Effect of Age on Glasgow Coma Scale in Patients with Moderate and Severe Traumatic Brain Injury at a Tertiary Care Centre

ABSTRACT

Background:

Traumatic brain injury (TBI) is a common cause of death and disability globally and is a strain on health services. The Glasgow Coma Scale (GCS) by Teasdale and Jennett from 1974 is the mainstay of neurological assessment in TBI. There has been growing recognition, however, that the presentation of the GCS can be significantly affected by age; older patients will often present with seemingly good GCS, despite having severe injuries. It creates a misconception of the extent of injury, poor triage and poor outcomes. It is therefore important to better understand how age affects GCS scores as well as how recovery curves for early recovery look across ages to optimize management of TBI across all ages.

Methods:

Teerthanker Mahaveer Hospital, Moradabad was a cross-sectional observational study conducted in the duration of six months (from January 2025to June 2025). Patients with moderate to severe TBI (GCS $\leq$ 12) within 24 hours of onset were convenience sampled to a total of 100 patients 15–65 years old. The GCS was measured on admission (Day 1) and on Day 7. Clinical information such as mechanism of injury, associated injuries, cerebral dysfunction subtype and hemodynamics parameters were documented. Data were analyzed descriptively and by employing one way ANOVA (Spss version 25.0).

Results:

The mean age was 43.07 ± 16.87 years. Mean GCS improved from 11.06 ± 2.88 at admission to 13.35 ± 1.97 at Day 7, yielding a mean improvement of 2.29 ± 1.69 points. Motor vehicle accidents were the second most common mechanism of injury (26%) while car accidents were the third most common (21%). No significance differences were seen between mechanism of injury and GCS change ($p = 0.100$), accompanying injuries and GCS change ($p = 0.525$) or cerebral dysfunction type and GCS change ($p = 0.135$).

Conclusion:

Results showed that there was significant neurological recovery in all subgroups of patients over 7 days, underscoring the importance of aggressive TBI care in young and old patients as

well as with different types of injuries, even when there is no age or injury precondition. Age correction may be needed for the standard GCS to best be used clinically. More accurate, age-specific assessment instruments for the nervous system should be developed from larger, multicenter, prospective studies employing age-specific protocols.

Keywords: Traumatic brain injury, Glasgow Coma Scale, age correlation, neurological assessment, mechanism of injury

INTRODUCTION

A traumatic brain injury (TBI) is an injury that affects the brain, causing damage due to trauma. Despite significant progress in the clinical care of traumatic brain injury (TBI) over the past few decades, a major challenge remains: how to ensure that conventional assessment tools are valid for the entire age range of patients.[1] The challenge is becoming increasingly urgent with both high income and middle income countries, the number of elderly people suffering traumatic injuries is dramatically higher than ever before.[2]

Although the Glasgow Coma Scale (GCS) was developed by Teasdale and Jennett in 1974, categories TBI severity into three bands mild (GCS 13–15), moderate (GCS 9–12), and severe (GCS 3–8).[3]

A number of large-scale observational studies have identified that when the same anatomical brain injury occurs, older patients present with higher GCS scores than younger patients, forming a paradox that suggests that clinical assessment grossly underestimates the actual burden of injury in these patients, and may result in inappropriate triage.[4,5] Kehoe et al. systematically analyzed over 20,000 patients, noting that elderly TBI patients with equivalent structural injury severity had higher GCS scores compared with younger patients, which also resulted in an underestimation of injury burden by clinical assessment and potentially inappropriate triage.[6]

There are multiple causes for these age-related differences, in terms of neurobiology. The clinical stability that is seen in older people may be caused by a number of age-related changes: cerebral atrophy, increased adherence of the dura with the skull, atherosclerosis of the cerebrovascular system, and changes in neurotransmitter systems, which can also alter the response of the brain to trauma and make it clinically appear stable.[7] A window of apparent stability may be created because the outward expression of neurological deterioration is delayed by reduced neuroplasticity and impaired compensatory mechanisms in older people.[8] Baseline dementia or prior stroke may make the GCS unreliable in elderly patients, and polypharmacy or other pre-existing cognitive problems may make GCS interpretation difficult.[9] Traditional GCS triage may therefore miss the mark in the identification of risk in older patients with TBI.[10]

