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ORIGINAL ARTICLE

Outcome of Patients with Traumatic Brain Injury Admitted to ICU UKMMC: A 5-Year Retrospective Study

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ABSTRAK

Kecederaan otak traumatis (TBI) telah menyebabkan morbiditi dan kematian yang signifikan di seluruh dunia, serta telah memberikan beban yang besar dari segi sosioekonomi. Pembangunan dan pematuhan terhadap garis panduan pengurusan telah menunjukkan hasil yang memberangsangkan dalam meningkatkan hasil rawatan pesakit dengan TBI, sama ada dari segi mortaliti mahupun fungsi. Walau bagaimanapun, garis panduan pengurusan yang teratur masih kurang di kebanyakan pusat rawatan di Malaysia. Kajian retrospektif tunggal di pusat ini bertujuan untuk menentukan hasil rawatan pesakit dengan TBI yang dirawat di Unit Rawatan Rapi (ICU) dan faktor risiko yang berkaitan. Kerja tambahan telah dilakukan untuk memantau status pekerjaan pesakit-pesakit ini. Pendaftaran ICU telah disaring untuk pesakit mengalami TBI yang dirawat dari tahun 2015 hingga 2019, dan maklumat yang berkaitan telah diekstrak. Regresi logistik telah digunakan untuk menentukan faktor risiko. Ujian McNemar telah digunakan untuk menganalisis status pekerjaan pasca-trauma pesakit-pesakit ini. Sejumlah 173 pesakit telah dikaji, dan kadar mortaliti tanpa mengira sebab adalah sebanyak 16.2%. Peningkatan usia, kejadian hipoksia dan hipotensi masing-masing dalam 24 jam pertama di ICU, dan tahap laktat yang lebih tinggi adalah faktor risiko bebas terhadap mortaliti. Sebanyak 40.9% pesakit yang bekerja atau merupakan pelajar sebelum TBI, tidak kembali bekerja/sekolah. Kami menyimpulkan bahawa mortaliti di hospital bagi pesakit yang dimasukkan di ICU disebabkan TBI adalah tinggi. Pengangguran selepas TBI juga tinggi, namun ia tidak secara langsung dikaitkan dengan kecacatan fungsi selepas kecederaan tersebut.

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ABSTRACT

Traumatic brain injury (TBI) has led to significant morbidity and mortality worldwide, imposing a substantial burden socioeconomically. Development and adherence to management guidelines has shown promising results in improving the outcomes of patients with TBI, in both mortality as well as functionality. Nevertheless, a protocolised management guideline remains lacking in many centres in Malaysia. This single-centre retrospective study aimed to determine the outcomes of patients with TBI admitted to Intensive Care Unit (ICU) and the associated risk factors. Additional work had been done to follow up on the employment status of these patients. ICU registry was screened for eligible patients with TBI admitted 2015-2019, and relevant information were extracted. Logistic regression was used to determine risk factors. McNemar test was used to analyse post-traumatic employment status of patients. A total of 173 patients were studied, and all-cause mortality of 16.2% was reported. Increasing age, occurrence of hypoxaemia and hypotension respectively during the first 24-hour in ICU, and higher lactate level were independent risk factors to mortality. As many as 40.9% of patients who were employed or was a student prior to TBI, did not return to work/school. We concluded that the in-hospital mortality for patients admitted to ICU for TBI was high. Unemployment after TBI is high, however, it is not directly attributed by functional disability following the injury.

Keywords: Brain injuries; functional status; intensive care units; mortality; risk factors; employment; traumatic

INTRODUCTION

Traumatic brain injury (TBI) is the main contributor to acute brain injury (Stocchetti et al. 2015). It is associated with significant short-term and long-term morbidity and mortality (Picetti et al. 2019; Shekhar et al. 2015; Stocchetti et al. 2015). Yearly, it is estimated that more than 10 million people hospitalised or die from TBI around the globe, imposing a substantial socioeconomic burden (Deepak et al. 2016). Mortality rate for patients with TBI has reduced across the decades as more studies and guidelines have been developed. Many have reported the positive impact of implementation of guidelines-based management protocols for patients with TBI on outcome including mortality rate, functional outcome scores, length of hospital stay and costs (Menon & Ercole 2017).

