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Original Research Article

Correlation of Random Blood Sugar and Glasgow Coma Scale in Traumatic Brain Injury

S.K. Fathimunnisa[1], Gangadharabhatla Padmini[2], D.N.V. Sameeraja[3], Gunturi Tej Kiran[4]

[1]Assistant Professor, Department of General Surgery, GMC, Rajamundry
[2]Assistant Professor, Department of General Surgery, GMC, Rajamundry
[3]Assistant Professor, Department of Surgical Oncology, GMC, Rajamundry
[4]Resident in General Surgery, GSL Rajamundry

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Corresponding author: Dr. Gunturi Tej kiran
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Abstract

Background: Traumatic brain injury (TBI) is a major cause of mortality and long-term disability worldwide. Secondary brain injury following TBI significantly influences prognosis, and hyperglycemia has been recognized as an important contributing factor. Elevated blood glucose levels may aggravate neuronal damage through metabolic and ischemic mechanisms. The relationship between random blood sugar (RBS) and neurological status assessed by Glasgow Coma Scale (GCS) may provide valuable prognostic information.

Aim: To determine the correlation between random blood sugar levels and Glasgow Coma Scale scores in patients with traumatic brain injury.

Materials and Methods: This prospective observational study was conducted on 90 patients with TBI admitted to the Department of Neurosurgery over 18 months in a Tertiary Care Hospital. Adult patients (>18 years) with mechanical brain injury and GCS scores between 3–15 were included. Patients with diabetes, metabolic disorders, malignancies, and other causes of brain injury were excluded. RBS levels and GCS scores were recorded at admission, 24 hours, 72 hours, and discharge. Statistical analysis was performed using SPSS version 26 with Pearson correlation, chi-square test, ANOVA, and logistic regression analysis. A p-value <0.05 was considered statistically significant.

Results: The majority of patients were males (75.6%) with the highest incidence in the 21–40 years age group (41.1%). Road traffic accidents accounted for 67.8% of injuries. Mean admission RBS was 192.14±58.65 mg/dL and progressively decreased to 131.84±28.22 mg/dL at discharge. Severe TBI patients had higher admission glucose levels (261.33±25.03 mg/dL) compared with moderate and mild injuries. A significant inverse correlation was observed between admission RBS and GCS ($r = -0.843$, $p<0.0001$).

Discussion: Hyperglycemia was associated with increased TBI severity and poorer neurological outcomes. Higher blood glucose levels corresponded with lower GCS scores and increased mortality risk.

Conclusion: Early hyperglycemia showed a strong negative correlation with GCS and may serve as an important predictor of TBI severity and prognosis. Early monitoring and management of elevated glucose levels could improve clinical outcomes.

Keywords: Traumatic Brain Injury, Random Blood Sugar, Glasgow Coma Scale, Hyperglycemia, Prognosis.

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Introduction

Head injury (HI) refers to physical damage to the skull, scalp, or brain, while traumatic brain injury (TBI) is defined by a disruption in the brain's normal functioning caused by an external force. TBI is one of the leading causes of death & disability worldwide, affecting people of nearly all ages, with fatality rates reaching as high as 40% in severe cases and dropping below 1% in mild cases. Annually, around sixty- nine million people globally suffer from TBI due to various factors, with the highest rates found in Southeast Asia (56%) and the Western Pacific regions (56%). Main cause of head trauma leads TBI is road traffic accidents (RTA), which accounts 60% of cases, followed by falls (20-30%), assaults (10%), & incidents related to sports & occupational activities (10%). [1,2] TBI significantly contributes to emergency hospital admissions and presents substantial socioeconomic & public health challenges. The initial brain injury that occurs

during the traumatic event is known as primary brain injury, which includes conditions such as intracranial hemorrhages, skull fractures, diffuse axonal injury (DAI), & contusions or coup-contrecoup injuries. Intracranial hemorrhages can present as epidural hematoma (EDH), subdural hematoma (SDH), subarachnoid hemorrhage (SAH), intraventricular hemorrhage (IVH), & intracerebral hematoma. [3,4]

Secondary brain injury includes various conditions such as hypoxia, hypotension, ischemia, hydrocephalus, cerebral edema, vasospasm, and second impact syndrome. These issues stem from physiological & metabolic changes that occur after the initial injury and can last for hours to days, potentially worsening the primary brain injury.