Although initial GCS scores tend to be better preserved in elderly patients with TBI, compared with younger patients, elderly patients continue to have higher mortality, functional disability, and poorer outcome following rehabilitation, which has led to development of age-adjusted modifications to standard neurological assessment instruments.[11]Salottolo et al. developed a recalibrated GCS (rGCS), employing age as an explicit parameter and incorporating it into a cohort of over 539,000 patients, the ability to better predict in-hospital mortality (0.800 vs 0.755, $p < 0.001$) and to better predict neurosurgical intervention and unfavorable discharge disposition.[12]

Moreover, other molecular markers, such as ubiquitin carboxy-terminal hydrolase-L1 (UCH-L1), have demonstrated decreased discriminatory value in older adults, suggesting that a combination of clinical scores and biological markers would be optimal for TBI assessment in older patients, and that there is a need for multimodal assessment strategies.[13]

There's a little more complexity to cerebrovascular reactivity research. These results suggest that with increasing age, there is a corresponding decrease in cerebrovascular autoregulation in moderate-to-severe TBI and suggest that age is a biological modifier of cerebrovascular physiology which may have implications for individualized monitoring and management.[14]

Machine-learning methods are also emerging as useful tools for prediction of outcomes in elderly TBI. Si et al. built a CatBoost model based on 24-hour data from the ICUs of 40 hospitals, which had an AUROC of 0.867 when applied to predicting 30-day mortality, with GCS, oxygen saturation, and prothrombin time being key predictors.[15,16] A parallel meta-analysis of 33 cohort studies that included 71,718 patients with severe TBI resulted in an overall mortality rate of 27.8%, with anaemia, diabetes, coagulopathy, and haemodynamic instability all being risk factors, highlighting the complexity of TBI management.[17]

Elderly TBI is a significant problem and is increasing worldwide. The high mortality rates of over 38% in developing countries.[18] and the chronic neuropsychiatric sequelae that often follow TBI that negatively affect functional independence, accentuate the burden.

TBI associated with road traffic accidents and falls in India is a significant public health problem, but there are few epidemiological and clinical studies that have been conducted by tertiary care centers in the country, especially ones that have been age-stratified which may not be directly applicable to the Indian healthcare environment because of variation in injury patterns, access to health care and patient demographics.[19,20] Most of the studies available were conducted in high income countries.[21]

Moreover, there are conflicting recommendations about the best age range for using modified neurological assessment protocols, with published data suggesting age cut-off points ranging from 55 to 70 years[22], and little detailed clinical description of the individual patient trajectories.[23]

In the light of this background, the present study was designed to explore the correlation between patient's age with the GCS score in patients with moderate and severe TBI and specific aims were set to characterize the mechanism of injury and to assess the acute neurological recovery and to find the clinical pattern which could guide age-specific assessment strategies.[24]

MATERIALS AND METHODS

Study Design and Setting

A cross sectional, single-center study was conducted in a tertiary care hospital of dedicated trauma, neurosurgery and intensive care unit at Teerthanker Mahaveer Hospital, Moradabad. The study was conducted in a prospective manner for 6 months (January2025 to June 2025).

Participants

A total of 100 patients between the ages of 15 and 80 years were enrolled by convenient sampling, with a diagnosis confirmed by neuroimaging (CT or MRI) that required within 24 hours after sustaining moderate to severe TBI (initial GCS $\leq$ 12). Patients with a previous neurological disorder, altered sensorium (into intoxication or sedation), incomplete medical records, and who not give informed consent were excluded.[25]

Outcome Measures

The main outcome was the GCS score changes between Day 1 (admission) and Day 7. The injury mechanism distribution and the association of accompanying injuries with cerebral dysfunction subtype and GCS trajectory were secondary outcomes.

