

## ORIGINAL ARTICLE

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## Predicting outcomes 3 months after traumatic brain injury in patients admitted to emergency department

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### ABSTRACT

#### BACKGROUND

Traumatic brain injury (TBI) is among the leading causes of mortality and long-term disability. Prognosis assessment is a primary factor of clinical decision-making by emergency physicians. This study aimed to investigate the prognostic factors of TBI in the patients admitted to a typical emergency department.

#### METHODS

This prospective cohort study was conducted involving 100 TBI patients. Baseline characteristics, including age, gender, Glasgow Coma Scale (GCS), and vital signs were recorded. Blood tests and brain CT scans were collected. The patients were followed-up three months after the date of admission. The observed outcomes were categorized as recovery without complication, recovery with complication, or death. Statistical analysis was performed using the simple and multivariate binary logistic regression in the software IBM SPSS version 19.

#### RESULTS

The most common brain CT scan findings were subarachnoid hemorrhage (21.0%) and epidural hemorrhage (20.0%). In the follow-up performed three months after the admission, 47 patients (47.0%) had died, 39 (39.0%) were suffering from complications, and 14 patients (14.0%) were recovering without complications. Simple binary logistic regression showed that older age (OR=3.28, 95% C.I.=1.27-8.41), minor/moderate head trauma (OR=13.93, 95% C.I.=1.73-112.11), severe head trauma (OR=54.40, 95% C.I.=5.71-517.56) and presence of deep skull fracture (OR=8.92, 95% C.I.=1.04-75.53) were statistically significant predictors of mortality. Multivariate logistic regression showed that mortality chance was higher in elderly (OR=7.45, 95% C.I.=2.02-27.36), minor/moderate head trauma (OR=26.87, 95% C.I.=2.42-298.25) and severe head trauma (OR=127.97, 95% C.I.=9.11-1796.28).

#### CONCLUSION

This study demonstrated that severe head trauma was the most predicted risk factor of poorer clinical outcomes after TBI.

**Keywords:** Traumatic brain injury, prognosis, emergency, mortality, trauma

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## INTRODUCTION

Traumatic brain injury (TBI) is one of the leading causes of mortality and long-term disability, especially among young people, and accounts for about 30% of accident-related deaths.<sup>(1,2)</sup> Every year, about 2.5 million people sustain TBI, of which about 50,000 die and more than 80,000 become permanently disabled.<sup>(2)</sup> In Iran, trauma, and especially TBI, is one of the leading causes of death following a hospital admission.<sup>(3,4)</sup> Because of its severe physiological, emotional, and physical complications, this injury imposes significant direct and indirect financial burdens that are hard to bear, especially in low- and middle-income countries, where more than 90% of TBI cases occur.<sup>(1,5)</sup> In the last decade, the hospitalization rate has shown no notable change and the mortality rate has declined, but there has been an increase in the number of TBI-related admissions to Emergency Departments (ED).<sup>(6)</sup> This trend calls for better management strategies for predicting, preventing, and addressing the short- and long-term complications of TBI.

Prognosis assessment is one of the primary factors of clinical decision-making and selection of therapeutic strategy for TBI patients, especially in emergency departments.<sup>(7,8)</sup> Studies have shown that more than two-thirds of physicians consider prognosis as the most important step in making a clinical decision particularly about ventilation, use of analgesics, or serum therapy.<sup>(4)</sup> In addition to helping physicians decide on the type and strength of treatment, having a confident prognosis is very important for answering the questions of patient's family members and companions about his or her condition.

