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Clinical characteristics of traumatic brain injury patients in Dr. Zainoel Abidin Public Hospital Banda Aceh, Indonesia

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ABSTRACT

Background: Traumatic brain injury (TBI) is responsible for more than one million hospital visits and causes disability and death in all age groups and genders. Every day, 153 people die from injuries including traumatic brain injury.

Methods: We use a retrospective, cross-sectional study design. We collect data from patients admitted to our hospital on November 2018 that was diagnosed and treated for traumatic brain injury. The clinical information at follow-up was also analyzed.

Results: We collected 60 cases of TBI, that consist of 35 male (58%) and 25 females (42%) with an average age of 18-39 years. 42% of patients had post-traumatic amnesia. The leading cause of TBI was from motor vehicles accident, which constitutes to 90%, followed by falls (8%) and others (i.e. self-harm and assaults 2%). Glasgow Coma Scale (GCS) were examined, and we found 85% of patients presented with GCS scores > 9 and 25% with score < 8. Brain Ct scanning findings showed cerebral oedema (25%), epidural hemorrhage (18%), intracranial hemorrhage (15%), and subarachnoid haemorrhage (12%). Laboratory findings showed an increase of leukocyte count (80%), high segmented neutrophils count (72%), hyperglycemia after TBI (7%), hyponatremia (2%), hypernatremia (11%), hypokalemia (22%), hyperkalemia (17%) and hyperchloremia (30%).

Conclusion: We reported 60 cases of TBI and collected demographic data upon patients arrival in the hospital, such as; GCS score, brain imaging and laboratory findings. This information can help prevention strategies, identify research and education priorities, and support service needs among those living with TBI.

Keywords: demographic, characteristics, brain, injury, post-traumatic amnesia.

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INTRODUCTION

Traumatic brain injury (TBI) is a staggering health issue that imposes serious threats and medical concerns to public health.[1] In the United States, patients will experience long-term disabilities more than 40%, 235.000 patients are hospitalized and 50.000 die, 1.4 million patients suffer traumatic brain injury (TBI).[2,3,4] In neurocritical treatment, nearly one-third of patients treated with severe TBI die, and less than half have good neurological outcomes, such as hospital-acquired infections, dementia-related injuries, hemiplegia, and depression.[2,5-8,11] Activation of the proinflammatory state (systemic inflammatory response syndrome (SIRS).[12,13,14] Neuroinflammation promoted by resident microglia and induced secondary injury. Additionally, an extracranial injury that occurs together and massive bleeding can cause hypoxia or arterial hypotension and hence promotes SIRS which can further aggravate the development of secondary injuries.[15] This series of complex events starts minutes after trauma but lasts for weeks or even months, especially for inflammation.[16] Different types of lesions can usually occur in combination. While bruising the brain, intracerebral, and subdural hematomas, and fast-growing non-evacuating epidural hematomas have a high risk for high ICP and sequential severe disability or mortality, the risk for ICP increase is low for axonal injury, traumatic subarachnoidal haemorrhage and petechial bleeding.[12,17] Risk for disability and mortality especially for axonal injuries in some areas of the brain but also for extensive traumatic subarachnoidal haemorrhage may be high, too.[18] Age-related comorbidities promote secondary brain damage and reduce (but not eliminate) plasticity and nerve repair in the elderly, which compromises the elderly.[19] Monitoring and treatment of comorbidities are therefore as important as TBI management itself in determining outcomes. That is, treatment of drug-induced coagulopathy by reversing anticoagulant or antiplatelet therapy is very important in impending bleeding or intracranial haemorrhage.[9,10] Epileptic seizures must be detected early and treated with immediate treatment. However, optimal therapy and prophylactic seizure duration in this population are unclear. In older patients, a lower ICP threshold is already associated with poor outcomes. This might reflect greater vulnerability of an older brain and

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might indicate worse brain injury due to atrophy and increased CSF space allowing for clearer lesion expansion and brain oedema before ICP increases. This will provide a reason to investigate whether reduced thresholds for ICP control can be beneficial in this population. However, with increasing ICP frequency in the elderly, it can be observed and intracranial probe insertion may be more risky, especially in patients using anticoagulant and antiplatelet drugs, a conservative approach to ICP monitoring in these patients must be considered.[12]

METHODS

We use a retrospective cross-sectional study design. We collect data from patients admitted in our hospital on November 2018 that was diagnosed and treated for traumatic brain injury. The clinical information at follow-up was also analyzed.