As a result, a key goal in treating patients with severe TBI is to reduce the risk of secondary brain injury. Patients with TBI often exhibit symptoms like headaches, vomiting, loss of consciousness, skull fractures with scalp injuries, & hematomas. The Glasgow Coma Scale (GCS) is an essential tool for assessing the neurological status of individuals who have suffered head trauma, helping to classify the severity of TBI. The GCS consists of three components—eye, motor, and verbal responses—resulting in a score that ranges from 3 to 15. Scores of 13 - 15 indicate mild injury, scores of 9 to 12 suggest moderate injury, and scores of 3 - 8 reflect severe injury. According to the modified ATLS classification, a GCS score of 13 is categorized as mild TBI. [5,6] The occurrence of secondary brain injury has a significant impact on the prognosis of individuals with traumatic brain injury (TBI), with hyperglycemia recognized as a major contributor to this secondary damage. Acute hyperglycemia, defined as blood glucose levels exceeding 200 mg/dL shortly after the injury, is commonly seen in patients with severe TBI. There is widespread agreement in the medical community about the strong link between acute hyperglycemia & negative outcomes in TBI patients, prompting recommendations for the immediate implementation of measures to control acute hyperglycemia following the injury.

Consequently, managing blood glucose levels in these patients has become a critical clinical issue due to its direct effect on prognosis. [7,8] It has been suggested that hyperglycemia may be a temporary condition, especially in cases of severe head trauma, and associated with a poorer neurological prognosis.

The exact mechanisms that lead to unfavorable neurological outcomes are not fully understood. However, many studies indicate that elevated glucose levels may worsen brain injury through mechanisms such as increased anaerobic glycolysis and the resulting lactic acidosis during ischemic events. Currently, there is no defined threshold for Random Blood sugar that is linked to negative outcomes following acute TBI, highlighting the absence of standardized glucose management targets for hyperglycemia in TBI patients. Nonetheless, most research has identified 11.1 mmol/l as the critical cutoff for diagnosing hyperglycemia. [9,10] Hyperglycemia, which is marked by both high peak glucose levels and persistently elevated glucose concentrations, often arises as a secondary complication after severe TBI and is associated with adverse clinical outcomes. The factors contributing to hyperglycemia following TBI include a range of elements such as the body's stress response, inflammatory processes, pre-existing diabetes mellitus, dysfunction of the pituitary and/or hypothalamus, as well as the effects of surgical interventions and anesthesia. The immediate effects of hyperglycemia can include lactic acidosis, electrolyte imbalances, inflammatory responses, vascular complications, disruption of the blood-brain barrier (BBB), and increased permeability. [11] Modern therapeutic strategies aimed at addressing secondary brain injury linked to hyperglycemia include nutritional interventions, glucose management, induced hypothermia, naloxone administration, and the use of mannitol. While considerable progress has been made in understanding the relationship between hyperglycemia & severe TBI in recent years, there are still many treatment challenges & gaps in knowledge. This study seeks to evaluate the relationship between the severity of TBI, initial Random Blood Glucose levels at admission, and the subsequent outcomes in patients with TBI.

Aim: To find correlation between random blood sugar levels and Glasgow coma scale in traumatic brain injury patients

Objectives: To estimate the random blood sugar levels in traumatic brain injury patients and to assess the level of consciousness by taking Glasgow coma scale.

Material & Methods

This was a Prospective observational study done in 90 Patients admitted for traumatic brain injuries in department of neurosurgery for 18 Months in a Teriary Care Hospital.

Inclusion Criteria: Patients above 18yrs with brain injury caused due to mechanical force only and with GCS between 3 & 15.

Exclusion Criteria: Brain Injury Due to other Causes like Ischemia, Hypertension, & Infection; Associated with other Soft Tissue/Musculoskeletal Injury Liver Disorders, Renal Diseases, Hyperuricemias, & Malignancies; Other brain disorders which alter blood sugar levels and Diabetics.

Data: Blood samples were taken upon patient admission after 24 hrs, 72 hrs, and during discharge to assess blood glucose levels. The Glasgow Coma Score (GCS) was recorded at the time of admission, after 24 hrs, 72 hrs and during discharge. Blood glucose concentrations were measured using the glucose oxidase-peroxidase method with reagent kits in an autoanalyzer. Patients with TBI were selected from the neuro intensive care unit and post- traumatic head injury wards in the Department of Neurosurgery, following specific inclusion & exclusion criteria. Both patients & their caregivers were informed about the study, & informed consent obtained. Data were collected on demographic variables such as age, sex, mechanism of injury, and clinical evaluations related to GCS, pupil response, and findings or diagnoses from computed tomography (CT) scans. Laboratory tests, including random blood sugar and HbA1c levels, were performed. Additionally, any changes in GCS, whether improvements or declines, were recorded. The severity of head injury was categorized using the Glasgow Coma Scale (GCS) into three levels: mild (GCS 13-15), moderate (GCS 9-12), & severe (GCS <9). The effectiveness of treatment was assessed using the Glasgow Outcome Score at the time of patient discharge.