Statistical Analysis

SPSS version 25.0 was used for data analysis. Continuous variables are presented as mean and SD and the categorical as frequencies and percentages. One-way analysis of variance was used to compare the changes in GCS between groups and a p value of less than 0.05 was considered statistically significant. All studies were done under the Declaration of Helsinki; informed consent was obtained from all participants or their legal guardians.[26]

RESULTS

Baseline Characteristics

A total of 100 patients with moderate to severe TBI were enrolled. The population was quite heterogenous with a mean age of 43.07 ± 16.87 years (range 18–78 years). Hemodynamic parameters at admission were mostly within acceptable limits: mean systolic blood pressure 124.1 ± 14.86 mmHg, mean diastolic blood pressure 87.02 ± 8.69 mmHg, mean heart rate 77.03 ± 9.54 beats per minute, mean respiratory rate 17.51 ± 2.36 breaths per minute, mean peripheral oxygen saturation 93.8 ± 5.35%. These values suggest that most of the patients were stable when they arrived and had severe neurological injuries.

Trajectory of Glasgow Coma Scale

The mean GCS at admission (Day 1) was 11.06±2.88, with a range from 3 to 16, making the overall group a moderate TBI group. By Day 7, mean GCS had improved to 13.35 ± 1.97 (range 7–15). Mean change in GCS was 2.29 ± 1.69 points (range −3 to +10) which is clinically significant, suggesting significant potential for early recovery of neurologic function among patients with moderate to severe TBI treated at this tertiary care center.

Mechanism of Injury

The most common mechanism of injury falls (36 cases); this was the greatest number. Falls represented the largest number of injuries (36 cases, 36%). 26% of motor vehicle accidents were two-wheelers or pedestrians, 21% were car accidents and 17% of accidents were other mechanisms. The overall road traffic-related injuries burden was high with all vehicular accidents accounting for 47% of the total injuries.

The mean GCS change between the four categories of injury mechanism did not significantly differ (ANOVA 1-way: F=2.145, p=0.100). The patients who fell showed the highest mean GCS score change (2.75 ± 1.92 points), and the patients in the "other mechanisms" group showed the lowest GCS score change (1.53 ± 1.55 points). Intermediate improvements were seen for patients with motor vehicle and car accidents, with scores increasing by 2.23 ± 1.45 and 2.19 ± 1.50 points, respectively. The trends were not statistically significant, but they may have clinical significance and should be investigated in larger studies.