In Malaysia, TBI is reported to be the top three reason for Intensive Care

Unit (ICU) admission, attributed 7.1% to the total admission to ICU (Tai et al. 2017). Similar to reports on TBI epidemiology, research related to TBI in Malaysia remains scarce generally (Arulsamy et al. 2020). Moreover, despite the magnitude of the problem caused by TBI, there is lacking of protocolised management guidance and optimal monitoring in the ICU setting (Arulsamy et al. 2020; Menon & Ercole 2017).

We aimed to explore the in-hospital mortality for patients with TBI admitted to ICU. In view of many controversies remain in the management guidelines for TBI (Mishra et al. 2006), this study also designed to explore risk factors associated with mortality. We took a further step to follow up on the employment status of patients who were discharged from hospital after the injury.

MATERIALS AND METHODS

This retrospective, single centre study has gained approval from the Research Committee of Department of Anaesthesiology & Intensive Care, UKMMC and the Medical Research & Ethics Committee, UKMMC (JEP-2020-607). Samples included all patients with TBI, who was admitted to ICU UKMMC, from January 1st 2015 to December 31st 2019. Foreigners, and patients who were discharged against medical advice, or refused recommended medical or surgical interventions were excluded.

Convenient sampling method was used. The ICU registry was screened through systematically, and medical records of patients with a diagnosis of neurological injury including TBI, any types of intracranial bleed, head injury and polytrauma were reviewed at the record office. Patients with a confirmed diagnosis of TBI were then recruited.

Information extracted were entered into the data collection sheet in Microsoft Excel. Demographic and personal data were first extracted, including the pre-trauma functional status, comorbidity and employment status. Subsequently, the best Glascow Coma Scale (GCS) recorded in the Emergency Department (ED) was used to classify the severity of TBI. (Refer Appendix A) First vital signs on arrival to ED (T0) including blood pressure (BP), heart rate (HR), oxygen saturation (SpO2), temperature, incidence of hypotension and hypoxia throughout the stay in ED (T1) were recorded. Hypotension was defined as Systolic BP (SBP) less than 100 mmHg, and hypoxia was defined as SpO2 less than 95%. The nature of injury, presence of extracranial injury, need for surgical interventions and the types of surgery were explored. The timing of admission and transfer out from ED were also recorded.

On arrival to ICU (T2), vital signs of patient and serum lactate level were further recorded. The ICU treatment charts and records were examined to extract the parameters related to secondary injury prevention during the first 24 hours in ICU (T3). The parameters were further analysed to determine if the recommended target was present. These included BP (SBP >100mmHg), temperature (<38°C), sugar level (Dextrostix 6-10 mmol/L),

arterial oxygen tension (PaO2) (60-100 mmHg), arterial carbon dioxide tension (PaCO2) (35-45 mmHg), lactate level, haemoglobin level (>10/dL), serum sodium level (within normal reference range), steroid use, seizure control and presence of intracranial pressure (ICP) monitoring.

Outcome of patients were classified based on Glascow Outcome Scale (GOS) and was then further classified into non-survivor and survivor (Jennett et al. 1976). Duration of ICU stay, days on ventilator, length of hospitalisation and tracheostomy were also recorded. Patients who were discharged were followed up via phone calls to enquire on employment status and self-rated functional status at least one year after the injury. A telephone script, which was available in both Malay and English version, was used to obtain verbal consent and to deliver patient information. At the end of the conversation, respondents or care givers were asked to self-rate their functional status as not affected, mildly affected, and severely affected.

Sample Size Calculation

Sample size was calculated using PS Software version 3.0 according to Fleiss (1976) based on percentage of mortality between moderate TBI and severe TBI (16% vs 38% respectively) (Fleiss et al. 1976; Moppett 2007). The alpha value was set at 0.05, power study of 80%, and drop out of 20%. Therefore, total sample size calculated was 173.

