathan et al reported that over the past two decades increasing number of elderly individuals are surviving moderate to severe TBI. Advances in neurosurgical care especially neuro intensive care has been attributed to this reducing trend of mortality among geriatric TBI patients. In our study overall mortality is 18.7% (n=34) which is comparable to other outcome studies in this age group.

Ernest et al. [39] in 2019 published a novel scoring system for risk stratification for elderly TBI patients. It includes GCS motor score, presence of comorbid cardiac, pulmonary, renal dysfunction or malignancy and laboratory results of RDW-CV ≥14.5 and platelets < 100,000 cells/dl. eTBI score ranges from -2 to 6. In our study majority of patients had eTBI score of 5 (n=53). All patients with eTBI scores of -2,-1,0,1,2 had unfavorable outcome and all patients with eTBI score 6 had favorable outcome.

Although no external validation of this score has been published, we could establish statistically significant correlation between eTBI scores (p-value=0.000) and clinical outcome at 3 months. As it is a derivative of GCS motor score with added features that proved to be independent outcome predictors it can be used as an objective tool to predict outcome for elderly patients and can help guide treatment.

Though our study had certain limitations like a relatively small sample size and a short follow-up period, we were able to establish a statistically significant association between number of variables like Clinical scores (admission GCS score, eTBI score) mode of injury, CT scores (Marshall, Rotterdam), comorbidities, preinjury antiplatelet/ anticoagulant and beta blocker intake with three months clinical outcome.

Conclusion

Number of geriatric patients with TBI continues to rise every year. Management and care of such patients is a significant issue faced by trauma care providers. The current care of elderly patients with TBI is based on guidelines derived from previous works done primarily on younger adults. The relative paucity of information regarding TBI in geriatric patients made the job of the clinician even more difficult.

Early diagnosis, management, surgical intervention whenever required and proper intensive care of these patients may produce excellent results with favorable outcome possible for 69.8% of patients as revealed by our study. However, the main focus while managing geriatric TBI patients should be to avoid prolonged institutionalization especially prolonged ICU stay and prolonged mechanical ventilation, both of which were found to adversely affect the clinical outcome in our study.

Both Marshall and Rotterdam CT scores were found to be reliable in our study and can be used for outcome prognostication. Lower eTBI scores were associated with unfavorable outcome. The preinjury intake of antiplatelets, anticoagulants and beta blockers play a significant role in elderly TBI patients as they seem to adversely affect clinical outcome in our study. Poor GCS score at admission, absent/effaced basal cisterns and/or presence of midline shift in CT, presence of co-morbidities

like diabetes, hypertension and CAD were all associated with unfavorable outcome in our study. We hope that these and future data may aid families and clinicians in making crucial decisions when dealing with geriatric TBI patients.

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