

Original Research Article

Effect of alcohol on Glasgow Coma score in traumatic brain injury: a single, center, cross-sectional study

Minnu Thomas¹, Appu Suseel^{1*}, Suresh Samuel David²

¹Department of Emergency Medicine, Jubilee Mission Medical College and Research Institute, Thrissur, Kerala, India

²Department of Emergency Medicine, Pushpagiri Medical College and Research Institute, Thiruvalla, Kerala, India

Received: 17 March 2023

Accepted: 16 April 2023

*Correspondence:

Dr. Appu Suseel,

E-mail: appuariyedath@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: To study the effect of blood alcohol levels on GCS in Traumatic brain injury patients (TBI) and relate the findings to brain injury severity based on the admission CT scan.

Methods: This cross-sectional study with a comparison group is conducted in Emergency Department(ED) of Pushpagiri Institute of Medical Sciences and Research Centre, Central Travancore, Kerala, India from April 2016 to September 2017 including all patients of 18-70 years of age presenting with head injury. 200 participants, 100 each of alcohol intoxicated and non- intoxicated were selected by consecutive sampling. GCS, Blood alcohol concentration-BAC (derived from the reading of alcohol breath analyzer) and admission CT Rotterdam Score are collected and analyzed at the end of study.

Results: When CT Rotterdam Score is 1-3, GCS was found to decrease with increase in BAC (Chi-square test p value=0.011; Spearman's Rank Correlation coefficient $r_s=-0.326$). Independent t-test showed that at BAC 1-100 mg/dl, mean GCS decrease by 1.6 while only same decrease is found when the BAC levels ranges from 100-400 mg/dl. When the CT Rotterdam score is 4-6, no significant correlation was found between GCS and BAC (p value=0.092; $r_s=0.214$). In the presence of alcohol, GCS had sensitivity 87.5% and specificity 70% in comparison to alcohol absent TBI patients (sensitivity 98.5%, specificity 69.7%). When features of hypoxia and shock are present, GCS have good agreement with actual CT findings of TBI.(Kappa coefficient: K 0.659, sensitivity 76%, specificity 100% in alcoholics and K 0.756, sensitivity 100%, specificity 80.6% in nonalcoholic).

Conclusions: Even in the setting of alcohol intoxicated TBI patient, Glasgow coma score is a useful tool for quick decision making in emergency department.

Keywords: Blood alcohol concentration, Glasgow coma score, Rotterdam CT score, Traumatic brain injury

INTRODUCTION

The Global Burden of Disease study shows that over a quarter of the world's trauma deaths occur in India of which approximately 14.7% of mortality rate is attributed to Traumatic Brain Injury (TBI).^{1,2} Glasgow coma score (GCS) introduced by Teasdale and Jenneth in 1974, has been extensively used as a quick means in emergency department for evaluating TBI severity.³ There are many

factors that can result in low GCS other than the actual insult to the brain; alcohol intoxication being the commonest of them. If the low GCS is wrongly attributed to alcohol, underestimation or unnecessary delay of diagnostic and therapeutic interventions can occur. Conversely, overestimation of injury severity leads to use of expensive and highly specialized treatment. This prospective study conducted in Kerala state of India, is aimed at studying the effect of blood alcohol levels on

GCS in Traumatic brain injury patients (TBI) and relate the findings to brain injury severity based on the admission CT scan. The amount of alcohol intoxication is measured by alcohol breath analyzer which is a faster modality of assessment: useful in an emergency department. A comparative study on GCS in TBI patients based on breath alcohol values and admission CT is not published in a similar study setting before, hence the need to do this study. Moreover, the study setting chosen-Kerala is one of the leading states in the country in terms of alcohol consumption and related accidents. The study objectives are to assess the correlation between (i) blood alcohol concentration and GCS, (ii) to compare GCS with Rotterdam CT Scores and also (iii) to find out the association of other systemic factors on GCS in subjects with TBI under the influence of alcohol. The study is hypothesized as the GCS will be lower in patients with traumatic brain injury under the influence of alcohol when compared to patients with TBI not under the influence of alcohol.