Statistical Methods: Categorical data were expressed as percentages, while continuous variables were presented as mean ± standard deviation (SD). Relevant statistical analyses were performed as needed, including independent t-tests, Pearson correlation coefficients, Spearman rank order correlation tests, and logistic regression analyses. A one-way analysis of variance (ANOVA) was utilized to evaluate the statistical significance of the relationships between admission random blood glucose (RBG) levels & the severity of head injuries, & between admission random blood glucose levels & patient outcomes. A significance threshold of $P < 0.05$ was set. Descriptive & inferential analyses were performed using SPSS version 26 software.

Results:

Table 1: Age Group and Sex Distribution

Age Category	Frequency	Percent
<20	5	5.6%
21-40	37	41.1%
41-60	33	36.7%
>61	15	16.7%
Total	90	100.0%
Female	22	24.4%
Male	68	75.6%

Table 2: Grading Of Severity of TBI Group

Time interval	GCS			
	13-15 score	9-12 score	<9 score	p-value
	Mild	Moderate	Severe	Chi- Square=65.872; p<0.0001
Admission	47 (52.23%)	31 (34.45%)	12 (13.33%)	
After 24hrs	55 (61.12%)	28 (31.12%)	7 (7.78%)	
@72 Hrs	75 (83.33%)	9 (10%)	6 (6.67%)	
Discharge	84 (93.34%)	1 (1.11%)	5 (5.56%)	

Table 3: Causes of Trauma

Mode of Injury	Frequency	Percent
Assault	5	5.6%
Fall	24	26.7%
RTA	61	67.8%
Total	90	100.0%

Table 4: Ct Scan Findings

Ct Scan Findings	Frequency	Percent
Contusion	16	17.8%
Diffuse axonal injury	1	1.1%
Extra dural hemorrhage	19	21.1%
Intracerebral hemorrhage	7	7.8%
Subarachnoid hemorrhage	9	10.0%
Subdural hemorrhage	38	42.2%
Total	90	100.0%

Table 5: Management of TBI Cases

Management	Frequency	Percent
Conservative	**59**	**65.6**
Surgery	**31**	**34.4**
Dead	**4**	**4.4%**
Live	**86**	**95.6%**

Table 6: Glucose Levels At Various Time Intervals

Time interval	Glucose Level				No. of Patients
	≤ 150	151-200	201- 300	>300	
Admission	28	31	30	1	**90**
After 24hrs	54	35	2	0	**90**
After 72 Hrs	75	13	2	0	**90**
Discharge	82	5	3	0	**90**

Table 7: Association between GCS and Time Intervals

Time interval	GCS			
	13-15 score (mild)	9-12 score (moderate)	<9 score (severe)	Total
Admission	47	31	12	**90**
After 24 Hrs	55	28	7	**90**
After 72 Hrs	75	9	6	**90**
Discharge	84	1	5	**90**
Chi-Square=65.872; p<0.0001				

Table 8: Admission Blood Glucose Level & Age

Age	Glucose Level				P value
	≤150	151- 200	201- 300	Above 300	
Below 20	2	3	0	0	Chi- Square=12.396, p=0.036
21-30	6	5	8	0	
31-40	6	5	5	1	
41-50	5	7	4	0	
Above 50	**9**	**11**	**12**	**0**	

Table 9: Admission GCS and AGE

Age (yr)	Glucose Level			P value
	13-15 score (mild)	9-12 score (moderate)	<9 score (severe)	
Below 20	3	2	0	Chi- Square=8.183, p=0.017
21-30	8	9	3	
31-40	10	5	2	
41-50	11	3	2	
Above 50	**15**	**12**	**6**	

Table 10: Mean GCS at Various Time Intervals

GCS score	At admission	After 24 hr	After 72 hr	At discharge
13-15 score(mild)	14.12766	14.06383	14.510638	14.74
9-12 score(moderate)	10.32258	11.87097	13.741935	14.39
<9 score (severe)	6.75	9.75	11.083333	11.83

Table 11: Association between RBS & severity of TBI at different time intervals

	GCS score	Mean	Std. Deviation
RBS Admission	13-15 score(mild)	148	20.526
	9-12 score(moderate)	232.2903	51.9141
	<9 score (severe)	261.333	25.0357
RBS after 24 Hrs	13-15 score(mild)	139.82	25.732
	9-12 score(moderate)	155.064	25.463
	<9 score (severe)	156.833	24.364
RBS after 72 HRS	13-15 score(mild)	132.425	22.29422
	9-12 score(moderate)	129.7419	29.8841
	<9 score (severe)	158	34.4884
RBS at discharge	13-15 score(mild)	130.489	20.6585
	9-12 score(moderate)	126.5806	26.3415
	<9 score (severe)	150.75	47.857

