International Journal of Research in Medical Sciences
Paydar S et al. Int J Res Med Sci. 2018 Jan;6(1):14-19
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20175705

Original Research Article

Initial GCS and laboratory findings of patients with TBI are associated with the GOSE and mortality rate at one year

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Received: 21 October 2017
Accepted: 21 November 2017

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ABSTRACT

Background: To evaluate the relationship between presenting Glasgow Coma Scale (GCS) or laboratory data of patients with TBI and Extended Glasgow Outcome Scale (GOSE) and final outcome (deceased, survived) at one year.
Methods: 74 patients (59 males and 15 females; mean age ±SD of 40±19years) who presented with TBI were entered into the study, and their GCS and laboratory data were recorded. After one year, GOSE level and final outcome were evaluated with 11 yes/no questions obtained from the patients or their first-degree relatives.
Results: The patients with lower GCS on admission or day six, significantly had lower GOSE. Moreover, the lower the GCS in the first week of admission, the poorer the final outcome. Among laboratory data, the base deficit (BD) level of -6 or worse on admission was an indicator of mortality at one year. Hypernatremia was the only laboratory factor which predicted poor GOSE after a year. Furthermore, patients with serum hypernatremia, hyperkalemia, or high PTT levels on the first week of admission had poor final outcome.
Conclusions: Presenting GCS and metabolic derangements are reliable indicators of long-term outcome and GOSE at one year.

Keywords: Base deficit, Coagulopathy, Glasgow coma scale, Glasgow outcome scale extended, Hypernatremia, Traumatic brain injury

INTRODUCTION

Traumatic brain injury (TBI) is the main cause of mortality and morbidity in people under 45 years old in developing countries, most likely due to increased motor vehicle usage and violence in the cities.[1,2]

A large number of patients with TBI are admitted to intensive care units, but outcomes after one year are difficult to anticipate during the acute period.[3] Some patients will have no chronic manifestations; whereas, others will have life-long neurologic deficits.[4] Patients and their immediate family often ask the physicians if any future deficits may occur. Therefore, physicians need some ability to predict patient outcomes at the time of admission to the hospital.

The Glasgow Coma Score (GCS) is a reliable measurement of consciousness that assesses the eye, motor and verbal responses of patients with depressed brain function, including TBI. The GCS can be a good predictor of short-term outcome in patients with TBI (10); however, its role as a predictor of long-term outcome is still under investigation.

With the Glasgow Outcome Scale (GOS), patients with TBI are categorized into five groups: dead, vegetative state, severe disability, moderate disability and good recovery; however, higher sensitivity can be achieved if the patients are classified into eight groups according to the Extended Glasgow Outcome Scale (GOSE) categorization.[2] The GOSE classification is as follows: dead, vegetative state, lower severe disability, higher

severe disability, lower moderate disability, higher moderate disability, lower good recovery and higher good recovery status.[5,6] Moreover, the GOSE also considers physical, social and cognitive outcomes.[2] Although the roles of GOS and GOSE in the prediction of TBI outcomes have not been completely proven, in general, they can be beneficial for predicting functional outcomes and as indicators for starting rehabilitation sooner which can, ultimately, reduce the cost of care.[7-10]

Arterial blood gas demonstrates the acid-base balance of a patient.[11] Base deficit (BD) measurements via blood gas tests can be easily obtained on the first day of hospital admission. Some previous studies evaluated the correlation between BD and injury severity, in-hospital length of stay, or Glasgow outcome scale (GOS) in pediatric and adult blunt trauma and head trauma patients.[12-15] Although one of these studies evaluated abnormal BD as a predictor of short-term prognosis, to the best of our knowledge, the role of the initial BD in predicting long-term outcomes of patients with TBI has not been addressed.[13]

Other laboratory data, such as coagulation profile, blood sugar and electrolyte levels, have been evaluated in patients with TBI. Coagulopathy is a frequent event in TBI and is associated with poor prognosis.[16,17] Since TBI is a stressful event, hyperglycemia can occur due to stress response. Moreover, hyperglycemia on admission has some relation with risk of developing coagulopathy.[18] Electrolyte levels, such as sodium and potassium, play important roles in the pathophysiology of brain edema, a critical issue for patient management and prognosis. Yet the relationship between electrolyte level and prognosis of patients with TBI is controversial.[19,20] The goal of this study is to assess the relationships between presenting GCS and initial laboratory data and the GOSE at one year and long-term outcome of patients with TBI.