METHODS

This was an observational study conducted in the Emergency Department(ED) of Pushpagiri Institute of Medical Sciences and Research Centre, 900-bed multi disciplinary super specialty hospital in Central Travancore, Kerala, India, after approval by the institutional research and ethics committee (PIMSRC/E1/388A/30/2016). The study design was cross-sectional study with a comparison group. It was conducted over a period of 18 months from April 2016 to September 2017.

All patients between 18 years and 70 years of age presenting with head injury to ED were included in the study.

Exclusion criteria includes intubated patients or those managed with sedatives and muscle relaxants from another hospital on arrival in our ED, unstable patients for whom emergency CT cannot be taken and high velocity penetrating head injury patients (such as gunshot wounds, depressed skull bone fractures), those unwilling to consent.

Sample size calculation

Assuming a significance level of 5% and a power of 80% with 50% of the non-intoxicated subjects having a GCS of 15 and 30% of alcohol intoxicated subjects having a GCS of 15, the sample size is calculated as 200 head injured patients of which 100 are alcohol intoxicated and 100 non intoxicated.⁴

The head injury patients of the above mentioned age group - both alcohol intoxicated and non-intoxicated were subjected to consecutive sampling. For every participant satisfying the inclusion criteria, GCS at arrival along with other clinical findings were assessed by

resident / consultant in ED and the findings are filled up in 'Proforma for GCS Assessment'. Based on GCS values subjects were grouped into three - Mild (12-15), Moderate (9-12) and severe (3-8) head injury. The Alcohol Breath analyzer reading was taken by a senior staff nurse using 'Alcosafe KX6000S' and is filled in 'Proforma for Breath Analyzer Reading'. For the patients who require urgent mechanical ventilation, the breath alcohol measurement was taken after intubation via the endotracheal suction port. Breath alcohol concentration (BrAC) measured in mg/l is converted to blood alcohol concentration (BAC) in mg/dl using the formula $BAC (mg/dl) = BrAC (mg/l) \times 210.5$. Based on the BAC, study subjects were subdivided into 4 groups of 0 mg/dl, 1-100 mg/dl, 101-150 mg/dl, >150 mg/dl. The CT scan for the head injury patient was taken after initial stabilization and findings are reported by a resident/consultant in the radiology department in the 'Proforma for Emergency Room CT Finding' which contains the various components of Rotterdam CT Score. Extra Dural Hemorrhage if present is reclassified by adding 1 to the actual score. Based on the CT findings study subjects were divided into two groups of 1-3 and 4-6 Rotterdam score and is termed as 'Mild TBI CT' and 'Severe TBI CT' respectively. The study variables GCS, Blood alcohol concentration (BAC) and the CT Rotterdam Score collected using these 3 different Pro Forma were kept confidential and subjected to analysis at the end of study period.

Statistical methods

Baseline clinico-social correlates were found out and tabulated. Mean, Standard deviation, Median, and Range of GCS scores at different breath alcohol levels were calculated. Agreement between GCS and Rotterdam Score were done for different levels of blood alcohol concentration (BAC) using Kappa statistics. Spearman's Rank Correlation coefficient (rs) is calculated to find out the relationship between BAC and GCS. Mantel Haenszel Chi-Square was done to find an association between systemic factors, BAC and GCS. P-value <0.05 was taken as statistically significant. Data entry was done using MS EXCEL. Data analysis was done using SPSS (Statistical Package for Social Sciences) version 20.0. .