Table 12: Descriptive statistical analysis of age, RBS & GCS levels

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	90	18.0	80.0	43.489	16.7515
ADMISSION RBS	90	101.0	330.0	192.144	58.6484
AFTER 24HRS RBS	90	92.0	250.0	147.344	26.3985
AFTER 72 HRS RBS	90	0.0	220.0	134.911	28.0960
DISCHARGE RBS	90	0.0	262.0	131.844	28.2198
ADMISSION GCS	90	3.0	15.0	11.833	2.8961
AFTER 24HRS GCS	90	3.0	15.0	12.7333	2.767
AFTER 72HRS GCS	90	0.0	15.0	13.789	2.7783
DISCHARGE GCS	90	0.0	15.0	14.233	2.9795
AGE CAT	90	1.00	4.00	2.6444	.82532
Valid N (list wise)	90				

Table 13: Correlation of GCS and RBS at various time intervals

	Admission GCS	After 24Hrs GCS	@72 Hrs GCS	Discharge GCS
Admission RBS	-0.843**	-0.491**	-0.229*	-0.141
	0.000	0.000	0.030	0.185
	90	90	90	90
AFTER 24HRS RBS	-0.338**	-0.244*	-0.132	-0.198
	0.001	0.020	0.216	0.062
	90	90	90	90
@72HRS RBS	-0.278**	-0.071	-0.022	-0.092
	0.008	0.504	0.835	0.389
	90	90	90	90
Discharge RBS	-0.277**	-0.139	-0.228*	-0.317**
	0.008	0.191	0.031	0.002
	90	90	90	90

Discussion

Traumatic Brain Injury (TBI) poses a significant socio-economic and public health challenge in today's society. The assessment of outcomes related to TBI is a relatively under-researched area, with only a few studies focused on creating scoring systems and evaluating their prognostic significance.

Globally, road traffic accidents are a major cause of TBI-related deaths, accounting for about 6% of such cases in India, despite the country having only 1% of the world's vehicles. This problem is particularly severe among young adults, many of whom endure lifelong disabilities as a result. The impact of TBI varies widely among survivors, largely influenced by their age. Falls from heights are the leading cause of TBI in children under four and in seniors over seventy. In contrast, vehicle and traffic-related injuries are more prevalent among younger individuals and those up to 50 YRS old, largely due to the necessity of daily travel for work, education, and commerce. In the us, it is estimated that around 5.3 million people live with disabilities resulting from TBI. This condition is a major contributor to mortality and severe morbidity, especially among young adults under thirty-five. In India, the National Health Portal indicates that 1.5

to 2 million people suffer injuries from TBI each year.

Demographic profile: In the present study, the mean age of the study population was 43.48±16.75 years. 5.6% belong to < 20 yrs, 41.1% belong to 20 - 40 years age group, 36.7% belong to 41- 60 years age group, & 16.7% belong to >60 years age group respectively. 75.6% were male & 24.4% were Female. Our research found that the demographic most affected is the 20 to 30-yr age group, which makes up 54.4% of the total cases. Palanisamy S et al identified the 21 + 30-year age range as the most impacted, accounting for 30% of their results. Palanisamy S et al conducted a study with a sample of 60 individuals diagnosed with TBI, where 60% were male & 40% were female.

Mode of Injury: Our research identified traffic accidents as the leading cause of injury, impacting 61 patients (67.8%). This was followed by falls, which affected 24 patients (26.7%). Furthermore, 5 patients (5.6%) sustained injuries due to assaults. In Arfat et al Road Traffic Accidents (RTA) 65.0%, & Fall from Height (FFH) 20.0%, being hit by Assault/hard object 10.0% & Gun shot 2.5%. In Ajay Yadav et al [12] road traffic accidents were most common mode of injuries seen in 57.6% followed by fall injury, physical assault

In another investigation by Vikul Kumar et al., [13] road traffic accidents (RTA) were identified as the primary cause of traumatic brain injury (TBI) in 70.8% of cases, while falls contributed to 22.6% & assaults accounted for 6.6%. Ahmad et al. [14] noted that altered sensorium was the most commonly observed clinical symptom, occurring in 66.7% & 54.8% of cases, respectively, followed by vomiting, which was reported in 20.1%, 46.3%, & 49.5% of cases.

Clinical presentation: 44.4% of TBI cases had altered sensorium, 86.7% had external injury, 48.9% had vomiting, 18.9% had shock, 28.9% had ear discharge, & 5.6% had Seizures. In Arfat et al [15] 58.75% of TBI cases had altered sensorium, 12.5% had external injury, 8.45% had vomiting. In Ajay Yadav et al [12] 35.7% of TBI cases had altered sensorium, 25% had external injury, 15.2% had vomiting, 6.7% had shock, 5.4% had ear discharge, & 2.2% had Seizures.

