

The Epidemiology and Impact of Traumatic Brain Injury in Intensive Care Unit in Baghdad / Iraq

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Abstract Author's Information Background: Traumatic brain injury has been one of the leading causes of 1. M.B.Ch.B morbidity, disability and mortality across all ages. 2. M.B.Ch.B \D.A\ F.I.C.M.S A & IC **Objective:** To determine the patterns of patients with traumatic brain injury and 3. M.B.Ch.B \ F.I.C.M.S A & IC to estimate the population-wide mortality and the contribution of traumatic brain injury to injury-related mortality. *Corresponding author: Patient and method: A retrospective study conducted in two major trauma Dr. Duaa salam atiya, duasalamaa44@gmail.com centers in Baghdad during the period from January 2016 to December 2018. It involved 238 patients who admitted to the traumatic centers when presented with traumatic brain injury at the intensive care unit. Patients with psychiatric illness, seizures, or those who died on arrival to intensive care unit were excluded from the study. Result: In this study, the highest proportion of study patients was discharged Funding information home (64.3%) and 20.2% of them were died. Five factors were found to be Self-funded significant independent risk factors for mortality. These factors were severe GCS (OR= 19.54), hypotension (OR=10.43), respiratory distress (OR= 17.53), skull Conflict of interest fracture (OR= 2.76), and conservative treatment (OR= 20.03). None declared by author **Conclusion:** About one-fifth of causalities died during hospitalization. Each of sever GCS, respiratory distress, hypotension, skull fracture, and conservative treatment are significantly risk factors which increase mortality rate among patients with traumatic brain injury. Received : October, 2022, Keywords: Brain injury, mortality, ICU, risk factors, Iraq. Published: December, 2022

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1. INTRODUCTION

Traumatic brain injury (TBI) is defined as an insult to the brain from an external force that leads to temporary or permanent impairment of cognitive, physical, or psychosocial function (1). It has been one of the leading causes of morbidity, disability and mortality across all ages (2). Globally, more than 50 million individuals suffer from TBIs each year (3). Although estimates across analyses vary, it is generally thought that 75% – 90% of these injuries would be classified as mild. These percentages likely underestimate the total number of mild TBIs since patients do not always present to the emergency department following a mild TBI, with patients following up with general practitioners and others not seeking any care (4). In developing countries, accident rates are increasing as traffic increases, and they greatly exceed those of developed countries. TBI increase is related to many factors including terrorism attacks, uses of not programmed weapon, and lack of road safety (5). In Iraq, the country that fought terrorism on behalf of the world, in the last two decades, the incidence of traumatic head injury increase to peak since the first gulf war (6). Clinical features of TBI include prolonged coma, headache, nausea, aphasia, seizures, amnesia and behavioral abnormalities such as aggression and anxiety, which occur within seconds to minutes after TBI; however, some of these manifestations can persist up to months and years (7). Given that the injuries that lead to TBI are frequently preventable, there is also value in measuring the extent to which different causes of injury lead to TBI to help to understand the effect that injury-prevention programs could have (8). The severity of TBI can be quantified using a variety of measures. The most commonly used assessment scales, the Glasgow Coma Scale (GCS) score, which use clinical assessment to grade the severity of the trauma. The GCS is used to grade TBI as mild, moderate, or severe. The major advantage of the GCS is its simplicity and its usefulness as a standardized measurement that can be used to compare outcomes across a series of patients (9). Over the past 30 years, deaths from severe TBI have reduced from 50% to fewer than 25% (10). TBI may be penetrating or non-penetrating, diffuse or focal, vary in severity, location, and patient characteristics, just to name a few, so this may lead to a problem in the development of reliable guidelines for treatment of TBI. The aim of this study is to determine the patterns of patients with TBI and to estimate the population-wide mortality and the contribution of TBI to injury-related mortality.

2. METHODOLOGY

Study design, setting, and time: This was a retrospective study conducted in two major trauma centers in Baghdad (Baghdad Medical City and Neurosurgical Teaching Hospital) during the period of three years from January 2016 to December 2018. Study Population and sample size: This study involved 238 patients who admitted to the traumatic centers during the years 2016, 2017, and 2018 when presented with TBI at the ICU. Patients with psychiatric illness, seizures, or those who died on arrival to ICU were excluded from the study. The data was collected from the patients' attendance register and transfer/ discharge records. The data extracted for the study included patients' age, gender, etiology, the severity of injury, the outcome of the management in the ICU, and the time of presentation. The severity of TBI was based on an assessment of the patient's consciousness on arrival to ICU, using the Glasgow Coma Scale (GCS) to categorize as was the practice in ICU. GCS 13 – 15 was regarded as mild injury, GCS 9 – 12 as moderate injury, and GCS \leq 8 as severe injuries.