RESULTS

Age- gender and mode of injury distribution

In this study, 200 individuals were included out of which 173 (86.5%) were males and 27 (13.5%) were females. The male-to-female ratio of Traumatic Brain injury victims was found to be 6.4:1. Gender in TBI was reported as a male-to-female ratio of 3.8:1, 79.2% male and 20.8% female patients in India. The male-to-female ratio was 2.7:1 among nonalcoholic TBI. Adolescents and young adults are the majority of TBI patients as described in a previous similar study. In the present study, 67% TBI victims (134 of 200) were 50 years of

age or younger. Of these 84 were under the influence of alcohol. When age is greater than 50 years, 75% TBI victims were non alcoholics (50 of 66) (Table 1). The mean age of TBI among alcoholics was 37±13.2 and that of non-alcoholics was 47.7±17.3. In the study it was observed that 83.5% (167 of 200) of the head injured patients were victims of road traffic accidents. 15.5% sustained head injury secondary to fall from height of more than 3 feet and 1% were assaulted.

Distribution of clinical features of TBI in study population

Loss of consciousness (LOC) was the commonest symptom identified in the study population that formed 61% (122 of 200). Chi-square test was done to find out the correlation between alcohol and LOC in TBI, which gave a p-value of 0.384, concluding that alcohol has no effect on the event of loss of consciousness in head injury. 138 subjects (69%) had features suggestive of base of skull fracture- like raccoon eyes or ENT bleed. 53% of total subjects (106 of 200) had signs of raised intracranial tension like vomiting, seizure, amnesia (antegrade or retrograde or both).

BAC and GCS adjusted to severity of TBI based on Head CT

The study observed that 84% of the alcoholics and 89% of the non-alcoholics were reported to have GCS that were consistent with the actual CT findings. In alcoholics when 12.5% of the Mild TBI patients were wrongly attributed to having severe head injury according to GCS, only 1.5% was mistaken in non-alcoholics. It was also observed that subjects were reported to have mild to moderate head injury in accordance with GCS assessment but had severe TBI in reality. These were 30% of the alcoholic group and 30.3% among the nonalcoholic group which is a very significant observation (Table 2). These might be attributed to the limitations of GCS. There is controversy over diminishing accuracy when the numerical scores of the three components in GCS are added. Apart from this, TBI victims who are at potential risk of ‘talk and die’ like those with extradural hemorrhage, contusions especially bifrontal, diffuse axonal injury, vault fracture, who are on anticoagulants or those who have other comorbidities, will all fall in this group assessed with mild to moderate head injury based on GCS but CT findings with severe TBI. ‘Talk and die’ patients describe victims who present with a mild head injury (GCS 13-15) and then subsequently deteriorate and die from intracranial causes.

While stratifying the severity of TBI using Head CT and comparing it with recorded GCS at various BAC grouped as four, it was observed in the study that the mean and median GCS was decreasing with increase in level of alcohol intoxication in Mild TBI whereas no similar finding was observed in case of Severe TBI patients (Figure 1).

Table 1: Baseline characteristics and vital parameters in study population.

Participant Characteristics	Alcoholics (n=100)	Nonalcoholics (n=100)		
Age (years)	18-50	84	50	
	51-70	16	50	
Gender	Male	100	73	
	Female	0	27	
Mode of injury	RTA	93	74	
	Fall from >3 feet	7	24	
	Assault	0	2	
Clinical features	LOC	64	58	
	Vomiting	22	32	
	Seizure	1	5	
	Amnesia	24	22	
	ENT Bleed	48	39	
	Raccoon eyes	31	20	
GCS	Local injury	54	49	
	Mild	60	64	
	Moderate	16	12	
Rotterdam Score	Severe	24	24	
	Mild TBI CT	80	67	
Severe TBI CT	Severe TBI CT	20	33	
	Vital Parameters (Mean±SD)	1 – 100mg/dl (n=45)	101 - 150mg/dl (n=21)	≥ 151mg/dl (n=34)
PR (/ min)	89±12.63	85.4±16.5	82.4±16.64	85±18.02
SBP (mm of Hg)	130±20.78	122±27.68	123.6±18.92	131.83±28.73
DBP (mm of Hg)	79.11±11.45	75.24±15.7	76.53±11	79.6±13
RR (/min)	19.78±4.14	20.52±4.6	18.88±4.58	19.32±4.102
SpO ₂ (%)	95.2±8.07	93.24±5.57	92.76±11.06	94.76±6.24
GCS	12.53±3.35	11.24±3.8	9.76±4.3	11.7±4.11