CT Findings: Based on CT brain scan findings, majority cases had Subdural hemorrhage in 38 (42.2%), followed by Extradural hemorrhage in 19 (21.1%), Contusion in 16(17.8%),

Subarachnoid hemorrhage in 9 (10%), Intracerebral hemorrhage in 7 (7.8%) cases, & Diffuse axonal injury in one case (1.1%). In Arfat et al [15] Subdural hemorrhage in 13.75%, followed by extradural hemorrhage in 12.5%, Contusion in 25%, subarachnoid hemorrhage in 13.75%. In Ajay Yadav et al [12] Subdural hemorrhage in 41.5%, followed by extradural hemorrhage in 32.1%, Contusion in 43.4%, subarachnoid hemorrhage in 35.8%, & Diffuse axonal injury in 14.1%.

Severity of TBI: In our study, we assessed the severity of traumatic brain injury (TBI) using the Glasgow Coma Scale (GCS), classifying injuries into three categories: mild (GCS scores of 6-8), moderate (9-12), & severe (13-15). The study results of GCS@ admission indicate that mild TBI accounted for the largest cases with 47 individuals (52.23%), followed by moderate TBI with 31 cases (34.45%), & severe TBI with 12 cases (13.33%).

TBI severity & Age: A research study conducted by Vikul Kumar et al found that a notable percentage of patients, specifically 118 individuals (54.6%), suffered from severe traumatic brain injury (TBI). The study included a total of 154 male participants & 62 female participants, resulting in a male-to-female ratio of 2.5:1. The average Glasgow Coma Scale (GCS) scores recorded were 14.39 ± 0.49 for mild TBI, 10.16 ± 1.33 for moderate TBI, & 6.01 ± 1.07 for severe TBI cases. In our study, the mean age in the present study was 43.48±16.75yrs in TBI cases.

Hyperglycemia & TBI: In our research, we found that the mean glucose levels for patients with mild traumatic brain injury (TBI) at the time of admission were 192.144±58.648 mg/dl, which declined to 147.34±26.39 mg/dl after 24 hours, 134.911±28.0960 mg/dl after 72 hours & finally reached upto 131.84±28.219 mg/dl during discharge.

Severity of TBI & Glucose levels: For those with severe TBI, the mean glucose levels were 261.33± 25.03mg/dl at admission, decreased to 156.83±24.36 at 24 hours, 158±34.48mg/dl at 72 hours, & 150.75±47.85mg/dl at discharge. In cases of moderate TBI, the initial glucose level at admission was recorded at 232.2±51.91mg/dl, which further decreased to 155.06±25.46 mg/dl after 24-hour, 129.74±29.8 mg/dl after 72-hour, & decreased upto 126.58±26.34 mg/dl at discharge. Whereas in cases of mild TBI, the initial glucose level at admission was recorded at 148±20.52mg/dl, which further decreased to 139.82±25.732 mg/dl after 24-hour, 132.425±22.29mg/dl after 72-hour, & decreased upto 130.48±20.6 mg/dl at discharge.

In our study, the results indicated that the mean glucose level for non-survivors was significantly higher than that of survivors (211.25± 57.789 mg/dL compared to 191.258 ± 58.86 mg/dL). We found a 43.34% prevalence of hyperglycemia among TBI patients at admission, whereas 3.33% of hyperglycemia recorded at discharge. These cases diagnosed with hyperglycemia, with blood

glucose levels exceeding 180 mg/dL, which associated with lower survival rates.

In cases of traumatic brain injury (TBI), the mean blood glucose level of 211.25 mg/dL upon admission was associated with an odds ratio (OR) of 1.2 for overall mortality, with a confidence interval ranging from 1.01 to 1.36 ($P < 0.0001$).

A retrospective study by Jeremitsky et al. analyzed 77 patients admitted to the intensive care unit (ICU) due to severe TBI, tracking serum glucose levels over five days. The results showed that the average glucose level in non-survivors was significantly higher at 187.0 mg/dL compared to 153 mg/dL in survivors. The study also found a relationship between hyperglycemia, defined as glucose levels of ≥170 mg/dL, & lower survival rates, particularly in patients who had two or more cases of elevated glucose levels. [16]

Increased blood glucose levels worsen existing brain injuries, leading to tissue acidosis, oxidative stress, & a state of cellular immunosuppression, which ultimately contributes to multiorgan failure in critically ill patients. Liu-DeRyke et al. conducted a retrospective study involving 380 ICU patients with TBI, finding that glucose levels at or above 160 mg/dL within the first 24 hours of hospital admission were associated with negative outcomes, including higher mortality rates and increased injury severity. [17]

GCS score: The mean admission GCS in patients with mild TBI was 14.12766, in moderate TBI 10.32258 & 6.75 in severe cases of TBI. Whereas GCS at 24 hours in patients with mild TBI was 14.06383, in moderate TBI 11.87097 & 9.75 in severe cases of TBI. Whereas GCS at 72 hours in patients with mild TBI was 14.51, in moderate TBI 13.741935 & 11.083333 in severe cases of TBI. Whereas discharge GCS in patients with mild TBI was 14.74, in moderate TBI 14.39 & 11.83 in severe cases of TBI. The Mean GCS was 11.83 ± 2.89 at admission, it was 12.73±2.7 at 24 hours, 13.78± 2.7 at 72 hours, & 14.2±2.97 at discharge.