Statistical Analysis

Data analysis was performed using SPSS for Windows version 25.0 (IBM Corp, Armonk, NY, USA). Mortality was described in percentage. Data was presented into 2 groups, non-survivor and survivor. Chi-Square test was used to analyse categorical data. Continuous variables were tested for normality. Mann-Whitney U-test was used for non-parametric data and the Student t-test for parametric data. Parametric data were presented as mean (Standard Deviation (SD)) and non-parametric data as median (Interquartile Range (IQR)). Logistic Regression test was used for univariate analysis. Variables with statistical significance were then analysed using enter method logistic regression in Model 1 to study the risk factors to the mortality in TBI patients. Statistically significance variables related to ICU care were analysed with the same method in Model 2. Employment status pre- and post-trauma was analysed using McNemar test. Post trauma employment status was cross tabulated with functional status and analysed using Chi-square test.

RESULTS

A total number of 173 patients with TBI were included with 145 patients survived to discharge, giving an all-cause in hospital mortality of 16.2% over five years. The demographic data and descriptive data were summarised in Table 1.

Univariate analyses between the 2 groups were displayed in Table 2.

TABLE 1: Demographical and Descriptive Data. Values expressed as mean ± SD, number (%), median [IQR]

Variable	n = 173
Age at injury, years	40.53 ± 17.34
Gender	
Male	155 (89.6)
Female	18 (10.4)
Comorbid	
No comorbid	116 (67.1)
≥ 1 comorbid(s)	57 (32.9)
Mechanism of Injury	
MVA	146 (84.4)
Fall	22 (12.7)
Assaults	5 (2.9)
Extracranial Injury	
No	39 (22.5)
Peripheral[a]	58 (33.5)
Central[b]	76 (43.9)
Days in ICU	8.75 [3.00 - 12.00]
Days on ventilator	7.90 [3.00 - 11.00]
Days in Hospital	25.68 [9.00 - 31.50]
Survivor	145 (83.8)

[a]Peripheral extracranial injury: injury involving limbs only; [b]Central extracranial injury: injury involving head, neck, spine, chest, abdomen, and pelvis; MVA: motor vehicle accident; ICU: intensive care unit

Patients who survived to hospital discharge were generally younger population with an average age of 38.5 year (P<0.001). Patients with at least one co-morbidity had 2.37 times the odds of mortality compared to patients with no comorbidity (P=0.048). Higher capillary sugar level on arrival to hospital was associated with increased mortality (P=0.023). Occurrence of hypotensive episodes in the ED showed an approximate 3 times risk of death among the patients (P = 0.014). An average serum lactate level on arrival to ICU admission in the survival group was 2.6 mmol/L, with 1.6 times increase in risk of mortality with every 1 mmol/L increase in serum lactate (P<0.001). Patients who had surgical intervention prior to ICU admission was found to have an increased risk of mortality (P<0.008), however, types of surgery, intraoperative hypotension and hypoxia did not affect the mortality risk.

Referring to the Table 2, for parameters obtained during the first 24 hours in ICU (T3), episodes of hypotension (P<0.001), hypoxia (P<0.001), uncontrolled blood sugar level (P = 0.002), and higher peak serum lactate level (P<0.001) were associated with increased mortality. Hyperoxia, abnormal PCO2 level, use of sedation, serum sodium level, use of prophylactic anti-epileptics, anemia, hyperthermia, use of ICP monitoring devices, commencement and duration of cerebral protection were not associated with increased mortality. ICP monitoring was applied in 6.9% of patients. Steroid was not administered during the first 24 hours of ICU admission in any patients.

In Model 1 multivariate analysis all the statistically significant variables were included. Age, episodes of hypotension and hypoxia within the first 24 hours in ICU were independent risk factors for increased mortality (Table 3).

In Model 2, we focused on statistically significant variables related to ICU management. Age, hypotension, hypoxaemia and higher serum lactate level during the first 24 hours in ICU independently increased risk for mortality (Table 4).