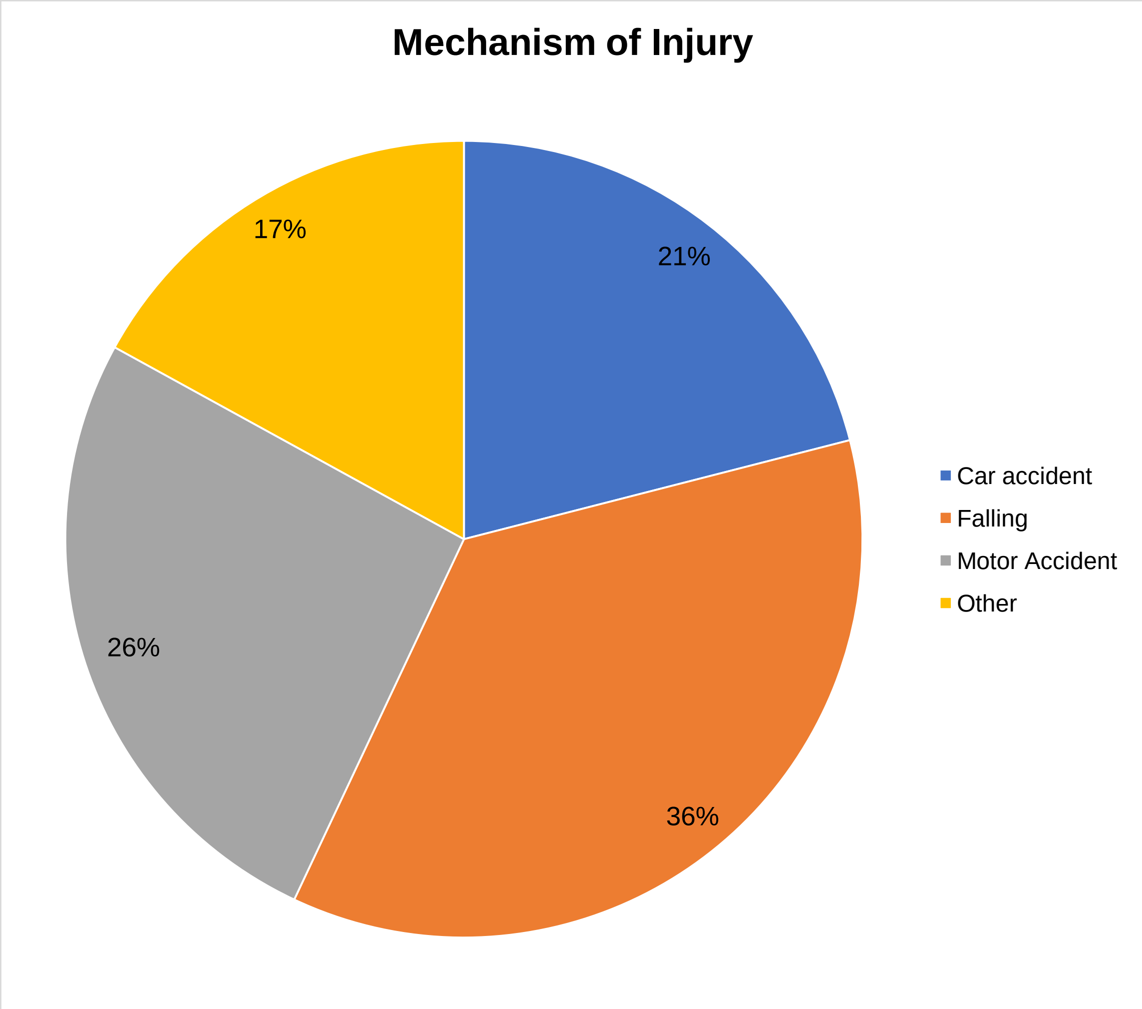

Figure 1: Percentage of cases with each Mechanism of Injury

Accompanying Injuries

In terms of any accompanying injuries, 34 patients (34%) were also injured in other unspecified ways, 27 patients (27%) had a fracture of the lower or upper limbs, 27 patients (27%) sustained rib fractures, and 11 patients (11%) had only TBI. A one-way ANOVA revealed that there was no statistically significant association between accompanying injury type and GCS change (F value – 0.800; p value – 0.525). Patients with rib fractures had the largest mean change (+2.48 ± 1.48 points) and patients with no accompanying injuries had the smallest mean change (+1.55 ± 1.69 points). These differences in this sample were not statistically significant.