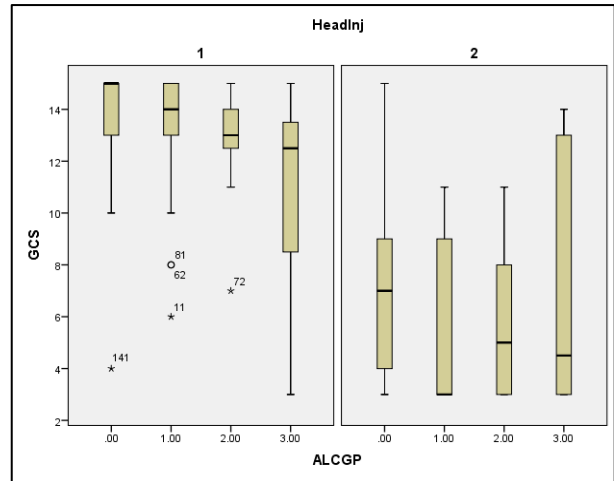
Correlation between GCS and Alcohol (BAC) were analyzed after confirmation of brain injury severity by CT Rotterdam Score. Spearman’s rank correlation showed that in Mild TBI CT increase in Blood alcohol concentration causes a decrease in GCS (rs=- 0.326; p value=0.003) indicating weak negative correlation, which is significant. In Severe TBI CT, there is no significant correlation between BAC and GCS (rs=0.214; p value =0.364). The Chi-square test showed that in Mild TBI, alcohol have a significant effect on GCS (p value 0.011),

whereas in Severe TBI alcohol has no significant effect on GCS (p-value 0.0927) (Figure 2).

The effect of alcohol on GCS in Mild TBI was studied using independent t test, in 2 groups 1-100 and 100-400 mg/dl (40 in each group). It was found that in Mild TBI, when alcohol increases from 1-100 mg/dl, the mean GCS decrease by 1.6 (p value 0.008). A similar decrease is found when alcohol increases above 100 mg/dl (1.6 (p value 0.008)).

Effect of alcohol and other systemic insults on GCS in head injured patients

From the 200 study subjects, individuals with hypoxia, hypotension and clinical features suggestive of shock were sorted. Study subjects with any/combination of characteristics of PR>100/min, SBP<90 mm of Hg, DBP<60 mm of Hg, RR>24/min, RR<8/min or SpO₂<94% in room air were selected as they have systemic insults and thereby more of secondary brain injury.



(Headinj 1 and 2 indicating mild CT TBI and severe CT TBI and ALCGP indicating BAC groups: 0,1-100,100-150, >150 mg/dl)

Figure 1: Relationship between GCS and BAC adjusted to brain injury severity.

Table 2: Distribution of study sample based on GCS and Rotterdam Score and relation between them.

Parameters	Distribution of study sample based on GCS and Rotterdam CT Score			Agreement between GCS and Rotterdam CT Score	
	Rotterdam CT Score	GCS 9-15	GCS 3-8	Fischer’s Exact test	Kappa Co-efficient
Alcoholics (n=100)	Mild TBI CT	70 (87.5%)	10 (12.5%)	p-value	
	Severe TBI CT	6 (30%)	14 (70%)	< 0.0001	0.535
Nonalcoholics (n=100)	Mild TBI CT	66 (98.5%)	1 (1.5%)	p-value	0.733
	Severe TBI CT	10 (30.3%)	23 (69.7%)	< 0.0001	

Table 3: Distribution of study sample with features suggestive of shock/hypoxia based on GCS and Rotterdam Score and relation between them.