Table 5 compared the employment status of patients pre- and post-

TABLE 2: Univariate Analysis

Variable	Survivor (n = 145)	Non-survivor (n = 28)	P-value	Univariate OR, (CI)	P-value
Age at injury, years	38.5 ± 16.8	51.3 ± 16.2	<0.001	1.043 (1.018-1.069)	0.001*
Gender					
Male	129 (89.0)	26 (92.9)	0.741	1.612 (0.349 - 7.440)	0.540
Female	16 (11.0)	2 (7.1)		reference	
Comorbid					
No comorbid	102 (70.3)	14 (50.0)	0.048	reference	0.039*
≥ 1 comorbid(s)	43 (29.7)	14 (50.0)		2.37 (1.043 - 5.397)	
Mechanism of injury					
MVA	126 (86.9)	20 (71.4)	0.070	N/A	
Fall	16 (11.0)	6 (21.4)			
Assaults	3 (2.1)	2 (7.1)			
GCS	8.6 ± 3.3	8.3 ± 4.4	0.743	0.975 (0.866 - 1.098)	0.682
Severity of TBI					
Mild	29 (20.0)	8 (28.6)	0.591	reference	0.595
Moderate	27 (18.6)	5 (17.9)		0.671 (0.195 - 2.306)	0.527
Severe	89 (61.4)	15 (53.6)		0.611 (0.235 - 1.588)	0.312
Timing of Admission					
Office hour	52 (35.9)	10 (35.7)	0.988	0.994 (0.427 - 2.311)	0.988
Non-office hour	93 (64.1)	18 (64.3)		reference	
Extracranial Injury					
No	30 (20.7)	9 (32.1)	0.412	reference	0.419
Peripheral	50 (34.5)	8 (28.6)		0.533 (0.186 - 1.531)	0.243
Central	65 (44.8)	11 (39.3)		0.564 (0.211 - 1.505)	0.253
HR T0, bpm	99.3 ± 23.5	95.2 ± 23.8	0.417	0.992 (0.974 - 1.011)	0.414
DXT at T0, mmol/L	9.48 (7.38-10.43)	11.64 (7.90-14.50)	0.017	1.107 (1.014 - 1.209)	0.023*
Hypotension at T0	10 (6.9)				
Yes	134 (93.1)	3 (10.7)	0.490	1.608 (0.413 - 6.259)	0.493
No		25 (89.3)		reference	
Hypoxia at T0					
Yes	30 (20.7)	5 (17.9)	0.851	0.826 (0.290 - 2.355)	0.721
No	114 (78.6)	23 (82.1)		reference	
Hypotension at T1					
Yes	26 (18.3)	11 (39.3)	0.014	2.887 (1.210 - 6.887)	0.017*
No	116 (81.7)	17 (60.7)		reference	
Hypoxia at T1					
Yes	41 (28.7)	7 (25.0)	0.693	0.829 (0.328 - 2.100)	0.693
No	102 (71.3)	21 (75.0)		reference	
Duration in ED, hours	9.0 (7.0 - 13.0)	8.0 (6.0 - 9.0)	0.054	0.917 (0.831 - 1.013)	0.087
Lactate at T2, mmol/L	2.6 ± 1.5	6.0 ± 4.2	<0.001	1.625 (1.344 - 1.965)	<0.001*
Hypotension at T3					
Yes	48 (33.1)	17 (60.7)	<0.001	4.908 (1.906-12.636)	0.001*
No	97 (66.9)	7 (25.0)		reference	
Hyperoxia at T3					
Yes	134 (92.4)	22 (78.6)	<0.001	1.806 (0.222-14.692)	0.580
No	11 (7.6)	1 (3.6)		reference	