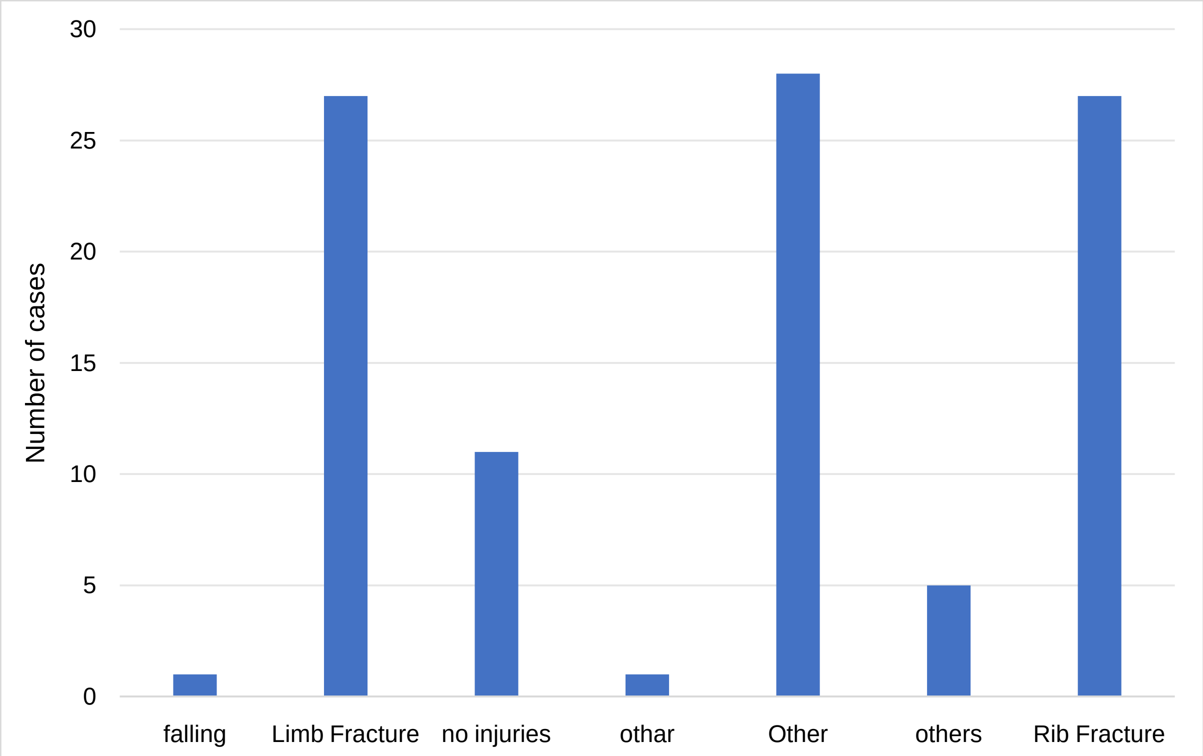

Figure 2: -Number of cases for each accompanying injury

Cerebral Dysfunction Subtypes

The other category of cerebral dysfunction was the most prevalent (43%), followed by brain contusion (17%), intracranial hemorrhage (14%), subdural hematoma (13%), a second intracranial hemorrhage subgroup (7%) and diffuse axonal injury (6%). The One-way ANOVA showed an F-value of 1.733 and a p-value of 0.135, suggesting that there were no statistically significant differences in change in GCS by subtype. Intracranial hemorrhage (3.14 ± 1.21 points) and subdural hematoma (3.08 ± 1.32 points) had the greatest mean change scores whereas the mean change scores for the "other" group and the second intracranial hemorrhage group were 1.91 ± 1.54 points and 1.86 ± 2.68 points, respectively. These numerical differences are not significant in this group of patients and could be due to underlying pathophysiological differences.