METHODS

This prospective study was conducted at Rajaee Hospital affiliated with Shiraz University of Medical Sciences (SUMS) and was approved by the Ethics Committee of SUMS. The patients and/or their immediate family members were informed about the aim of this study, as well as safety and security measures, before consent was obtained. During the 18months from October 2010 to March 2011, a total of 110 patients presented to an emergency room due to TBI and agreed to participate in the study. The patients were examined by an expert neurosurgeon and a general surgeon. Injury severity score (ISS) and GCS at the first, second, fourth and sixth day of hospital admission were evaluated. The patients received routine treatment and management as indicated in the course of hospitalization.

Laboratory data including sodium (Na), potassium (K), prothrombin time (PT), partial thromboplastin time (PTT), international normalization ratio (INR), base deficit (BD), calcium (Ca), creatinine phosphokinase (CPK), lactate dehydrogenase (LDH) and blood sugar (BS) on days 1, 2, 4, and 6 were collected. Furthermore, the final outcomes (deceased, survived) were recorded.

The aim of this study is to determine which parameters can predict the GOSE after a year. Therefore, we translated the GOSE categories into descriptions which are precise and could be easily understood by the general population. After one year, a neurosurgeon called the patients and asked eleven yes/no questions to evaluate the patients' conditions. The GOSE scores were assessed according to the replies to the questions. The main parts of these questions are cognitive, behavioural or emotional functioning, as well as daily living and employment activities. These are the eleven yes/no questions:

- Does the patient breathe independently or is a ventilator needed?
- Does the patient have tracheostomy tube?
- Does he/she communicate verbally?
- Does he/she understand others' conversations?
- Does he/she follow commands?
- Can he/she eat food independently?
- Can he/she go to the bathroom independently?
- Can he/she go out for shopping independently?
- Can he/she do his/her personal affairs (e.g. driving, social communication) independently?
- Has he/she returned to his/her previous job?
- Has he/she returned to what he/she was like before the injury?

Statistical analysis was performed using Statistical Package for Social Sciences version 15.0 (SPSS Inc., Chicago, Illinois). Before selecting the appropriate analysis test, the normality of data was checked and parametric (t-test, ANOVA) or non-parametric (Kruskal-Wallis and Mann-Whitney) analyses were performed. For nominal and ordinal variables, the Chi-square analysis test was also performed. For all analyses, the statistically significant difference was defined as $p<0.05$. Data are reported as the mean ±SD.

RESULTS

Patient characteristics

Table 1: Demographic characteristics of patients.

	N (%)	Mean age ±SD	Male	Female
Survived	47 (63.5)	38 ± 17	39	8
Deceased	27 (36.5)	45 ± 22	20	7
Total	74 (100)	40 ± 19	59	15

In the present study, initial physical exam and laboratory data were obtained on 110 patients with TBI. The GOSE outcomes at one year were evaluated in 74 cases; the remaining 36 cases were lost to follow-up; therefore, they

were excluded from the study. The mean ±SD age of 74 patients was 40±19 years (Table 1). After one year, 27 (36.4%) patients had died and 47 (63.5%) patients had survived. In terms of GOSE categorization, half of the patients were in the extreme ranges. For instance, one quarter had died (27/74, 25.6%); whereas, another quarter were in good condition (lower good recovery and upper good recovery; 27/74, 25.6%). Table 2 shows the GCS and outcome scores of patients who entered the study.

Table 2: Different outcome scores and GCS of patients.