Mean GCS score: The current study indicates that within a group of 90 participants, the average age of patients with mild traumatic brain injury (TBI) was 43.7 years. In comparison, those with moderate TBI had an average age of 42.09 years, while patients with severe TBI had a mean age of 45.92 years.

On the other hand, the research by Vikul Kumar et al. included 216 individuals aged 18 to 65 years, revealing an average age of 36.02±16.08 years for mild TBI, 31.03±18.44 years for moderate TBI, & 28.18±13.9 years for severe TBI.

The demographic profile was predominantly younger males, particularly in the 20 to 40 age range, with 154 males (71.3%) & 62 females (28.7%). In terms of treatment outcomes, effectiveness was evaluated using the Glasgow Outcome Scale at the time of patient discharge from the hospital. The study reported a mortality rate of 13.3% (n=12). In contrast, existing literature suggests that mortality rates for TBI can vary from 35% to 42%, particularly among individuals aged 15 - 25 years. [18]

Outcome & Glucose levels:

Patients with severe traumatic brain injury (TBI) showed significantly elevated average blood glucose levels upon admission, 24 hours, & 72 hours after the injury. The persistence of hyperglycemia at the discharge time mark was associated with adverse clinical outcomes, including increased mortality rates & a higher risk of entering a vegetative state. In contrast, patients whose blood glucose levels fluctuated by less than 20 mg/L between admission & 24-hour assessment exhibited better recovery & moderate disability levels.

RBS levels & outcome: In our investigation, we observed a mortality rate of 4.4% among 90 cases of traumatic brain injury (TBI), all of which were classified as severe TBI. The mean random blood sugar (RBS) level at the time of admission for the deceased cases was 211.25±57.789 mg/dL, in contrast to a mean RBS of 191.2558±58.8697 mg/dL for those who survived.

Blood glucose levels: Upon admission, the mean blood glucose level was noted to be 192.144±58.6 mg/dl, which decreased to 147.344± 26.4 mg/dl after 24 hours, 134.911±28.0960 mg/dl at 72 hrs, & 131.844±28.2198mg/dl. Importantly, changes in trauma severity were significantly associated with variations in blood glucose levels. In our study, the mean blood glucose levels at admission for patients with mild traumatic brain injury were 148 ± 20.526 mg/dl, which declines to 130.48 ± 20.65 mg/dl at the discharge. For patients with moderate TBI, the initial glucose levels were 232.2 ± 51.9 mg/dl, decreases up to 126.5 ± 26.3 mg/dl at discharge. In cases of severe TBI, the initial blood glucose level was 261.3 ± 25.03 mg/dl, which further declines up to 150.7 ± 47.8 mg/dl at the time of discharge.

Our results showed that patients with severe head injuries had higher serum glucose levels, averaging 261.333 mg/dL, compared to those with moderate head injuries.

Walia et al. proposed that hyperglycemia is a more significant predictor of outcomes in a group of 338 patients with head injuries than mean arterial blood pressure. In a regression analysis that included both factors, each was independently associated with

mortality; however, the relationship between blood glucose levels & mortality was notably stronger than that of mean arterial blood pressure. The exact mechanisms by which glucose worsens the ischemic threshold in neurons following injury are not yet fully understood. Research involving neuronal cultures exposed to ischemic conditions showed that removing glucose from the medium reduced post-ischemic superoxide production & cell death. These findings suggest that glucose plays a crucial role.19

Management:

In our study cases of TBI, the conservative management done in 59 (65.6%), surgery management in 31 cases (34.4%). The TBI cases those were going surgical process had blood sugar levels of 193.45±55.87 mg/dl at admission, 145.5±19.5mg/dl at 24 hrs of admission, 130.16±18.5 mg/dl at 72 hrs of admission, & 133.16±27.4 mg/dl at discharge. The TBI cases those were managed through conservative method had blood sugar levels of 191.457±60.5 mg/dl at admission, 148.27±29.4mg/dl at 24 hrs of admission, 137.406±31.8 mg/dl at 72 hrs of admission, & 131.15±28.8 mg/dl at discharge.