CO2 controlled at T3					
Uncontrolled	107 (73.8)	20 (87.0)	0.172	2.368 (0.666 - 8.420)	0.183
Controlled	38 (26.2)	3 (23.0)		reference	
Highest lactate at T3, mmol/L	2.63 ± 1.65	6.72 ± 5.56	<0.001	1.468 (1.233 - 1.747)	<0.001*
Sugar control at T3					
Uncontrolled	64 (44.1)	19 (82.6)	0.001	6.012 (1.948-18.553)	0.002*
Controlled	81 (55.9)	4 (17.4)		reference	
Acidosis at T3					
Yes	58 (40.0)	16 (69.6)	0.080	3.429 (1.328 - 8.850)	0.011*
No	87 (60.0)	7 (30.4)		reference	
Sedation in ICU					
Yes	134 (93.1)	22 (95.7)	0.641	0.609 (0.074 - 4.996)	0.644
No	10 (6.9)	1 (4.3)		reference	
Normal Serum Sodium at T3					
Yes	130 (90.9)	18 (78.3)	0.139	2.778 (0.886 - 8.713)	0.080
No	13 (9.1)	5 (21.7)		reference	
Antiepileptic use					
Yes	81 (55.9)	12 (48.0)	0.466	1.371 (0.586 - 3.209)	0.467
No	64 (44.1)	13 (52.0)		reference	
Hb >10 at T3					
Yes	121 (85.2)	19 (82.6)	0.756	1.213 (0.375 - 3.922)	0.747
No	21 (14.8)	4 (17.4)		reference	
Steroid use in ICU					
No	145 (100.0)	25 (100.0)	N/A	N/A	
Yes	0 (0.0)	0 (0.0)			
ICP Monitoring					
Yes	11 (7.6)	1 (4.0)	0.518	1.97 (0.243 - 15.972)	0.525
No	134 (92.4)	24 (96.0)		reference	
Hyperthermia at T3					
Yes	97 (66.9)	12 (52.2)	0.169	0.54 (0.222 - 1.312)	0.174
No	48 (33.1)	11 (47.8)		reference	
Highest Temperature at T3, ℃	38.2 (37.6 - 38.8)	38.0 (36.0 - 39.3)	0.436	0.732 (0.520 - 1.029)	0.073
Operation					
Yes	64 (44.1)	20 (71.4)	0.008	3.164 (1.308 - 7.651)	0.011*
No	81 (55.9)	8 (28.6)		reference	
Cranial operation					
Yes	42 (65.6)	15 (75.0)	0.433	1.571 (0.505 - 4.894)	0.435
No	22 (34.4)	5 (25.0)		reference	
Intraop Hypotension					
Yes	36 (60.0)	15 (78.9)	0.132	2.500 (0.740 - 7.450)	0.140
No	24 (40.0)	4 (21.1)		reference	
Intraop Hypoxia					
Yes	4 (6.5)	3 (15.8)	0.346	2.719 (0.551-13.412)	0.219
No	58 (93.5)	16 (84.2)		reference	
Cerebral Protection					
No	102 (70.8)	17 (63.0)	0.415	1.429 (0.605 - 3.375)	0.416
Yes	42 (29.2)	10 (37.0)		reference	
Duration of CP, hours	20.4 ± 18.3	15.5 ± 19.7	0.223	0.984 (0.961 - 1.008)	0.195

Days in ICU	7.0 (4.0 - 12.5)	2.0 (1.0-8.0)	<0.001	0.982 (0.928 - 1.038)	0.522
Days on ventilator	6.0 (3.0 - 11.0)	2.0 (1.0 - 8.0)	0.005	0.999 (0.951 - 1.050)	0.978
Days in Hospital	19.0 (10.0 - 37.0)	3.0 (1.25 - 18.0)	<0.001	0.986 (0.965 - 1.007)	0.183
Tracheostomy					
Yes	58 (40.0)	6 (21.4)	0.062	0.409 (0.156 - 1.070)	0.069
No	87 (60.0)	22 (78.6)		reference	

*p<0.05 showed statistical significance; CP: cerebral protection; DXT: Dextrostix; intraop: Intraoperative; MVA: motor vehicle accident