CHARACTERISTICS	N (%)	CHARACTERISTICS	N (%)
GCS ON DAY 1		GCS ON DAY 2	
GCS<5	20 (27)	GCS<5	14 (18.9)
6≤ GCS ≤8	32 (43.2)	6≤ GCS ≤8	31 (41.9)
GCS>8	22 (29.8)	GCS>8	29 (39.2)
GCS ON DAY 4		GCS ON DAY 6	
GCS<5	13 (17.6)	GCS<5	16 (21.6)
6≤ GCS ≤8	25 (33.8)	6≤ GCS ≤8	13 (17.6)
GCS>8	36 (48.6)	GCS>8	45 (60.8)
Change in GCS in the first week		Final outcome	
Decreased	19 (25.6)	Survived	47 (63.5)
Increased	46 (62.2)	Deceased	27 (36.5)
Unchanged	9 (12.2)		
Injury severity score		GOSE	
Mild (0-8)	0 (0)	Dead	27 (36.5)
Moderate (9-15)	3 (4.1)	Vegetative state	2 (2.7)
Severe (16-24)	6 (8.1)	Lower severe disability	4 (5.4)
Critical (≥25)	65 (87.8)	Upper severe disability	2 (2.7)
		Lower moderate disability	6 (8.1)
		Upper moderate disability	6 (8.1)
		Lower good recovery	9 (12.2)
		Upper good recovery	18 (24.3)

GOSE at one year and the GCS during the first week in the hospital

On admission, 22/74 (29.7%) had GCS>8, 32 (43.3%) had 6≤ GCS ≤8 and the remaining 20 cases (27%) had GCS<6. Most patients demonstrated improvements in the GCS score between the first day to the sixth day of admission (Figure 1). On the sixth day of admission, 45/74 (60.8%) had GCS>8, 13 (17.6%) had 6≤ GCS ≤8, and the remaining 16 (21.6%) had GCS<6. Both the presenting GCS and the GCS on day 6 correlated significantly with the GOSE after one year. The lower the GCS on admission or day 6, the poorer the GOSE score after one year. These differences were statistically significant with p=0.032 and p=0.007 on day 1 and day 6, respectively.

GOSE and initial laboratory data

Laboratory data (Na, K, Ca, CPK, LDH, PT, PTT, INR, BS, and BD) from the first week of admission were collected, and their values in the prediction of GOSE outcome were evaluated. Most of our patients (59/74, 79.7%) had normal sodium levels (135-145 mEq/L), 11 (14.9%) had hypernatremia (Na>145 mEq/L), and only four (5.4%) had hyponatremia (Na<135 mEq/L). The sodium level on the fourth day of admission was significantly associated with GOSE outcome (p=0.048). Hypernatremia was seen more often in patients with lower GOSE scores, and 6 out of 10 hypernatremic patients had poor GOSE outcome. Other laboratory data (K, Ca, CPK, LDH, PT, PTT, INR, BS, and BD) showed no statistically significant relationship with GOSE outcome (Table 3).

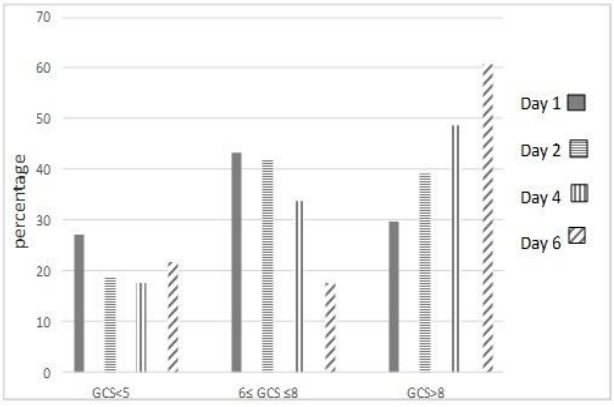

Figure 1: GCS changes during the first week.