Statistical analysis:

The data analyzed using Statistical Package for Social Sciences (SPSS) version 26. The data presented as mean, standard deviation and ranges. Categorical data presented by frequencies and percentages. Chi square test was used to assess the association between outcome and certain information, while fisher exact test was used instead when the expected frequency was less than 5. Logistic regression analysis applied, using mortality as the dependent variable and the variables that were found significant in the binary analysis were included in the model as the independent variables. A level of P – value less than 0.05 was considered significant.

3. RESULTS

In this study, mean age of the patients was 33.35 ± 12.52 years, ranging from 10 - 68 years. The most affected age group was 21 - 30 years accounting for 35.7% of cases. We noticed that the majority of patients were males (89.1%); 47.1% of them were military. The most common cause of TBI in this study was the blast injury (39.5%) as shown in (**Table 1**). In the current study, 42.9% of patients presented with moderate GCS; 57.1% of them were normotensive; 52.1% complained from respiratory distress; the most common finding by CT scan was Contusion / edema (45%); 34% of them presented with fixed pupil and 39.9% with pupil asymmetry;

posttraumatic epilepsy was presented as local complication in 22.3% of cases; 66% of study patients stayed in hospital for less than month; and 73.1% of them were treated conservatively as shown in (**Table 2**). The highest proportion of study patients was discharged home (64.3%) and 20.2% of them were died as shown in (**Figure 1**). We noticed that the highest prevalence of death was seen significantly among those injured by blast injury (37%, P= 0.001), with severe GCS (57.4%, P= 0.001), with hypotension (52.5%, P= 0.001), with respiratory distress (42.7%, P= 0.001), with skull fracture (36.2%, P= 0.012), those who treated conservatively (62.7%, P= 0.001), and those who had cerebrospinal fluid leak (56.7%, P= 0.001) as shown in (**Table 3**). Logistic regression analysis was applied (Table 4) using death outcome as the dependent variable and the variables that showed significant association in the binary analysis as the independent variables. Five factors were found to be significant independent risk factors for greater likelihood of death. These factors were severe GCS (OR= 19.54), hypotension (OR=10.43), respiratory distress (OR= 17.53), skull fracture (OR= 2.76), and conservative treatment (OR= 20.03). All of these findings are demonstrated in (**Table 4**)

Variable		No.	%
Age (Year)	< 10	17	7.2
	10 - 19	29	12.2
	20 - 29	85	35.7
	30 - 39	64	26.9
	40 - 49	22	9.2
	≥ 50	21	8.8
Gender	Male	212	89.1
	Female	26	10.9
Occupation	Military	112	47.1
	Employee	35	14.7
	Private work	43	18.1
	Other	48	20.2
Causes of TBI	Blast injury	94	39.5
	RTA	61	25.6
	Bullet injury	26	10.9
	Assault	24	10.1
	FFH	22	9.2
	Other	11	4.6

Table 1. Distribution of study patients by certain characteristics (n= 238)

Variable	No.	%	
	Mild	70	29.4
GCS	Moderate	102	42.9
	Severe	66	27.7
	Hypotension	85	35.7
Blood pressure	Normal	136	57.1
	Hypertension	17	7.2
Respiratory distress		124	52.1
	Unremarkable	33	13.9
	Contusion / edema	107	45
CT finding	Extradural hematoma	59	24.8
	Epidural hematoma	29	12.2
	Intracerebral hematoma	10	4.2
	Skull fracture	66	27.7
Comorbidity	Bone fragment	28	11.8
	Mixed	144	60.5
Pupil fixed		81	34
Pupil asymmetry		95	39.9
	Unremarkable	96	40.3
	Posttraumatic epilepsy	53	22.3
Local complication	Cerebrospinal fluid leak	44	18.5
	Meningitis	24	10.1
	Hydrocephalus	21	8.8
Hospital stay (Month)	< 1	157	66
	≥1	81	34
Treatment	Surgery	174	73.1
Treatment	Conservative	64	26.9

Table 2. Clinical Characteristics of the patients at admission (n= 238)

