Factors Affecting Mortality in Severe Traumatic Brain Injury

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Objective: To evaluate the factors affecting mortality in severe traumatic brain injury patients.

Design: A Retrospective Study.

Setting: Bahrain Defence Force Hospital, Bahrain.

Method: All patients admitted to the Intensive Care Unit (ICU) from 1 January 2010 to 31 December 2015 were included in the study. The following data were documented: age, gender, mechanism of injury, type of brain injury, surgical intervention as craniotomy for decompression or evacuation of intracranial hematoma and the prognosis.

Result: One hundred and five patients who were admitted to ICU from 1 January 2010 to 31 December 2015 were included in the study. Glasgow Coma Scale (GCS) (\leq 8) and Subdural Hematoma (SDH) were identified as risk factors for mortality. Females' traumatic brain injury was associated with higher risk of mortality compared to males. Skull fracture has 1.58 times risk mortality. Age is not a predictive factor.

Conclusion: Patients who present with a low Glasgow Coma Scale or with a Subdural Hematoma are at a higher risk of mortality.

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Severe traumatic brain injury (TBI) is defined as patients with a Glasgow Coma Scale of 3 to 8. It is considered one of the major public health hazards throughout the world with a socioeconomic burden. The incidence worldwide is rising, mainly due to the increase in motor vehicle collisions, especially in middle to low-income countries¹.

In 2010, the number of patients who sustained TBI was approximately 2.5 million. It is considered as one of the contributing factors to injury-related deaths^{2,3}. Data from the CDC indicates that each year in the USA, 1.7 million people sustain TBI⁴.

In Bahrain, the implementation of new traffic laws and restrictions in 2015 might decrease the rate of motor vehicle collisions.

The aim of this study is to evaluate the factors affecting mortality in severe traumatic brain injury patients.

METHOD

All patients admitted to the Intensive Care Unit from 1 January 2010 to 31 December 2015 were included in the study. Patients with GCS ≤ 8 associated with head trauma on presentation were included. Patients with GCS ≥ 9 were excluded as well as patients who were declared dead in the trauma bay.

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The following data were documented: age, mechanism of head injury, type of brain injury such as epidural or subdural hematoma (EDH or SDH), intraventricular hemorrhage (IVH), intracranial hemorrhage, subarachnoid hemorrhage (SAH), brain contusion or brain edema and diffuse axonal injury, surgical intervention as craniotomy for decompression or evacuation of intracranial hematoma.

Statistical data was analyzed using SPSS software version 19.0.

RESULTS

One hundred five patients were included in the study from 2010 to 2016; the mortality ranged from 2% to 4%, see table 1 and figure 1.

Table	1:	Patients	and	Mortality
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Year	Number of Patients	Mortality
2010 - 2011	16 (15.2%)	2 (1.9%)
2011 - 2012	18 (17.1%)	4 (3.8%)
2012 - 2013	19 (18.1%)	4 (3.8%)
2013 - 2014	13 (12.4%)	4 (3.8%)
2014 - 2015	21 (20%)	6 (5.7%)
2015 - 2016	18 (17.1%)	4 (3.8%)



Figure 1: Mortality Rate

The majority of TBI patients were males, 94 (89.5%); the age ranged from 20 to 50; the majority of the mechanism of injury was Motor Vehicle Collision (MVC), see tables 2 to 4 and figure 2.

Table 2: Gender Distribution

Gender	Number of Patients
Female	11 (10.5%)
Male	94 (89.5%)

Table 3: Age Distribution

Age (Years)	Number of Patients
0-20	35 (33.3%)
21-50	63 (60%)
>50	7 (6.7%)

Table 4: Mechanism of Injury

Mechanism of Trauma	Number of Patients		
Assault	8 (7.6%)		
Fall	22 (20.9%)		
Motor Vehicle Collision (MVC)	75 (71.4%)		



Figure 2: Mechanism of Injury

CT Brain findings revealed that most patients had skull fractures, Subarachnoid Hemorrhage and edema; craniotomy was performed in 36 patients, see table 5, figure 3 and table 6.

Table 5: CT Brain Findings

CT Finding	Number of Patients			
Skull Fracture				
No	64 (60.9%)			
Yes	41 (39%)			
Epidural Hematoma				
No	100 (95.2%)			
Yes	5 (4.8%)			
Subdural Hematoma				
No	71 (67.6%)			
Yes	34 (32.4%)			
Subarachnoid Hemorrhage				
No	65 (61.9%)			
Yes	40 (38.1%)			
Contusion				
No	67 (63.8%)			
Yes	38 (36.2%)			
Edema				
No	62 (59.1%)			
Yes	43 (40.9%)			
Intraventricular Hemorrhage				
No	94 (89.5%)			
Yes	11 (10.5%)			
Intracerebral Hemorrhage (ICH)				
No	99 (94.3%)			
Yes	6 (5.7%)			
Axonal Injury				
No	102 (97.1%)			
Yes	3 (2.9%)			



Figure 3: CT Brain Findings

Table 6: Intervention Required

Intervention Required	Number of Patients
No	69 (65.7%)
Yes (Craniotomy)	36 (34.3%)

Table 7 reveals the following: gender, age range, GCS severity, mechanism of injury, skull fracture, epidural hematoma, subdural hematoma, contusion, edema, intraventricular hemorrhage, intracerebral hemorrhage and axonal injury.