In recent decades, researchers have proposed several models, including the Glasgow Outcome Scale (GOS) and its extended version Glasgow Outcome Scale Extended (GOSE), Rancho Los Amigos Scale Revised (RLAS-R),

and Clinical Randomization of an Antiúbrinolytic in Significant Hemorrhage (complete CRASH-2), for predicting the outcomes of TBI patients.<sup>(2,5)</sup> In these models, factors such as age, Glasgow Coma Score (GCS), hypoxia and hypotension upon arrival to the ED, cause of the accident, biochemical parameters, and CT-scan findings have been used as the determinants of prognosis.<sup>(9-11)</sup> However, considering the differences in the services of EDs, epidemiological patterns of TBIs, driving safety levels, and the demographics of TBI patients (e.g. age and gender) in different parts of the world, these models may not be uniformly applicable to all countries.<sup>(11,12)</sup> In Iran, which has a particularly high rate of traffic and occupational accidents, only one retrospective study has investigated the prognostic factors of TBI,<sup>(3)</sup> therefore, more prospective studies are needed to develop a localized model for predicting the outcomes of TBI for Iranian patients.

Although GCS is a useful tool for TBI evaluation, it has some limitations and GCS documentation have some differences and pitfalls in patients.<sup>(13,14)</sup>

Generally, in GCS level documentation, GCS in admission to ED and highest or lowest GCS in 24 hours of admission can be used. Moreover, some interventions that can change the GCS level are medical intervention, administration of drugs such as sedatives or paralytic drugs, drug toxicity, and facial trauma.<sup>(15,16)</sup> The objective of this study was to investigate the prognostic factors of TBI in the patients admitted to a typical emergency department.

## METHODS

### Research design

This prospective cohort study was conducted on TBI patients admitted to the Emergency Department of Hasheminejad Hospital, Mashhad, Iran from January 2015 to December 2016.

### Research subjects

Patients with moderate or severe TBI and age of more than 15 years were included in the study. Exclusion criteria were (i) simultaneous trauma in other organs, (ii) discharge from the emergency department before the final evaluation, and (iii) lack of cooperation in subsequent follow-ups. The sample size was calculated based on the results of Yang et al.<sup>(17)</sup> using the formula for comparison of a quantitative variable in two groups, with  $\alpha$  and  $\beta$  set to 0.05 and 0.2 respectively. Accordingly, the minimum sample size was calculated as  $n=138$ . Patients were entered into the study consecutively until the target sample size was reached.

### Data collection

An emergency medicine specialist recorded the basic information of patients including age, gender, GCS, and vital signs, upon their admission to the ED. Blood pressure and oxygen saturation were measured by a digital pressure meter and a pulse oximeter respectively. Mean arterial pressure (MAP) was calculated using the formula  $[(2 \times \text{diastolic blood pressure}) + \text{systolic blood pressure}] / 3$ . Hypotension was defined as a systolic pressure of less than 70mm Hg, and hypoxia was defined as an oxygen saturation of less than 90 percent. For all patients, arterial blood samples were taken to measure arterial blood gas and venous blood samples were taken to measure hematocrit (HCT). The normal range of pH was 7.35-7.45. Acidemia was defined as a serum pH of less than 7.35, and alkalemia as  $\text{pH} > 7.45$ . The normal range of HCT was 38-42% and anemia was defined as hematocrit of less than 42% for men and less than 38% for women. The normal range of  $\text{HCO}_3^-$  was defined as 22 to 28 milliequivalents per liter; metabolic acidosis as  $\text{HCO}_3^- < 22$  milliequivalents per liter; metabolic alkalosis as  $\text{HCO}_3^- > 28$  milliequivalents per liter. The normal range of  $\text{PCO}_2$  was defined as 35–45 mmHg; respiratory acidosis as  $\text{PCO}_2 > 45$  mmHg; and respiratory alkalosis as  $\text{PCO}_2 < 35$  mmHg and pH 7.35 - 7.45. Vital signs were categorized as

follows: bradycardia:  $\text{HR} < 60$ ; normal HR:  $0 \leq \text{HR} \leq 100$ ; tachycardia:  $\text{HR} > 100$ ; bradypnea:  $\text{RR} < 12$ ; normal:  $12 \leq \text{RR} \leq 20$ ; tachypnea:  $\text{RR} > 20$ ; normal GCS=15; minor or moderate head trauma  $8 \leq \text{GCS} < 15$ ; severe head trauma  $\text{GCS} < 8$ ; children: age  $< 16$  years; adults:  $16 \leq \text{age} < 65$  years; elderly: age  $\geq 65$  years. A brain CT scan with an axial cross section was taken on admission to the ED (less than 6 hours after trauma) and its result was interpreted by a radiologist.