The cases those underwent surgical management had GCS score of 11.612±3.148 at admission, 13±2.7 at 24 hrs of admission, 14.19±2.48 at 72 hrs of admission, & 14.48±2.24at discharge. Whereas cases those underwent conservative treatment had GCS score of 11.9±2.7 at admission, 12.59±2.7 at 24 hrs of admission, 13.5±2.9at 72 hrs of admission, & 14.1±3.3at discharge.

Correlation analysis:

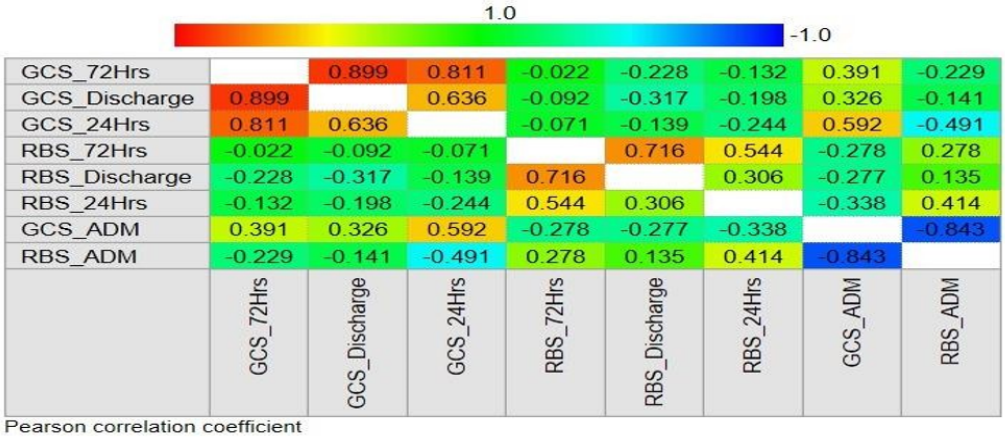

Figure 1:

A significant negative correlation noted between admission RBS & admission GCS with r value -0.843 (p<0.0001), admission RBS & 24 hours GCS with r value -0.491 (p<0.0001), admission RBS & 72 hours GCS with r value -0.229 (p=0.030). Whereas non-significant negative correlation found between admission RBS & discharge GCS with r value -0.141 (p=0.185). Blood sugar levels at 24 hours were negatively correlated admission GCS (r=- 0.338, p=0.001), 24hrs GCS (r=-0.244, p=0.020). But non-significant negative correlation found between 24hrs RBS & 72hrs GCS (r=-0.132, p=0.216), & Discharge GCS(r=-0.198, p=0.062).

Blood sugar levels at 72 hours were negatively correlated Admission GCS (r=- 0.278, p=0.008). Whereas non-significant negative correlation found between 72hrs RBS & 24hrs GCS (r=-0.071, p=0.504), 72 hrs GCS (r=-0.022, p=0.835), and discharge GCS (r=-0.092, p=0.389).

Blood sugar levels at discharge shows significant negative correlation with admission GCS (r= -0.277, p=0.008), 72 hrs GCS (r=-0.228, p=0.031), and the discharge GCS (r=-0.317, p=0.002). Whereas non-significant negative correlation found between discharge RBS & 24 hrs GCS (r=-0.139, p=0.191). Overall, a notable negative correlation was found between GCS scores & blood glucose levels, with a correlation coefficient of r = −0.548 (p < 0.0001). The exposure of the ischemic brain to high glucose levels has been shown to trigger anaerobic glycolysis, resulting in the detrimental buildup of lactic acid. Neuronal damage from traumatic brain injury (TBI) is worsened by oxidative stress, hypoxia-induced cell death, & neuroinflammation, all of which are aggravated by the neurotoxic effects of elevated glucose. Numerous studies have indicated that increased glucose levels exhibit pro-apoptotic & pro-oxidant properties. Research has found that endothelial cells subjected to high glucose conditions undergo changes in their growth, adhesion, & synthetic functions. Additionally, it has been confirmed that functional endothelial cells can undergo apoptosis in high glucose environments. [20] Lorenzi et al. argue that elevated glucose levels disrupt cell cycle regulation, leading to programmed cell death.21

The link between hyperglycemia & negative outcomes in TBI patients is highlighted by the increased mortality rates in this group, which range from 39% to 51%. [22]

Blood glucose levels & outcome by GCS:

This study found that individuals with severe traumatic brain injury (TBI) had significantly higher average blood glucose levels upon admission & 24 hrs after the injury. Persistent hyperglycemia at the 24-hour mark was associated with negative outcomes, such as increased mortality & a vegetative state. In contrast, patients who showed positive recovery & moderate disability had a difference in blood glucose levels of less than 20 mg/dL between admission & the 24-hour post-surgery period. Specifically, those with favorable recovery had an average admission glucose level of 191.25±58.86 mg/dL, while patients who did not survive had an average admission glucose level of 211.25 ± 57.789 mg/dL. In a study by Vikul Kumar et al, it was found that individuals with severe traumatic brain injury (TBI) had significantly higher average blood glucose levels at the time of admission & 24 hours after the injury. Persistent hyperglycemia at the 24-hour mark was linked to negative outcomes, such as increased mortality rates and a greater chance of entering a vegetative state. In contrast, patients whose blood glucose levels fluctuated by less than 20 mg/dl between admission & the 24-hour post-surgery evaluation typically showed better recovery & lower levels of disability.