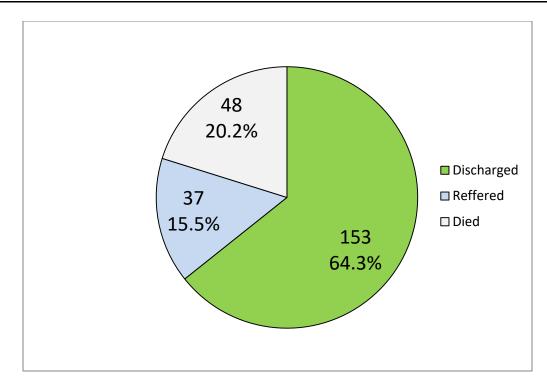


Figure 1. Distribution of study patients by outcome

Variable		Outcome				Total		
		Dead (n= 48)		Discharged (n= 153)		Total (n= 201)		P. value
		No.	%	No.	%	No.	%	
Age (Year)	< 30	33	30.0	77	70.0	110	54.7	
	31 - 49	13	18.8	56	81.2	69	34.3	0.052
	≥ 50	2	9.1	20	90.9	22	10.9	
Gender	Male	41	23.0	137	77.0	178	88.6	0.433
	Female	7	30.4	16	69.6	23	11.4	
Occupation	Military	23	23.2	76	76.8	99	49.3	0.088
	Employee	5	19.2	21	80.8	26	12.9	
	Private work	6	15.0	34	85.0	40	19.9	
	Other	14	38.9	22	61.1	36	17.9	
Causes of TBI	Blast injury	27	37.0	46	63.0	73	36.3	0.047
	RTA	9	15.0	51	85.0	60	29.9	
	Bullet injury	4	18.2	18	81.8	22	10.9	
	Assault	3	17.6	14	72.4	17	8.5	
	FFH	4	20.0	16	80.0	20	10.0	
	Other	1	11.1	8	88.9	9	4.5	
GCS	Mild	4	6.5	58	93.5	62	30.8	0.001
	Moderate	13	15.3	72	84.7	85	42.3	
	Severe	31	57.4	23	42.6	54	26.9	

Table 3. Association between outcome and certain characteristics

Variable		Outcome Dead Discharged		Total				
		(n= 48)		(n= 153)		(n= 201)		P. value
		No.	%	No.	%	No.	%	
Blood	Normal	12	9.6	113	90.4	125	62.2	
pressure	Hypotension	31	52.5	28	47.5	59	29.4	0.001
	Hypertension	5	29.4	12	70.6	17	8.5	
Respiratory	Yes	44	42.7	59	57.3	103	51.2	0.001
distress	No	4	4.1	94	95.9	98	48.8	0.001
CT finding	Unremarkable	6	20.0	24	80.0	30	14.9	
	Contusion / edema	21	22.6	72	77.4	93	46.3	
	Extradural hematoma	11	23.4	36	76.6	47	23.4	0.492
	Epidural hematoma	6	26.1	17	73.9	23	11.4	
	Intracerebral hematoma	4	50.0	4	50.0	8	4.0	
Comorbidity	Skull fracture	21	36.2	37	63.8	58	28.9	
	Bone fragment	6	31.6	13	68.4	19	9.5	0.012
	Mixed	21	16.9	103	83.1	124	61.7	
Pupil fixed	Yes	15	19.5	62	80.5	77	38.3	0.248
	No	33	26.6	91	73.4	124	61.7	
Pupil	Yes	14	17.5	66	82.5	80	39.8	0.084
asymmetry	No	34	28.1	87	71.9	121	60.2	0.084
Treatment	Conservative	37	62.7	22	37.3	59	29.4	0.001
	Surgery	11	7.7	131	92.3	142	70.6	
Local complication	Unremarkable	12	15.4	66	84.6	78	38.8	0.001
	Posttraumatic epilepsy	14	26.9	38	73.1	52	25.9	
	Cerebrospinal fluid leak	17	56.7	13	43.3	30	14.9	
	Meningitis	4	18.2	18	81.8	22	10.9	
	Hydrocephalus	1	5.3	18	94.7	19	9.5	

Table 3. continued

Table 4. Results of Logistic regression analysis for association of various risk factors with prevalence of death

Variables	Odds ratio	95% C.I for odd's ratio	P - Value	
Severe GCS	19.54	6.2 – 61.59	0.001	
Hypotension	10.43	4.76 – 22.84	0.001	
Respiratory distress	17.53	5.99 – 51.3	0.001	
Skull fracture	2.76	1.35 – 5.62	0.005	
Conservative treatment	20.03	8.91 – 45.05	0.001	