### Clinical outcomes

Follow-up was performed three months after admission to the ED and the outcome at that date was categorized as (i) recovery without complications, (ii) recovery with complications (emergence of new neurological symptoms after TBI), or (iii) death.

### Ethical considerations

The research plan was fully explained to the patient's legal guardian and written informed consent was acquired before collecting any information. Personal information of all patients remained confidential and was handled and processed in a codified form. The research proposal was authorized by the Research Deputy Department of the Mashhad University of Medical Sciences and received approval from the Ethics Committee of this university (registration code: 920748).

### Data analysis

Descriptive statistical analyses were performed using SPSS for Windows, version 19 (Armonk, NY: IBM Corp.). Simple binary logistic regression was used to find predictor variables for mortality. Then all predictors that were significant were entered into a multivariate logistic regression. Findings were represented as odds ratio (OR) alongside with 95% confidence interval (CI). In all tests, p-values of less than 5% were considered statistically significant. Using the per-protocol analytical approach, the analyses were restricted to those

patients whose information up to the final stage of follow-up was available.

## RESULTS

### Baseline characteristics

A total of 138 patients entered the study, of which 38 patients were excluded because of simultaneous trauma in other organs (31 patients), discharge from the emergency department before the final evaluation (4 patients), and lack of cooperation during the follow-up (3 patients). Ultimately, the study was performed on 100 patients (Figure 1).

Participants (n = 100) were predominately male (85%) with a mean age of  $43.7 \pm 23.0$  years. In 76.0% of patients, GCS on admission was more than 8. The most common disorders of vital signs were tachycardia (67.0%) and hypotension (42.0%). The most common brain CT scan findings were subarachnoid hemorrhage (21.0%) and epidural hemorrhage (20.0%). In the follow-up performed three months after admission, 47 patients (47.0%) had died, 39 (39.0%) were suffering from complications, and 14 patients (14.0%) were recovering without complications. Other results regarding the background variables, physical examination findings, vital signs, and paraclinical findings are presented in Table 1.

### Variables associated with the outcome (death or survival)

Simple binary logistic regression showed that older age group (OR=3.28, 95% C.I.= 1.27-8.41), minor/moderate head trauma (OR=13.93, 95% C.I.=1.73-112.11), severe head trauma (OR=54.40, 95% C.I.=5.71-517.66) and presence of deep skull fracture (OR=8.92, 95% C.I.=1.04-75.53) were statistically significant predictors of mortality (Table 2). Cox and Snell R square for multivariate binary logistic regression was 37% showing an acceptable overall prediction. In this model, only severe head trauma, minor/moderate head trauma and elderly age group remained statistically significant predictors (Table 3).

## DISCUSSION

The findings of this study showed that older age, minor/moderate head trauma, and severe head trauma on admission were the factors associated with the death of these patients.

The first factor that this study found to be related to the outcome of death following a TBI was age. The mean age of the subjects of our study was about 44 years. On average, patient who died during the follow-up period were about 10 years older than those who survived this period with or without complications. The presence of