RESULTS

We collected 60 cases of TBI, that consist of 35 male (58%) and 25 females (42%) with an average age of 18-39 years. 42% of patients had post-traumatic amnesia. The leading cause of TBI was from motor vehicles accident, which constitutes to 90%, followed by falls (8%) and others (i.e. self-harm and assaults 2%). Glasgow Coma Scale (GCS) were examined, and we found 85% of patients presented with GCS scores > 9 and 25% with score < 8. Brain Ct scanning findings showed cerebral oedema (25%), epidural hemorrhage (18%), intracranial hemorrhage (15%), and subarachnoid haemorrhage (12%). Laboratory findings showed an increase of leukocyte count (80%), high segmented neutrophils count (72%), hyperglycemia after TBI (7%), hyponatremia (2%), hypernatremia (11%), hypokalemia (22%), hyperkalemia (17%) and hyperchloremia (30%).

Tabel Clinical Characteristics Of Traumatic Brain Injury

Clinical Characteristics	Frequency (n)	Percentage (%)
Age (years)		
<18	17	28
18-39	28	47
40-64	9	15
>65	6	10
Gender		
Male	35	58
Female	25	42
Admission GCS Score		
GCS 13-15	32	53
GCS 9-12	19	32
GCS 3-8	9	15
Injury Severity Score		
GCS 13-15	32	53
GCS 9-12	19	32
GCS 3-8	9	15
Cause Of Injury		
Traffic Accident	54	90
Fall	5	8
Assault	1	2
Post Traumatic Amnesia		
Positive	25	42
Negative	35	58

Tabel ***Continues***

Clinical Characteristics	Frequency (n)	Percentage (%)
Brain Imaging		
Epidural Hemorrhage (EDH)	11	18
Subdural Hemorrhage (SDH)	4	7
Intracranial Hemorrhage (ICH)	9	15
Subarachnoid Hemorrhage (SAH)	7	12
Cerebral Oedema	16	27
Cerebral Comutio	13	21
Serum Hemoglobin (g/dl)		
Anaemia	6	10
Normal	54	90
Serum Platelet		
<150,000	2	3
150,000-450,000	57	95
>450,000	1	2
Serum Haematocrit		
<37	33	55
37-47	25	42
>47	2	3
Serum Erythrocyte		
<4.2	32	53
4.2-5.4	25	42
>5.4	3	5
Serum Leukocyte		
<4.5	-	-
4.5-10.5	12	20
>10.5	48	80
Random Plasma Glucose (mg/dl)		
<200	56	93
>200	4	7
BUN (mg/dl)		
<13	1	2
13-43	50	83
>43	9	15
Serum creatinine (mg/dl)		
<0,51	9	15
0,51-0,95	30	50
>0,95	21	35
Serum Sodium (mEq/l)		
<132	1	2
132-146	52	87
>146	7	11

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Tabel ***Continues***

Clinical Characteristics	Frequency (n)	Percentage (%)
Serum Potassium (mEq/l)		
<3,7	13	22
3,7-5,4	46	77
>5,4	1	17
Serum Chloride (mEq/l)		
<98	8	13
98-106	34	57
>106	18	30
Serum Monocyte		
2-8	51	85
>8	9	15
Serum Lymphocyte		
20-40	58	97
>40	2	3
Serum Neutrophils Segmented		
50-70	17	28
>70	43	72
Serum Eosinophils		
0-6	58	97
>6	2	3
Serum Basophils		
0-2	60	100
>2	-	

DISCUSSION

From the research conducted in the November 2018 period, it is known that the highest number of head injury sufferers treated in young adults is 18-39 years, 28 with the majority of male patients 58% of the total patients having head injuries. 53% GCS entry is 13-15. Age is one of the strongest predictors and is proven best in head injuries, where the output will get worse with increasing age.