TABLE 3: Multivariate analysis model 1

Variable	Adjusted OR	95% CI (Lower-Upper)	P-value
Age at injury, years	1.056	1.009 - 1.104	0.018*
Comorbid	1.423	0.156 - 3.175	0.647
Hypotension at T1	0.270	0.044 - 1.657	0.157
Lactate at T2	1.265	0.842 - 1.899	0.258
Hypotension at T3	5.382	1.395 - 20.759	0.015*
Hypoxaemia at T3	11.95	1.894 - 75.384	0.008*
Sugar control at T3	2.871	0.771 - 10.698	0.116
Acidosis at T3	1.338	0.303 - 5.905	0.700
Highest lactate at T3	1.215	0.875 - 1.688	0.246
Surgical intervention	1.203	0.302 - 4.786	0.793

Variable(s) entered in the Model 1: Age at injury, Comorbid, Hypotension at T1, Lactate at T2, Hypotensive at T3, Hypoxaemia at T3, Sugar control at T3, Acidosis at T3, Highest lactate at T3, Surgical intervention.
*P<0.05 showed statistical significance

TABLE 4: Multivariate analysis model 2

Variable	Adjusted OR	95% CI (Lower-Upper)	P-value
Age at injury, years	1.056	1.017 - 1.096	0.004*
Hypotension at T3	4.593	1.315 - 16.042	0.017*
Hypoxaemia at T3	8.057	1.450 - 44.760	0.017*
Sugar control at T3	3.102	0.839 - 11.468	0.090
Acidosis at T3	0.853	0.232 - 3.141	0.811
Highest lactate at T3	1.384	1.083 - 1.768	0.009*

Variable(s) entered Model 2: Age at injury, Hypotensive at T3, Hypoxaemia at T3, Sugar control at T3, Acidosis at T3, Highest lactate at T3.
*P<0.05 showed statistical significance

TABLE 5: McNemar analysis on employment status

	Post Trauma (N=103)		
Prior to Trauma	Employed / Student	Unemployed	P value
Employed / Student	52 (50.5%)	36 (35.0%)	<0.001*
Unemployed	6 (5.8%)	9 (8.7%)	

*p<0.05 showed statistical significance

trauma. Patients who had deceased, retired, or were uncontactable were excluded from this analysis. A total of 36 out of 88 (40.9%) patients, who were employed or was a student prior to TBI, did not return to work/school during the follow-up call (P<0.001).

In Table 6, post-trauma employment status was cross tabulated with functional status. Eight patients refused to disclose their functional status. Three were among the unemployed post-trauma, and five were among the employed post-trauma. Amongst those who were unemployed after trauma (N = 42), majority were functionally affected.

DISCUSSION

The all cause in-hospital mortality of patients with TBI admitted to ICU UKMMC between year 2015-2019 is high. According to a recent cross-national study involved 4 Asian countries including Malaysia, poor functional outcome including death was reported in only 11.4% of the patients with TBI (Chen et al. 2020). In developed country like in the United States, the reported mortality has reduced from 22% to 13% among patients with severe TBI from year 2001 to 2009 (Gerber et al. 2013). In New Delhi (India), the mortality of patients with TBI was reported to be as low as 10% (Shekhar et al. 2015). However, the authors have included all the patients with TBI in the centre, in which only 37% of the patients had moderate to severe TBI, which could have attributed to the low case fatality rate. When considering only patients with severe TBI, Shekhar et al. (2015) reported a mortality of as high as 73.5%. Similarly, the mortality reported in our study could be attributed to the unequal distribution of the severity of TBI among the selected samples in which 78% of the samples had moderate to severe TBI. Many authors have independently reported that employment of effective protocolised management guidelines had led to reduction of mortality (Deepak et al. 2016; Gerber et al. 2013).