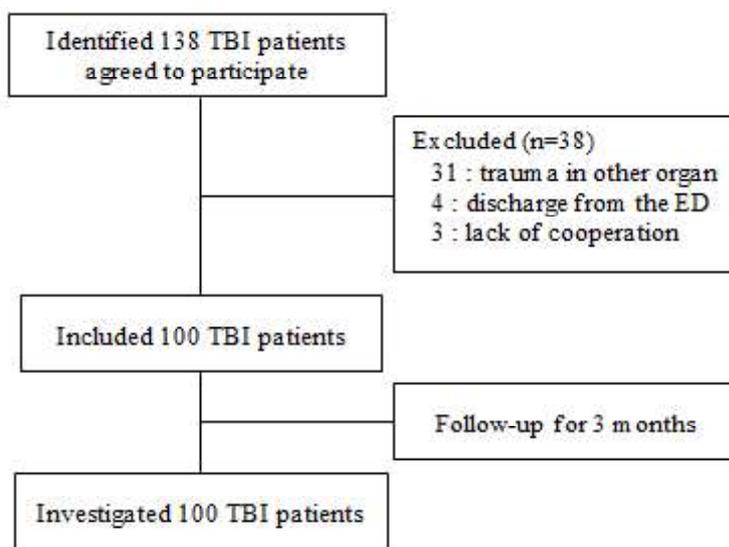


Figure 1. Flow diagram of traumatic brain injury

Table 1. Distribution of demographic, clinical findings and outcomes of TBI subjects (n=100)

Variable	n (%)
Age	
Children	67 (67.0)
Adults	6 (6.0)
Elderly	27 (27.0)
Gender	
Male	85 (85.0)
Female	15 (15.0)
O2 saturation	
Normal	90 (93.8)
Hypoxia	6 (6.3)
RR	
Normal	88 (92.6)
Bradypnea	3 (3.2)
Tachypnea	4 (4.2)
HR	
Normal	83 (85.6)
Bradycardia	2 (2.1)
Tachycardia	12 (12.4)
MAP	
Normal	87 (90.6)
Hypotension	9 (9.4)
Head trauma	
Normal	17 (17.3)
Minor/ moderate	59 (60.2)
Severe	22 (22.4)
HCO <sub>3</sub>	
Normal	41 (42.3)
Metabolic acidosis	48 (49.4)
Metabolic alkalosis	8 (8.2)
pCO <sub>2</sub>	
Normal	48 (49.5)
Respiratory acidosis	18 (18.5)
Respiratory alkalosis	31 (31.9)
pH	
Normal	46 (47.4)
Acidemia	38 (39.1)
Alkalemia	13 (13.4)
HCT	
Normal	14 (19.4)
Anemia	58 (80.6)
Subdural hemorrhage	
Yes	18 (18.0)
No	82 (82.0)
Subarachnoid hemorrhage	
Yes	24 (24.0)
No	76 (76.0)
Epidural hemorrhage	
Yes	21 (21.0)
No	79 (79.0)
Depressed skull fracture	
Yes	8 (8.0)
No	92 (92.0)
Intracranial hemorrhage	
Yes	10 (10.0)
No	90 (90.0)
Pneumocephalus	
Yes	4 (4.0)
No	96 (96.0)
Contusion	

# Normal GCS=15; minor or moderate head trauma 8≤GCS<15; severe head trauma GCS<8

similar reports in other studies suggests that age is likely to be a key factor of the clinical outcome of TBI, and younger people have a lower chance of mortality following a TBI.<sup>(3,10)</sup> It has also been reported that each year of increase in age is associated with 5% higher chance of post-concussion syndrome and 2% higher chance of a GCS of less than 7.<sup>(10,18,19)</sup>

Both quantitative and qualitative analyses of our study showed that reduced GCS is another important factor in the adverse clinical outcomes of TBI. The frequency of GCS of less than 8 among the patients who did not survive the follow-up period was significantly higher than among other patients. Other studies have also reported an association between GCS and the prognosis of TBI patients.<sup>(20,21)</sup> Although our finding in regard to the association of GCS with the clinical outcome of TBI is not new and has been previously mentioned in other studies,<sup>(9-11)</sup> this is the first prospective study that reports such an association in Iran, although a cross-sectional study in Iran found age, gender, GCS, pupillary reflex, hypernatremia, and increased intracranial pressure (IICP) as the predictors of death in severe TBI.<sup>(3)</sup>