Traumatic brain injury leading cause of disability and death worldwide. In Europe and the US, more than 13 million people estimated disabilities after TBI, also increasingly affecting people older than 65 years, usually after falling from low heights in developed countries while aged 15-44 as victims of high-speed traffic accidents has declined due to improved road conditions, improved enforcement stronger traffic regulations, and improved safety features of the vehicle.[20] In low and middle-income countries, burden of TBI in younger patients is becoming more and more prominent. Due to road traffic accidents incidence rates varying from 150-170 per 100,000 compared to global rates of 106 per 100,000 in Latin America and Sub-Saharan Africa. Patients with TBI have serious injuries, about 10-15% require specialist care, especially in the intensive care unit (ICU).[21]

Older patients usually suffer from low energy impacts, leading to subdural hematomas and fewer bruises or epidural hematomas.[22,23,24] Cerebral atrophy in Older patients can increase CSF space resulting in a lower incidence of increased ICP.[25] Age- related comorbidities with a reduced physiological process in acute phase reactions, neurohumoral and metabolic changes, and the fact that post-traumatic seizures are more common in older patients. Incidence and severity of initial secondary insults (especially hypoxia and hypotension) increase secondary brain damage.[12] TBI results from external physical force transmitted to the head that disrupts function of the brain and normal structural brain.[26,27]

Several clinical tools, including radiography (Marshall CT classification) and clinical (Galveston Orientation and Amnesia Test, Glasgow Coma Scale (GCS).[28,29] GCS has limitations, including provider subjectivity and loss of the verbal

component after patient intubation. That said, the best motor response to GCS, after the patient has been resuscitated, is the most reliable prognostication metric. Technological advances such as head computed tomography do not add to the predictive value of the clinical scale.[27,30,31] Most TBI cases (around 80%) are mild (GCS 13-15), with about 10% moderate (GCS 9-12), and 10% severe (GCS 3-8).[32] As expected, mortality rates tend to be more severe TBI. Overall, TBI deaths were 17 per 100,000 for TBI deaths outside the hospital and 6 per 100,000 for TBI deaths in the hospital.[26]

The most common complaints in head injuries were headache (25.6%) and vomiting (20.9%) while loss of consciousness was 6.5%. The mechanism of injury in this study was 90% due to accidents. A total of 25 patients suffered from post-traumatic amnesia. Of the 60 patients included in this study, 16 were patients with cerebral oedema head scan, 11 patients with EDH images. A more recent classification scheme for TBI uses length of loss of consciousness (LOC), alteration of consciousness (AOC), and post-traumatic amnesia (PTA) as well as imaging findings to categorize TBI.[33]

The American Congress of Rehabilitation Medicine defines mild TBI (considered synonymous with the term "concussion" for this review) as a traumatic brain injury that results in the following, if present: 1) loss of consciousness for up to 30 minutes, 2) alteration of consciousness for less than 24 hours, 3) posttraumatic amnesia for less than 24 hours, and 4) a Glasgow Coma Scale score of 13-15 at 30 minutes after injury.[34] These variables are also used to distinguish moderate TBI (loss of consciousness for 30 minutes to 24 hours, alteration of consciousness or posttraumatic amnesia for 24 hours to 7 days, Glasgow Coma Scale score of 9-12) and severe TBI (loss of consciousness >24 hours, alteration of consciousness/posttraumatic amnesia for >7 days, Glasgow Coma Scale score <9), in which recovery is generally more prolonged and functional recovery less likely.[35] Postconcussive symptoms typically fall into one of four categories: vestibular (e.g., nausea, imbalance, dizziness), sensory (e.g., migraines, tinnitus, photo/phonophobia, blurry vision), cognitive (e.g., forgetfulness, difficulty focusing), and emotional (e.g., insomnia, fatigue, depression, irritability).[36]