The mean GCS score was significantly lower in patients who succumbed to TBI compared to those who survived. Specifically, the average GCS score for individuals who died was 9.25, whereas survivors exhibited a mean score of 11.95, indicating a statistically significant difference ($p = 0.002$)

The logistic regression analysis revealed that RBS is a predictive factor for mortality at 211.25 mg/dl & GCS is a predictive factor for mortality at 9.25 ($p < 0.001$, odds ratio [OR]: 1.55). Traumatic brain injury initiates a complex pathophysiological response involving various cellular mechanisms. Key components of this response include increased cerebral glucose uptake, reduced cerebral blood flow, uncontrolled release of excitatory neurotransmitters, ionic imbalances, and the buildup of intracellular calcium. Restoring homeostasis requires a significant boost in glucose metabolism. Experimental studies have shown that TBI results in a substantial increase in glucose utilization within the first 30 min post-injury, followed by a decrease in glucose uptake that remains low for about 5 to 10 days.

The initial phase of hyperglycolysis is linked to the disruption of ionic gradients across neuronal cell membranes, which activates energy-dependent ionic pumps. Effective management of blood sugar levels is essential after traumatic brain injury to improve clinical outcomes; however, it is important to note that most current clinical & preclinical evidence does not support strict glucose control (keeping blood glucose levels below 110-120 mg/dL) during the acute treatment of patients with severe TBI.

Liu DeRyke et al found that the highest random blood glucose level recorded within the first twenty-four hours after injury & hospital admission is the most reliable prognostic factor for predicting patient outcomes. The study noted a low incidence of hyperglycemia, which may suggest either normal or decreased serum cortisol levels. This observation contrasts with previous research & indicates a potentially higher prevalence of pituitary dysfunction among patients with head injuries in the studied group. Further research is needed to confirm these findings. While caution is advised regarding the use of glucose-containing intravenous fluids in patients with head injuries, the implications of this study are particularly relevant for Nigerian Africans facing such injuries. If these intravenous fluids are given, it is crucial to continuously monitor blood glucose levels to detect hypoglycemia, as many patients may initially present with normal glucose levels. [17]

In our examination of the effects of hyperglycemia on traumatic brain injury (TBI), we noted a significant difference in patient outcomes associated with hyperglycemia at the time of admission. Patients with hyperglycemia showed improvements in Glasgow Coma Scale (GCS) scores, longer hospital stays, & higher mortality rates compared to those with normal glucose levels. The study highlighted considerable fluctuations in blood glucose levels among patients with isolated TBI, with severe cases resulting in a significant rise in glucose levels upon admission. The average glucose level was found to correlate with the severity of isolated TBI, indicating that higher blood glucose levels at admission are associated with worse patient outcomes. As a result, blood glucose levels are identified as a crucial independent predictor of prognosis.

Effective management of hyperglycemia through an insulin protocol is vital for improving outcomes in patients with traumatic brain injury (TBI). This study involved administering a regular insulin protocol via subcutaneous injection to control blood glucose levels exceeding 200 mg/dl. It is crucial that treatment for hyperglycemia begins within the first 24 hours of patient admission. Further research is needed to determine the specific serum glucose threshold that may pose risks to TBI patients. Implementing an insulin protocol to

manage hyperglycemia when glucose levels rise above 200 mg/dl is essential for enhancing patient outcomes in TBI cases. The study included a large cohort of trauma patients in the ICU & utilized data from a Level 1 trauma center known for its high-quality surgical care, which is a significant strength of this research.

Conclusion

A significant inverse relationship was found between GCS scores & blood glucose levels ($r=-0.54$, $p<0.0001$). This study explored the correlation between hyperglycemia & the progression of traumatic brain injury (TBI) in affected patients, indicating that hyperglycemia is associated with increased morbidity & mortality in this group. Specifically, a random blood glucose measurement above 200 at admission was correlated with more severe injuries or worse outcomes. The results suggest that early hyperglycemia, especially when paired with a low GCS, may be a reliable indicator of TBI severity & prognosis, with elevated blood glucose levels potentially indicating a poor prognosis & increased mortality risk. Higher glucose levels recorded at admission, 24 hours, 72 hrs after injury, & discharge were associated with negative outcomes. Surgical interventions did not affect significant changes in blood glucose levels.

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