Mortality rate and the GCS during the first week in the hospital

The mortality rate after one year was assessed in 74 patients. Approximately 64% of our patients were alive at one year (47/64). The mean GCS on admission, and at day two, four, and six were significantly lower in patients who died ($p<0.05$). The GCS was categorized to low (GCS<6), medium (6≤ GCS ≤8), and high (GCS>8) in day one, two, four and six, and statistical analysis showed that mortality rate in these three categories was significantly different with p-value <0.001, <0.001, 0.017 and 0.003 respectively. Both Chi-square and T-test analyses showed that surviving patients had higher GCS than deceased patients.

Table 3: Some evaluated laboratory factors and their associated P value.

	N (%)	Mortality P value	GOSE p value
Sodium (Na)			
<135	4 (5.4)	0.017*	0.048*
135-145	59 (79.7)		
>145	11 (14.9)		
Potassium (K)			
>3.5	12 (16.2)	0.005*	0.837
3.5-4.5	51 (68.9)		
>4.5	11(14.9)		
Base deficit (BD)			
≤ -6	41 (55.4)	0.043*	0.151
> -6	33 (44.6)		
PTT			
25-35	10 (13.5)	0.030*	0.146
>35	64 (86.5)		

Mortality rate and laboratory data

Among our patients, 41 (55.4%) had a BD level of -6 mEq/L or worse r (≥-6 mEq/L) on the first day of admission. The relationship between BD level on the first day of admission and mortality rate was statistically significant. The analysis showed that those who had died by one-year follow-up had worse BD on the first day of admission ($p=0.043$). The BD and mortality rate were evaluated in ROC curve, and the threshold (cut-off point) was -6mEq/L. In Chi-square analysis, all patients who were alive at one year had an initial BD level of better than -6 mEq/L.

In this study, we found that hypernatremia was associated with a poor final outcome. Of the 85% of patients with normal sodium levels or hyponatremia (63/74), 36% (23/63) were alive at one year. In contrast, 45% (5/11) of the patients with hypernatremia had died at one year. This difference in outcome was statistically significant at day four ($p=0.017$) and day 6 ($p=0.022$).

Evidence of coagulopathy was common in our patients, with 86.5% (64/74) having abnormal PTT on the first week of admission. Interestingly, the mean PTT level was lower in the group that survived (42.7±7) compared to the deceased group (47.5±10). This difference was significant via T-test analysis, $p=0.03$ (Table 3).

We also found that the mean level of potassium on admission was normal in the group that survived (4±0.6 mEq/L) but elevated in deceased patients (5.8±8.1 mEq/L). This difference was significant, $p=0.005$.

We found no statistically significant relationship between other laboratory data (Ca, CPK, LDH, PT, INR and BS) and final outcome. Moreover, no significant relationship between ISS and GOSE or mortality rate was observed.

DISCUSSION

We have shown that higher GCS scores on admission are strongly associated with improved GOSE after one year in patients with TBI. Our study also shows that GCS on day six predicts long-term outcome. These observations are in accordance with some previous studies.[21,22] Corral et al evaluated 214 patients with TBI and reported that GOS and GOSE at 6months and 1year were better in patients with higher GCS score on admission (6-8) than in the group with lower GCS (3-5).[23]

Furthermore, GCS scores are generally regarded as the most accurate predictor of outcome in patients with head injury when combined with age and pupillary response and when broad outcome categories are used.[21] The motor component of the GCS yields similar prediction rates as the summed GCS score, and better prediction occurs with very high or very low GCS scores.[21] One study revealed that after six months, the evaluation of outcome of patients with TBI is inconclusive, so making an assessment of patient's outcome after one year seems more reasonable.[23]

Some studies have indicated that BD evaluation on admission is important in the assessment of patients with trauma. When a patient presents with trauma, the chance of bleeding and hypotension is high. Low blood pressure and concomitant tissue hypoxia lead to anaerobic oxygen metabolism and lactic acidosis, which result in increasing BD level.[24]

One study demonstrated that the BD level is significantly different between survived and deceased patients with TBI 14, and BD worse than -5 was an indicator of mortality and injury severity.[12] In two other studies, patients with BD more negative than -8 Eq/L and -5mEq/L had a higher risk of mortality.[12,13] Accordingly, in our study, the BD level of -6mEq/L or worse was the best indicator of mortality in patients with TBI.