Table 7: Personal and Clinical Characteristics andTraumatic Brain Injury Outcome

	Alive	Dead	P-value			
Gender						
Female	7 (6.7%)	4 (3.8%)	0.252			
Male	75 (71.4%)	19 (18%)				
Age						
0-20	29 (27.6%)	6 (5.7%)				
21-50	49 (46.7%)	14 (13.3%)	0.283			
>50	4 (3.8%)	3 (2.9%)				
Glasgow Coma Score						
Moderate	3 (2.9%)	0	>0.05			
Severe	79 (75.2%)	23 (21.9%)				
Mechanism						
Assault	5 (4.8%)	3 (2.9%)				
Fall	16 (15.2%)	6 (5.7%)	0.324			
MVC	61 (58.1%)	14 (13.3%)				
Skull Fracture		`,				
No	53 (50.5%)	11 (10.5%)				
Yes	29 (27.6%)	12 (11.4%)	0.156			
Epidural Hematoma						
No	77 (73.3%)	23 (21.9%)				
Yes	5 (4.8%)	0				
Subdural Hematoma						
No	62 (59%)	9 (8.6%)	0.001*			
Yes	20 (19%)	14 (13.3%)				
Subarachnoid Hemor	rhage					
No	51 (48.6%)	14 (13.3%)	>0.05			
Yes	31 (29.5%)	9 (8.6%)				
Contusion						
No	51 (48.6%)	16 (15.2%)	0.627			
Yes	31 (29.5%)	7 (6.7%)				
Edema						
No	50 (47.6%)	12 (11.4%)				
Yes	32 (30.5%)	11 (10.5%)	0.479			
Intraventricular Hem	Intraventricular Hemorrhage					
No	74 (40.5%)	20 (19%)				
Yes	8 (7.6%)	3 (2.9%)	0.702			
Intracerebral Hemor	rhage					
No	77 (73.3%)	22 (20.9%)				
Yes	5 (4.8%) 1 (0.95%		>0.05			
Axonal						
No	82 (78%)	20 (19%)	0.009*			
Yes	0	3 (2.9%)				

*P-value<0.05 was considered statistically significant

The most important risk factors for mortality were GCS and Subdual Hematoma, see table 8.

 Table 8: Factors Predicting Mortality in Severe Traumatic

 Brain Injury

Variable	OR	95% CI		D voluo
variable		Lower	Upper	r-value
Gender	2.277	0.255	20.3	0.461
Age	1.007	0.964	1.052	0.744
GCS	0.398	0.221	0.716	0.002*
Mechanism of Injury				0.863
Assault versus Fall	1.878	0.193	18.286	0.587
Assault versus MVC	1.532	0.181	12.995	0.696
CT Brain Findings				
Skull Fracture	1.584	0.377	6.651	0.53
SDH	10.996	1.753	68.974	0.01*
SAH	2.023	0.469	8.724	0.345
Contusion	2.617	0.548	12.499	0.228
Edema	2.333	0.586	9.29	0.229
IVH	3.704	0.424	32.332	0.236
ICH	0.229	0.01	5.282	0.357

*P<0.05 was considered statistically significant

DISCUSSION

This study revealed that GCS and Subdual Hematoma were potential risk factors for mortality.

Classification of the severity of TBI is necessary in the management because many patients with severe TBI have already presented with GCS ≤ 8 , requiring sedation and intubation. Therefore, it is difficult to utilize GCS in such cases; CT scan is mandatory in such cases.

The most widely used methods for CT classification of TBI are the Marshal Score and Rotterdam Score^{7,8}. The Marshall scoring system divides patients into six categories of increasing severity based on CT brain findings. It focuses mainly on the degree of swelling (midline shift, basal cistern compression) and the presence of contusions/hemorrhage; patients with a higher score have a higher risk for mortality. The Rotterdam score added more variables to their classification which were categorized as subarachnoid/intraventricular hemorrhage, extradural hematoma and the extent of basal cistern compression.

A comparison of both systems showed that the Marshall CT classification had a higher predictive value, whereas the Rotterdam score incorporated more variables into their classification which may be a preferable scoring method for patients with diffuse injury^{10,11}.

There are a number of limitations in our study. Many of the patients that present with TBI have other injuries which could increase their risk of mortality. The classification of CT brain findings was limited to the presence of injury, not the extent or size of the lesion. The presence of midline shift and compression of the basal cisterns were not included in our study, which would be helpful compared to other studies.

CONCLUSION

The most predictive factors for mortality in severe TBI are low GCS on presentation and the finding of SDH on CT scan.

Further study is advised to combine CT characteristics for prognosis of patients with severe TBI.

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