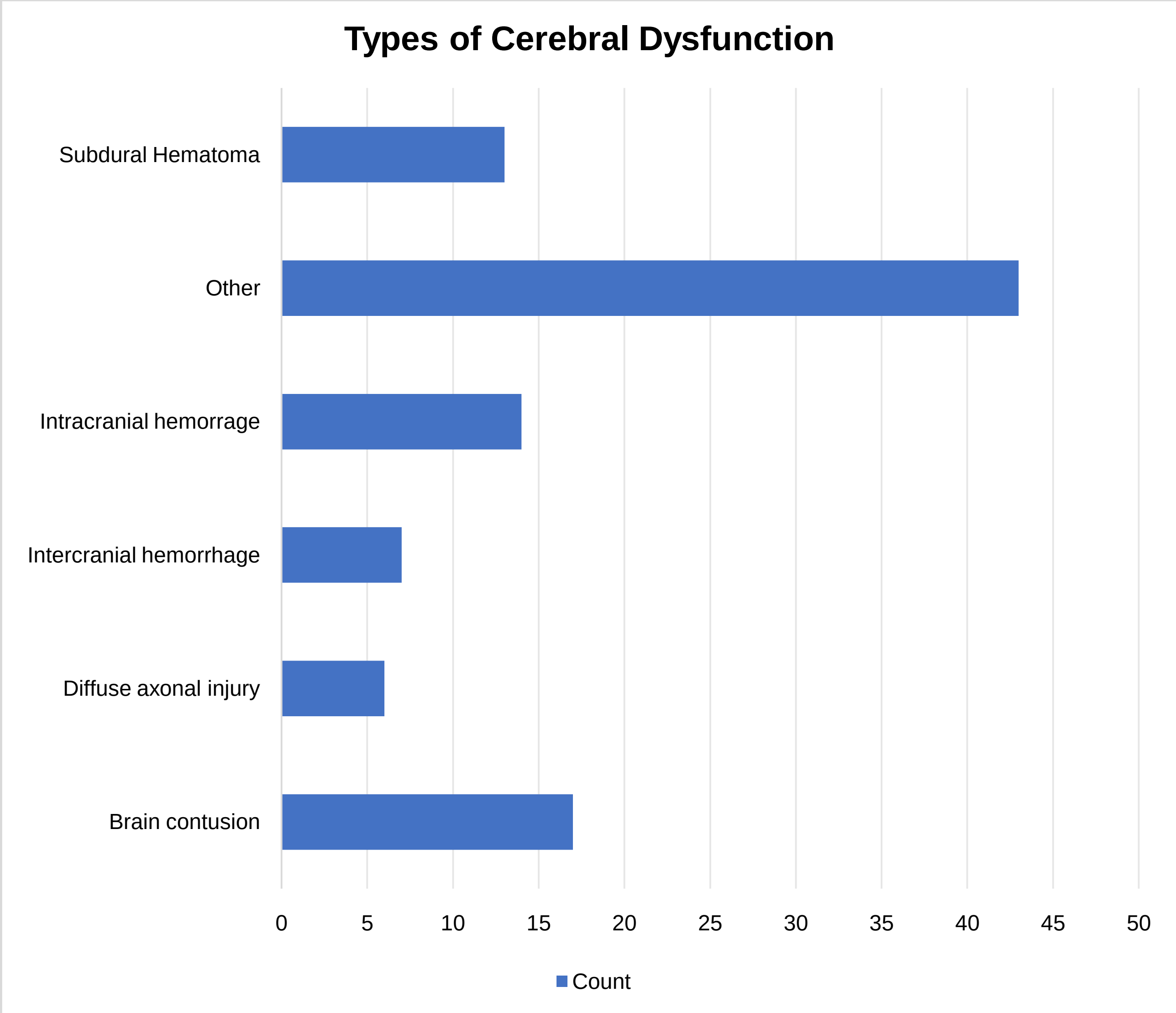

Figure 3: Number of cases with each type of Cerebral Dysfunction

DISCUSSION

The clinical profile of 100 patients with moderate to severe TBI was studied in a tertiary care center and northern India in a wide age spectrum. The overall trend of neurological improvement seen (mean GCS increasing from 11.06 to 13.35 at 7 days) is like other acute TBI cohorts and highlights the critical importance of early and sustained clinical intervention.[27]

The mean age of 43.07 years is also consistent with the working-age population that is most vulnerable to traumatic injuries in low- and middle-income countries (LMICs), which are usually caused by fall-related or road traffic-related incidents, with the latter accounting for 47% of injuries and the former accounting for 36%.[28] This highlights the dual public health challenge of protecting working-age people against injuries and ensuring safe enforcement of traffic regulations.[29]