4. DISCUSSION

The current study aimed to determine the patterns of patients with TBI and to estimate the population-wide mortality and the contribution of TBI to injury-related mortality. In the present study, 48 (20.17%) of the patients were died during hospitalization, while 64.29% were discharged home. This is in keeping with the study of Onwuchekwa RC et al in Nigeria 2018 where the case fatality rate was 22.6% (11), and with Tran TM et al in Uganda 2015 (25.8%) (12). Also, in agreement with the present results are different studies in developed countries in which the reported mortality rate ranged from 19% to 31% (13, 14). However, the present rate of mortality was higher than that reported by Reza A et al in Iran 2018 (16%) (15) and Karasu A et al in Turkey 2009 (16). This high rate of mortality in the present study may be related to the causes of TBI where most causes were blasts which usually associated with severe head injury. In the present study, five clinical characteristics showed a significant association with the outcome (severe GCS, hypotension, respiratory distress, skull fracture, and conservative treatment. Severe GCS agrees with a large number of previous studies in that patients with severe GCS are less likely to survive compared with those with mild or moderate GCS (12, 17, 18). Thus, patients who survived, their level of consciousness was more stable in the first hours post trauma than those who died. Hypotension as a risk factor is in accordance with this result is the study of Tseng WC et al in 2011 (19) and Spaite DW et al in 2017 (20) studies. There is some evidence from animal models that the brain is selectively vulnerable to hypotensive insults following trauma. These physiologic insults do not damage a healthy brain but can lead to cell death in selectively vulnerable neurons following trauma (21). The mechanisms mediating the effect of respiratory distress in worsened outcome in TBI are not well understood. In a cohort study of 200 patients with severe TBI, Aisiku IP et al. in 2016 (22) found an association between respiratory distress and an elevation in early inflammatory plasma cytokines, interleukin (IL)-6 and IL-8, and anti-inflammatory cytokine, IL-10. However, more studies are required to fully illustrate this association. Regarding skull fracture, its presence brings to the attention of Health Care providers the role of immediate surgery in the management of patients. In the European Union, most of these patients are usually managed outside Neurosurgical Units, but there are recommendations in favor of either immediate

transfer of patients with a positive CT to specialized centers (23). About conservative treatment, surgical intervention prevents secondary brain injury, which is thought to be caused by a number of mechanisms. Extravasated blood is believed to be neurotoxic, leading to secondary injury that may be avoided by early surgical removal. Larger traumatic intracerebral hemorrhage may be associated with an ischemic penumbra of brain tissue that could be salvaged and some hemorrhages expand to the point where they cause mass effect, resulting in secondary brain injury (24). In conclusion, approximately one-fifth of causalities died during hospitalization. Each of sever GCS, respiratory distress, hypotension, skull fracture, and conservative treatment are significantly risk factors which increase mortality rate among patients with TBI.

5. CONCLUSIONS

About one-fifth of causalities died during hospitalization. Each of sever GCS, respiratory distress, hypotension, skull fracture, and conservative treatment are significantly risk factors which increase mortality rate among patients with traumatic brain injury.

Ethical Approval:

All ethical issues were approved by the author. Data collection and patients enrollment were in accordance with Declaration of Helsinki of World Medical Association, 2013 for the ethical principles of researches involving human. Signed informed consent was obtained from each participant and data were kept confidentially.

6. BIBLIOGRAPHY

- 1. National Academies of Sciences E, Medicine. Evaluation of the disability determination process for traumatic brain injury in veterans. 2019.
- 2. Dewan MC, Rattani A, Gupta S, Baticulon RE, Hung Y-C, Punchak M, et al. Estimating the global incidence of traumatic brain injury. Journal of neurosurgery. 2018;130(4):1080-97.
- 3. Maas AI, Menon DK, Adelson PD, Andelic N, Bell MJ, Belli A, et al. Traumatic brain injury: integrated approaches to improve prevention, clinical care, and research. The Lancet Neurology. 2017;16(12):987-1048.