	Distribution of study sample with features suggestive of shock/ hypoxia based on GCS and Rotterdam CT score			Agreement between GCS and Rotterdam CT Score of study sample with features suggestive of shock/ hypoxia	
	Rotterdam CT Score	GCS 9- 15	GCS 3-8	Pearson Chi-square test	Kappa statistics
Alcoholics n=36	Mild TBI CT	19 (76%)	6 (24%)	p-value <0.0001	0.659
	Severe TBI CT	0	11 (100%)		
Nonalcoholics n= 33	Mild TBI CT	12 (100%)	0	p-value <0.0001	0.756
	Severe TBI CT	4 (19%)	17(81%)		

Table 4: Comparison of measures of diagnostic accuracy of GCS based on CT findings in entire study population as well as in patients with features suggestive of shock/ hypoxia.

GCS compared to Rotterdam CT findings	Alcoholics		Non alcoholics	
	n=100	n=36	n=100	n=33
Sensitivity	87.5%	76%	98.5%	100%
Specificity	70%	100%	69.7%	80.95%
Positive likelihood ratio	2.92	-	3.25	5.25
Negative likelihood ratio	0.18	0.24	0.02	-

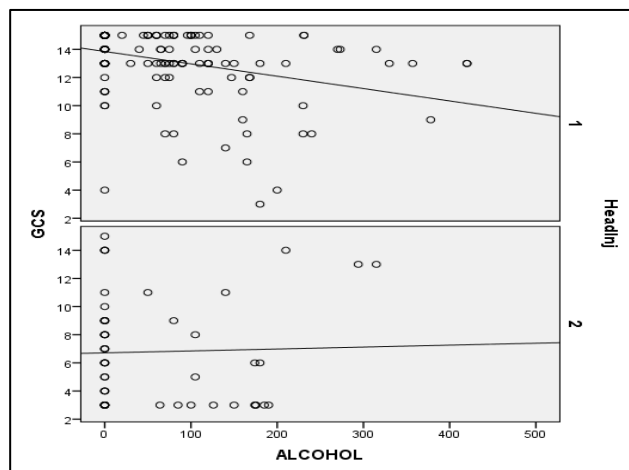


Figure 2: GCS against BAC in CT based Mild TBI CT (1) and of Severe TBI CT (2).

In the study, 36% of the alcoholics and 33% of the non-alcoholics satisfied the conditions for systemic insults. Statistical analysis proved that both in alcoholics and non-alcoholics, even in the presence of features suggestive of hypoxia /hypotension /shock, GCS have good agreement with actual CT findings of traumatic brain injury (Table 3 and 4).

DISCUSSION

The major results of the present study are: 1) When the CT Rotterdam Score is 1-3, GCS was found to decrease with increase in BAC: when the BAC ranges from 1-100mg/dl mean GCS decrease by 1.6 while only same decrease is found when the BAC levels ranges from 100 -400mg/dl, 2) When the CT Rotterdam score is 4-6, no significant correlation was found between GCS and BAC, 3) In alcohol intoxicated TBI, GCS had sensitivity 87.5% and specificity 70% in comparison to non-intoxicated TBI subjects (sensitivity 98.5%, specificity 69.7%), 4) When features of hypoxia and shock are present, GCS have good agreement with actual CT findings of TBI (sensitivity 76%, specificity 100% in alcoholics and sensitivity 100%, specificity 80.6% in non-alcoholics).