This does not indicate a lack of statistical power as it is possible that with a larger sample size, statistically significant associations between these variables and the GCS change would be present. However, the lack of statistically significant associations between age, injury mechanism, accompanying injuries, cerebral dysfunction subtype and GCS change may be due to lack of power with a small sample size of 100 patients. In larger studies, age has consistently been shown to be an independent predictor of both GCS presentation and outcome in TBI.[5] the landmark study by Kehoe et al. showed that elderly patients present with paradoxically high GCS scores because of brain atrophy, which helps to increase intracranial compliance and to buffer against hematoma pressure effects on consciousness.[6]

The higher number of patients in the fall group showing an improvement in GCS may be a result of the different pattern of injuries between the fall group and the vehicular accident group. This is because low-energy falls tend to cause more localized injuries to the brain, such as cortical or extradural injuries, which may be more likely to be amenable to neurosurgical decompression, while high-velocity vehicular collisions are more likely to cause diffuse axonal injury, which is associated with more persistent neurological deficits.[15]

The unexpected finding that subdural hematoma and intracranial hemorrhage had numerically higher GCS improvement is perhaps a reflection of the often-dire prognosis given to these lesions. In the present results, however, this can be explained by successful neurosurgical evacuation restoring cerebral perfusion and thus enabling early recovery; this has been shown to have significantly better short-term GCS trajectories even in older patients.[24]

Consideration should be given to the use of a standardized TBI triage for older adults, given the increasing body of literature surrounding age adjusted assessment tools. Age adjusted scoring systems have also been found to be better at predicting in-hospital mortality in subgroups of patients, such as older children and those with more serious head injuries.[22]

New data on cerebrovascular reactivity also supports the age-stratified approach to TBI management. In older TBI patients, impaired pressure autoregulation is a risk factor for secondary brain injury, and the use of cerebral perfusion pressure (CPP) targets, in addition to the clinical scoring system, is a key clinical consideration to address in the future.[15]

There are several implications for practice from the results of the present study. Reliable neurological improvement by all injury subgroups supports early aggressive management of moderate to severe TBI regardless of age, injury etiology or cerebral dysfunction subtype.

The lack of strong single factor predictors further underscores the importance of a multi-modal clinical assessment rather than any one factor.[30]

Weights of this study are a convenience sample of 100 patients from a single center, resulting in limited statistical power and generalizability, the short follow-up duration of seven days, which only covers the acute recovery phase, the lack of a systematic inter-observer variability control in the evaluation of the GCS, and incomplete documentation of pre-injury medication, comorbidities, and exact surgical timing. Larger multicenter designs with longer follow-up and standardized protocols should be studied to overcome these limitations in future research.[17]

CONCLUSION

The present cross-sectional study of 100 patients with moderate to severe TBI in a tertiary center of northern India showed that there was clinically significant early neurological recovery with a mean GCS improvement of 2.29 ± 1.69 points in 7 days. Falls and road traffic accidents were mostly injuries, as is typically seen in injury epidemiology across the region, and there is a need for specific public health interventions in both areas.

While no statistically significant differences were found between patient age, mechanism of injury, type of accompanying injury, or cerebral dysfunction subtype in terms of the magnitude of GCS change, all clinical subgroups trend toward neurological recovery, favoring the concept of early and aggressive TBI management regardless of demographic or injury-related factors. The data supports the importance of each clinical variable predicting the clinical course independently andemphasizes the need for a comprehensive clinical evaluation in the acute stage of TBI.

The findings underscore a relevant gap in clinical practice today: the importance of the GCS, which may need age-specific supplementation to enhance its predictive value and usefulness, especially when aging populations around the world are resulting in an increase in the number of elderly TBIs. More precisely, age-specific standards of neurological assessment for the entire lifespan of patients with TBI will require future multicenter prospective studies with large, representative enrollment, longer follow-up period and systematic use of molecular biomarkers, cerebrovascular reactivity monitoring, and validated age-adjusted scoring protocols.

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