- 4. Taylor CA, Bell JM, Breiding MJ, Xu L. Traumatic brain injury–related emergency department visits, hospitalizations, and deaths—United States, 2007 and 2013. MMWR Surveillance Summaries. 2017;66(9):1.
- 5. FICMS M. Hospital Treated Head injury in Basrah: clinical and epidemiological aspects. Thi-Qar Medical Journal. 2016;11(1).
- 6. Jaafar SA, Abed RI. Nurses' Knowledge toward Traumatic Head Injury During Golden Hour. Medico Legal Update. 2020;20(4):1594-8.
- 7. Ng SY, Lee AYW. Traumatic Brain Injuries: Pathophysiology and Potential Therapeutic Targets. Front Cell Neurosci. 2019;13:528-.
- 8. James SL, Theadom A, Ellenbogen RG, Bannick MS, Montjoy-Venning W, Lucchesi LR, et al. Global, regional, and national burden of traumatic brain injury and spinal cord injury, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. The Lancet Neurology. 2019;18(1):56-87.
- 9. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. The Journal of head trauma rehabilitation. 2006;21(5):375-8.
- 10. Algattas H, Huang JH. Traumatic Brain Injury pathophysiology and treatments: early, intermediate, and late phases post-injury. Int J Mol Sci. 2013;15(1):309-41.
- 11. Onwuchekwa RC, Echem RC. An epidemiologic study of traumatic head injuries in the emergency department of a tertiary health institution. Journal of Medicine in the Tropics. 2018;20(1):24.
- 12. Tran TM, Fuller AT, Kiryabwire J, Mukasa J, Muhumuza M, Ssenyojo H, et al. Distribution and characteristics of severe traumatic brain injury at Mulago National Referral Hospital in Uganda. World neurosurgery. 2015;83(3):269-77.
- 13. Basso A, Ignacio Previgliano M, Jm Duarte M, N Ferrari M. Advances in management of neurosurgical trauma in different continents. World journal of surgery. 2001;25(9):1174.
- 14. Corral L, Ventura JL, Herrero JI, Monfort JL, Juncadella M, Gabarrós A, et al. Improvement in GOS and GOSE scores 6 and 12 months after severe traumatic brain injury. Brain injury. 2007;21(12):1225-31.
- 15. Reza A, Riahi E, Daneshi A, Golchini E. The incidence of traumatic brain injury in Tehran, Iran. Brain injury. 2018;32(4):487-92.
- 16. Karasu A, Sabanci PA, Cansever T, Hepgül KT, Imer M, Dolaş I, et al. Epidemiological study in head injury patients. Ulusal travma ve acil cerrahi dergisi= Turkish journal of trauma & emergency surgery: TJTES. 2009;15(2):159-63.

- 17. De Silva MJ, Roberts I, Perel P, Edwards P, Kenward MG, Fernandes J, et al. Patient outcome after traumatic brain injury in high-, middle-and low-income countries: analysis of data on 8927 patients in 46 countries. International journal of epidemiology. 2009;38(2):452-8.
- 18. Maas AI, Steyerberg EW, Butcher I, Dammers R, Lu J, Marmarou A, et al. Prognostic value of computerized tomography scan characteristics in traumatic brain injury: results from the IMPACT study. Journal of neurotrauma. 2007;24(2):303-14.
- 19. Tseng W-C, Shih H-M, Su Y-C, Chen H-W, Hsiao K-Y, Chen I-C. The association between skull bone fractures and outcomes in patients with severe traumatic brain injury. Journal of Trauma and Acute Care Surgery. 2011;71(6):1611-4.
- 20. Spaite DW, Hu C, Bobrow BJ, Chikani V, Barnhart B, Gaither JB, et al. The effect of combined out-ofhospital hypotension and hypoxia on mortality in major traumatic brain injury. Annals of emergency medicine. 2017;69(1):62-72.
- 21. Mikrogianakis A, Shaye RE, Griffin P, Kawesa S, Lockwood J, Gendron NH, et al. Hypoxia alters the expression of inhibitor of apoptosis proteins after brain trauma in the mouse. Journal of neurotrauma. 2007;24(2):338-53.
- 22. Aisiku IP, Yamal J-M, Doshi P, Benoit JS, Gopinath S, Goodman JC, et al. Plasma cytokines IL-6, IL-8, and IL-10 are associated with the development of acute respiratory distress syndrome in patients with severe traumatic brain injury. Critical care. 2016;20(1):1-10.
- 23. Patel H, Bouamra O, Woodford M, King A, Yates D, Lecky F. Trends in head injury outcome from 1989 to 2003 and the effect of neurosurgical care: an observational study. The Lancet. 2005;366(9496):1538-44.
- 24. Mendelow AD, Gregson BA, Rowan EN, Francis R, McColl E, McNamee P, et al. Early surgery versus initial conservative treatment in patients with traumatic intracerebral hemorrhage (STITCH [Trauma]): the first randomized trial. Journal of neurotrauma. 2015;32(17):1312-23.

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