Some of the previous studies have used AIS (Abbreviated Injury Score) for stratifying severity of TBI.¹²⁻¹⁴ But only few studies have used the Gold Standard–Head CT, for grading severity of TBI.^{4,6,15} Lange et al. retrospectively studied 475 people involved in motor vehicle accident with available BAC, GCS and admission CT scan and concluded that - GCS scores are useful in vast majority of intoxicated patients but will likely overestimate severity of brain injury in patients with abnormal CT scans and BAC greater than 200 mg/dl.⁶ The more recent prospective study by Rundheug et al. done during a 7 year period (2004-11) took into consideration 217 patients with measured BAC. This study concluded the influence of alcohol significantly decreases GCS score in dose dependent manner in patients with Rotterdam CT

score 1-3 and in patients with 4-6 scores effects of brain injury overran the depressing level of alcohol on central nervous system. The results of the current study are comparable with that of Rundheug et al. The various factors affecting GCS such as hypoxia and hypotension were not considered previous studies except this one.⁴

While evaluating the effect of alcohol in general on TBI, multiple factors including the timing of intoxication in relation to time of injury, the degree and chronicity of intoxication, as well as the influence of other secondary injury processes determines the net effect of alcohol in a given individual.¹⁶ BAC is most commonly used metric for legal or medical purposes employed by laboratories in India. A blood sample for forensic analysis might not be taken until several hours after the time of incidence. So although accurate, the results of these tests are often delayed several hours and are not really appropriate in the clinical scenario. Though this forms the advantage of the present study there are other confounding factors while using breath analyzer. Eating/drinking /smoking 20 minutes prior to testing, low-calorie diet/ diabetic ketoacidosis, measuring too early in the expiratory phase, shallow expiration or hyperventilation or holding breath, pulmonary disease can all cause inaccurate readings.¹⁷⁻²⁰ Though there may be many adverse factors involved in bringing down the accuracy of breath test, a gross idea of the level of intoxication might save the patient from unnecessary active interventions like intubation or pitfalls of delaying treatment due to the underestimated severity of TBI.

It should be mentioned that the subjects included might not be representative population of whole of Kerala, as it was carried out in a single centre. Therefore multi-centric trials are recommended for more accurate prediction of effect of alcohol on GCS in Traumatic Brain injury patient thereby involving larger population to improve accuracy. Confounders of GCS also form another limitation of the study. The major drawback of GCS is that it is not originally intended to be converted into a single score- the components (E4, V5, M6) are more important than the total score. The motor response alone is considered by some to be the best predictor; therefore the same score will predict different TBI mortality depending on the components.^{7,21} The other disadvantages of GCS include non-incorporation of brain stem reflexes, unreliability in patients in middle range GCS 9-12, poor inter-observer reliability and reproducibility.²²⁻²⁴

CONCLUSION

Despite its drawbacks, the GCS remains the most universally utilized level of consciousness scale worldwide. Though some of the more detailed scales may be more suitable for assessment of inpatients, the GCS, by virtue of its simplicity, seems destined to be used in emergency medicine. In the light of the present study it is