Risk factors for mortality can be generally divided into non-modifiable and modifiable. Among the non-modifiable risk factors include, age, gender, nature of injury, pupillary signs, severity of TBI or GCS, and a positive CT findings (Czosnyka et al. 2005; Moppett 2007; Roozenbeek et al. 2012;

TABLE 6: Cross tabulate employment status with functional status

Functionality	Employed / Student	Unemployed	P-value	P-value[a]
Not affected	49 (92.4)	20 (47.6)		Not affected vs others: < 0.001
Mildly affected	3 (5.7)	14 (33.3)	< 0.001	Mildly affected vs others: < 0.001
Severely affected	1 (1.9)	8 (19.1)		Severely affected vs others: 0.004

[a]Post Hoc analysis with Bonferroni correction, P<0.017.

Shekhar et al. 2015). Similar to our findings, many authors have reported age as independent risk factors to poorer outcome in TBI. The postulated theory behind this is that as patient aged, the autoregulatory mechanism in the brain deteriorates and increases its vulnerability to secondary insults (Czosnyka et al. 2005). Although lower GCS and increase in severity have both proven to increase mortality, we could not demonstrate the statistical significance probably due to the difference in study design. However, we did not look into data on pupillary reflexes and CT-brain findings in our study. Like all other studies on TBI, we reported male predominance among our patients with TBI. Although some has reported worse imaging findings in women, gender has not been related to outcome (Arulsamy et al. 2020; Moppett 2007; Roozenbeek et al. 2012; Raj et al. 2014; Shekhar et al. 2015). Contrary to developed countries where fall and assault attributed to majority of the cause for TBI, we reported that MVA was the major cause of TBI (Thornhill et al. 2000).

In the present study, we found that hypoxaemia and hypotension are two independent risk factors, which could result in increased serum lactate level similar to findings by Fu et al. (2019). Modifiable risk factors are generally those which causes secondary injury, and are the targets of many guidelines developed to improve the outcome of patients (Dash & Chavali 2018; Menon & Ercole 2017). These risk factors include, but not limited to, hypotension, hypoxia, hyperglycaemia (Mishra et al. 2006), hypercapnia and hypocapnia, failure to detect and delayed management of raised ICP (Carney et al. 2017; Menon 1999), administration of steroid (CRASH trial collaborators 2005), inadequate reduction in cerebral metabolic demands with the use of anaesthetics (Menon 1999).

Despite so many studies had been done over the decades, certain controversial risk factors remained unproven of its significance (Mishra et al. 2006). These include hypothermic therapy (Yokobori & Yokota 2016), use of preventive antiepileptics (Menon 1999), and avoidance of hyperoxia (Vincent et al. 2017). Similarly in this study, we could not demonstrate benefits of avoidance of hyperthermia and hyperoxia to mortality.

In the present study, we found that 36 out of 88 (40.1%) patients who were working or going to school became unemployed at least 1 year after the injury. TBI has been labelled as "silent epidemic", leaving long term disability in patients regardless of its severity (Dahm & Ponsford 2015; Thornhill et al. 2000). Thornhill et al. (2000) reported that 67% of patients with severe TBI and 45% of patient with moderate TBI had moderate to severe disability. Dahm and Ponsford (2015) found that18% of the patients who were in labour-force prior to sustaining TBI became unemployed post-injury. Severity of the TBI, and thence possibly more severe disability, had been shown to attribute to the post-injury unemployment. Interestingly, we detected amongst those who were unemployed post-trauma, only minority were self-rated as severely affected functionally while

the rest were mildly or not affected at all. The present study was conducted amid the COVID-19 pandemic when the economy was bad, and this could be the explanation to the high rate of unemployment among our patients.

The present study is limited by its retrospective design using data from a single centre. It is also limited by the variable reception due to the uncommon practice of follow-up calls in our local settings. Despite its limitations, this study provides the basis for larger scale study with more focus on particular risk factors. It also provides support for future prospective study to explore the employment and functional status using more structured questionnaires such as Modified Rankin Scale (MRS), Structured Outcome Questionnaire (SOQ) and Quality of Life after Brain Injury (QOLIBRI).

CONCLUSION

The ICU mortality for patients admitted for TBI is high. Increasing age, incidence of hypotension and hypoxaemia, and increase lactate level are the independent risk factors to poorer outcome. Unemployment after TBI is high, however, it is not directly attributed by functional disability following the injury.

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