Among the vital signs, only the heart rate exhibited an association with undesirable clinical outcomes. However, this association was only observed in quantitative analyses (there was no relationship between tachycardia and outcome in the qualitative analysis). Since tachycardia, as the prominent sign of hemodynamic instability, can be caused by other underlying disorders such as cerebral hemorrhage, its association with the adverse clinical outcomes in TBI patients can be explained from this perspective. While primary vital signs are rare among the main factors of prognostic models, our findings suggest that vital signs can serve as a prognostic factor for the assessment of the clinical outcome of TBI. However, it should be noted that the number of studies in this area is still very low to draw a definite conclusion.

The last factor that our study found to be associated with the final condition of TBI

Table 2. Simple binary logistic regression to find predicting factors of mortality

	OR (95%CI)	p value
Age		
Children	3.28(0.55-19.23)	0.188
Adults	1	
Elderly	3.28(1.27-8.41)	0.013
Gender		
Male	1	
Female	0.69(0.22-2.13)	0.530
O2 saturation		
Normal	1	
Hypoxia	2.45(0.42-14.07)	0.315
RR		
Normal	1	
Bradypnea	2.46(0.21-28.16)	0.469
Tachypnea	1.23(0.16-8.14)	0.839
HR		
Normal	1	
Bradycardia	-	0.999
Tachycardia	2.43(0.67-8.71)	0.172
MAP		
Normal	1	
Hypotension	1.95(0.44-8.70)	0.377
Head trauma		
Normal	1	
Minor/moderate head trauma	13.93(1.73-112.11)	0.013
Severe head trauma	54.40(5.71-517.65)	0.001
HCO3		
Normal	1	
Acidemia	1.61(0.70-3.66)	0.255
Alkalemia		
pCO2		
Normal	1	
Acidosis	1.28(0.57-2.87)	0.540
pH		
Normal	1	
Acidemia	0.92(0.41-2.07)	0.858
HCT		
Normal	1	
Anemia	1.23(0.38-3.96)	0.728
SDH		
No	1	
Yes	1.96(0.69-5.58)	0.205
SAH		
No	1	
Yes	1.60(0.62-4.11)	0.323
EDH		
No	1	
Yes	0.52(0.18-1.45)	0.215
DSF		
No	1	
Yes	8.92(1.04-75.53)	0.045
Pneumocephalus		
No	1	
Yes	0.35(0.0-3.53)	0.377
Contusion		
No	1	
Yes	1.70(0.27-10.67)	0.569

OR: Odds Ratio; AOR: Adjusted Odds Ratio; RR: respiratory rate; HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; GCS: Glasgow coma scale; HCT: hematocrit; SDH: subdural hemorrhage; SAH: subarachnoid hemorrhage; EDH: epidural hemorrhage; DSF: deep skull fracture

Table 3. Multivariate binary logistic regression to find predicting factors of mortality

	AOR (95%CI)	p value
Age		
Children	0.48(0.007-32.29)	0.735
Adults		
Elderly	7.45(2.02-27.36)	0.002
HR		
Normal	1	
Bradycardia		
Tachycardia	2.51(0.42-15.01)	0.311
Head trauma		
Normal	1	
Minor/moderate head trauma	26.87(2.42-298.25)	0.007
Severe head trauma	127.97(9.11-1796.28)	0.001
SDH		
No	1	
Yes	1.86(0.49-6.98)	0.353
DSF		
No	1	
Yes	28.71(0.99-835.41)	0.051