Our study revealed that patients with lower GCS on the first week of admission (GCS scores 3-5) had higher rates of mortality (P<0.001). This result was completely compatible with most previous studies, which showed the significant difference in mortality rate between patients with GCS scores of 3-6 and those with higher GCS scores 3. This result was confirmed by a meta-analysis study including 24 studies, which showed that the mortality is significantly different between patients with higher GCS and patients with lower initial GCS.[25]

Brain edema is an important issue in management of patients with TBI and has complex cellular and molecular mechanisms. Sodium-water balance plays an important role in its occurrence. However, the exact mechanism and effect on the outcome are not completely figured out.[26] Since dysnatremia can affect cerebral edema, we hypothesized that abnormal sodium levels would be associated with changing the outcomes.[27] According to the present study, patients with hypernatremia experienced worse long-term outcome (GOSE) compared to isonatremic or hyponatremic patients. Nevertheless, most of our patients had normal sodium level but to be more precise, it is recommended to re-evaluate the relation between sodium level and GOSE in future studies considering more patients. This study did not show any significant correlation between serum levels of BD, K, Ca, CPK, LDH, PT, PTT, INR, BS and GOSE at one-year. To the best of our knowledge, no study in the literature assessed the relation between these laboratory factors and long-term GOSE outcome.

Clinicians have a trend to keep the sodium level of patients with TBI slightly higher than normal so that it helps reducing water content of the brain, declining intra cranial pressure, morbidity and mortality consequences.[28] On the other hand, some studies reported higher mortality rate in hypernatremic patients. Aiyagari et al showed that mortality rate of hypernatremic patients admitted to the neurologic/neurosurgical intensive care units was 20% more than that of isonautremic patients.[19] Also, in this study, mortality rate was higher in patients with hypernatremia.

A significant correlation between the potassium level on the first day of admission and mortality rate was observed. A study showed that patients with lower potassium levels revealed higher rates of mortality. Lazar et al claimed that hypokalemia occurred frequently in pediatric patients with TBI; however, no correlation between potassium level and GCS level, severity of trauma or patient outcomes was observed.[29] Another study reported the difference between potassium level in serum sample and cerebral microdialysis sample. They suggested that brain extracellular potassium may serve as a biomarker for brain tissue injury in patients with poor-grade aneurysmal sub-arachnoid hemorrhage.[20] It seems that potassium plays a role in pathophysiology of brain injury.The relation between coagulopathy and TBI was evaluated in previous studies and they reported that coagulopathy can occur frequently in patients with poor outcome.[30,31] This study also showed higher mortality rate among patients presented with higher levels of PTT but no statistically significant relation between coagulation factors and long-term GOSE was observed.

CONCLUSION

In this study, the patients presenting with worse BD level or abnormal Na, K and PTT had poor outcome. The patients with hypernatremia on the fourth day of admission had lower GOSE scores at one year; however, other laboratory factors were not associated with GOSE at one year. On the other hand, initial GCS was associated with both mortality and GOSE at one year.

ACKNOWLEDGEMENTS

Authors would like to thank all the physicians and personnel of Trauma Research Center, Rajaee Hospital for their cooperation, and we would like to give very special thanks to Dr. Rika Messer, at Department of Neurology and Neurosurgery, Johns Hopkins Medical Institutions, for her great recommendations and editing the manuscript.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee SUMS

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Cite this article as: Paydar S, Bordbar E, Taghipour M, Khalili H, Jafari M. Initial GCS and laboratory findings of patients with TBI are associated with the GOSE and mortality rate at one year. Int J Res Med Sci 2018;6:14-9.