concluded that even in the setting of alcohol intoxicated TBI patient, Glasgow coma score is a useful tool for quick decision making in emergency department.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Global burden of disease study 2013 collaborators. global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990-2013: a systematic analysis for the global burden of disease study. *Lancet Lond Engl.* 2015;386(9995):743-800.
2. Massenburg BB, Veetil DK, Raykar NP, Agrawal A, Roy N, Gerdin M. A systematic review of quantitative research on traumatic brain injury in India. *Neurol India.* 2017;65(2):305-14.
3. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. *Lancet.* 1974;2:81-4.
4. Rundheug NP, Moen KG, Skandsen T, Mikalsen KS, Lund SB, Hara S, Vik A. Moderate and severe traumatic brain injury: effect of blood alcohol concentration on GCS and relation to computed tomography findings. *J Neurosurg.* 2015;122:211-8.
5. Jones AW. Medicolegal alcohol determination blood or breath alcohol concentration. *Forensic Sci Rev.* 2000;12(1-2):23-47.
6. Lange RT, Iverson GL, Brubacher JR, Franzen MD. Effect of blood alcohol level on Glasgow Coma Scale scores following traumatic brain injury. *Brain Inj.* 2010;24(7):919-27.
7. Teasdale G, Jennett B, Murray L, Murray G. Glasgow coma scale: to sum or not to sum. *Lancet Lond Engl.* 1983;2(8351):678.
8. Stålhammar D, Starmark JE. Assessment of responsiveness in head injury patients. The Glasgow Coma Scale and some comments on alternative methods. *Acta Neurochir Suppl (Wien).* 1986;36:91-4.
9. Shibahashi K, Sugiyama K, Okura Y, Hoda H, Hamabe Y. Multicenter retrospective cohort study of "talk and die" after traumatic brain injury. *World Neurosurg.* 2017;107:82-6.
10. Peterson EC, Chesnut RM. Talk and die revisited: bifrontal contusions and late deterioration. *J Trauma.* 2011;71(6):1588-92.
11. Goldschlager T, Rosenfeld JV, Winter CD. Talk and die patients presenting to a major trauma centre over a 10 year period: a critical review. *J Clin Neurosci J Neurosurg Soc Australas.* 2007;14(7):618-23.
12. Stuke L, Arrastia R, Gentilello LM, Shafi S. Effect of alcohol on glasgow coma scale in head-injured patients. *Ann Surg.* 2007;245(4):651-5.
13. Sperry JL, Gentilello LM, Minei JP, Diaz-Arrastia RR, Friese RS, Shafi S. Waiting for the patient to "sober up": Effect of alcohol intoxication on glasgow coma scale score of brain injured patients. *J Trauma.* 2006;61(6):1305-11.
14. Rønning P, Gunstad PO, Skaga N-O, Langmoen IA, Stavem K, Helseth E. The impact of blood ethanol concentration on the classification of head injury severity in traumatic brain injury. *Brain Inj.* 2015;29(13-14):1648-53.
15. Shahin H, Gopinath SP, Robertson CS. Influence of alcohol on early Glasgow Coma Scale in head-injured patients. *J Trauma.* 2010;69(5):1176-81.
16. Kelly DF. Alcohol and head injury: an issue revisited. *J Neurotrauma.* 1995;12(5):883-90.
17. Andersson A, Hök B, Ekström M, Hedenstierna G. Influence from breathing pattern on alcohol and tracer gas expirograms--implications for alcohol use. *Forensic Sci Int.* 2011;206(1):52-7.
18. Grubb D, Rasmussen B, Linnet K, Olsson SG, Lindberg L. Breath alcohol analysis incorporating standardization to water vapour is as precise as blood alcohol analysis. *Forensic Sci Int.* 2012;216(1):88-91.
19. Hlastala MP, Anderson JC. The impact of breathing pattern and lung size on the alcohol breath test. *Ann Biomed Eng.* 2007;35(2):264-72.
20. Sadler DW, Fox J. Intra-individual and inter-individual variation in breath alcohol pharmacokinetics: The effect of food on absorption. *Sci Justice J Forensic Sci Soc.* 2011;51(1):3-9.
21. Healey C, Osler TM, Rogers FB, Healey MA, Gance LG, Kilgo PD. Improving the Glasgow coma scale score: motor score alone is a better predictor. *J Trauma.* 2003;54:671-80.
22. Segatore M, Way C. The Glasgow Coma Scale: time for change. *Heart Lung J Crit Care.* 1992;21(6):548-57.
23. Sternbach GL. The Glasgow coma scale. *J Emerg Med.* 2000;19(1):67-71.
24. Zuercher M, Ummenhofer W, Baltussen A, Walder B. The use of Glasgow coma scale in injury assessment: a critical review. *Brain Inj.* 2009;23(5):371-84.

Cite this article as: Thomas M, Suseel A, David SS. Effect of alcohol on Glasgow Coma Score in traumatic brain injury: a single, center, cross-sectional study. *Int J Res Med Sci* 2023;11:1735-40.