AOR: Adjusted Odds Ratio; HR: heart rate; SDH: subdural hemorrhage; DSF: deep skull fracture

patients was the findings of cerebral CT scan taken on admission. Analysis of the CT scan findings suggests that non-surviving patients had a higher frequency of subdural hematoma (SDH), intracranial hemorrhage (ICH), depressed skull fracture (DSF), and cerebral contusion, while the patients who recovered with complications had a higher frequency of epidural hematoma (EDH). One of the most common consequences of brain trauma is extra-axial hemorrhages, which are categorized as epidural and subdural hemorrhages and subarachnoid hemorrhage (SAH). It has been reported that 58% of all patients undergoing hematoma removal and 21% of all patients with severe head trauma have subdural hematomas.<sup>(22)</sup> The prevalence of subdural hematoma among our subjects was close to the figures reported in other studies, which suggests that the pattern of brain pathologies following TBI is likely to be independent of the country.<sup>(9,11)</sup> The association of the discovery of a lesion in CT scan with the outcome of treatment has been already reported in other studies and our results further support these reports.<sup>(10,22,23)</sup>

In the present study, which was conducted on a reasonably large-sized sample, there were

significantly more (almost five times more) male TBI patients than female TBI patients. This difference in the gender distribution can be attributed to the men's riskier social activities and occupations.<sup>(24)</sup> This finding is consistent with the majority of other studies, which report that men are more likely to sustain TBI than women, although the reported male to female ratio varies between 1.5 and 4.<sup>(8,12,25)</sup> Interestingly, the other study conducted on TBI in Iranians reported that its subjects were 85% men and 15% women, figures that are identical to the gender ratio of our patients.<sup>(3)</sup> However, our comparison of clinical outcomes of TBI shows that gender has no effect on the incidence of death following a TBI. Considering that several other studies have reported a similar conclusion,<sup>(3,7,26)</sup> it can be claimed with sufficient confidence that gender has no impact on the outcome of TBI.

Our study showed that hypoxemia was not associated with the clinical outcome of TBI. However, we believe that this lack of significant association is purely statistical because while 34% of the patients in the non-surviving group had hypoxia, 30% of the patients who recovered with complications and only 7% of those who recovered without complications had hypoxia,

which seems clinically significant. In this regard, a study in the United States has shown that brain hypoxia exacerbates the short-term outcome of TBI independent from the raise in intracranial pressure. In contrast, Manley et al.,<sup>(21)</sup> like us, found that hypoxia is not associated with increased mortality in the early stages of recovery. However, we found that factors other than hypoxia, namely acidosis and anemia also lack any effect on the outcome of TBI, a claim that have many opponents and proponents in the literature. For example, while one study has reported that anemia with cerebral hypoxia is a prognostic factor for negative outcome,<sup>(19)</sup> another study has found that elevation of hematocrit above 28% is not associated with reduced mortality from head trauma;<sup>(26)</sup> moreover, another study in Taiwan<sup>(20)</sup> has shown that initial anemia does not increase the risk of mortality in TBI patients.

The present study suffers from several limitations. First, this study did not examine the parameters such as the type of accident leading to TBI, the time between accident and admission, pupil reflex condition, extracranial damage, and patient's economic status, whereas many studies have claimed that these parameters play a significant role in predicting the clinical outcomes of TBI patients. In addition, the follow-up of this study was performed via phone calls, and this may have led to a higher number of patients being categorized as recovering with complications, because TBI-related complications were recorded as stated by the patients or their family members. We recommend in-depth management of patients with a GCS of less than 8 presenting to the emergency department.

## CONCLUSION

Our study demonstrated that severe head trauma in the initial examinations are associated with poorer clinical outcomes after TBI. Considering the factors identified to be associated with the outcomes of TBI among Iranians, emergency departments are recommended to

give a higher priority to the older TBI patients with lower GCS and unstable hemodynamics.

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## CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest regarding the publication of this manuscript.

## AUTHOR CONTRIBUTIONS

All authors contributed to compilation of the subject matter, writing of the manuscript drafts, data collection, and data analysis. HFD and MA contributed to manuscript finalization. YR and MF contributed to the study concept and design. EB, MF, and FR contributed to manuscript revision. All authors have read and approved the final manuscript. 

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