After laboratory tests, 54 patients with normal haemoglobin levels were found, 57 patients with platelet levels of 150,000-450,000, 33 patients with hematocrit levels <37. Thirty-two patients with erythrocytes <4.2. 80% patients about leukocyte count increase, 72% neutrophils segmented high, 7% hyperglycemia after TBI, 2% hyponatremia, 11% hypernatremia, 22% hypokalemia, 17% hyperkalemia and 30% hyperchloremia. Increased ICP, hemorrhagic contusions, and cerebral oedema are closely related to morbidity and mortality of TBI and very important to the prevention of secondary brain injury.[37,38] Malignant cerebral oedema is very difficult to manage because it is usually refractory to medical therapy and causes permanent injury, with mortality approaching 100% when not treated.[39] Clinical, surgical studies (decompressive hemicraniectomy, ICP monitor placement, CSF drainage) and medical (hyperosmolar therapy, sedation and coma, hypothermia) interventions have shown a reduction in ICP, with some reduction in mortality, but no increase in functional outcome. There are no current recommendations regarding proper haemoglobin or hematocrit concentrations in patients with severe TBI. When 67 patients with moderate to severe TBI from Transfusion in the Critical Care Trial 40 were examined retrospectively, there was no benefit from a liberal transfusion strategy (goal, 10 g/dL) compared to a restrictive strategy (goal, 7 g/dL) could be shown.[40] Furthermore, no significant differences were found when the same haemoglobin threshold of 7 and 10 mg / dL was tested, together with placebo injection or erythropoietin, in a prospective multicenter Phase III TBI trial.[41] Before this study, evidence for haemoglobin concentrations affecting the outcome of severe TBI patients was limited to analysis and retrospective associations, which suggested that patients with lower haemoglobin fared worse.[42,43] Known as "acute traumatic coagulopathy," it is thought to be caused by the release of tissue factors, which are in high concentration in the brain. This condition was first described in 1974 and is associated with an almost increase in mortality and worse cognitive outcomes in survivors.[44,45,46] Coagulopathy, including thrombocytopenia and increase international normalisation ratio, must be corrected immediately in TBI patients. TBI is a common cause of seizures.[47] Posttraumatic seizures can be classified as direct seizures (occurring less than 24 hours after injury), premature seizures (occurring 24 hours to 7 days after injury), and slow seizures (occurring more than 7 days after injury). Late seizures often cause lifelong epilepsy.[47] Hypermetabolism, catabolism, and nitrogen loss all accompany severe TBI. BTF CPG 2016 recommends feeding patients with at least a basal calorie replacement on the fifth day (no later than the seventh) to reduce mortality.[48]

ED glucose was increased and the amount of WBC was both predictive of parenchymal or hemorrhagic contusions (p=0.0028, p=0.0279,). Increased ED glucose alone was significantly associated with subdural hematoma (p=0.0451) and subarachnoid haemorrhage (p<0.0001), whereas an

increase in WBC count was significantly associated with skull fracture (p <0.0001) and diffuse axonal injury (p=0.0096).[49] Hypernatremia is a significant independent predictor of mortality (hazard ratio for mild: 3.4, moderate: 4.4, and severe: 8.4; p<0.001).[50] The patients (n = 15) who had severe hypokalemia, hypernatremia (Na>160 mmol/L), and hypophosphatemia (p<0.3 mmol/L) all died in the hospital. Multiple logistic regression analysis resulted in decreased GCS (OR=1.27; 95% CI = 1.15-1.41; p<0.001) and potassium (OR=4.35; 95% CI = 2.04-9.26; p <0.001) was associated with a significant increase in the risk of death.[51]

We reported 60 cases of TBI and collected demographic data upon patients arrival in the hospital, such as; GCS score, brain imaging and laboratory findings. This information can help inform TBI identify research, education priorities, prevention strategies and support the need for services among those living with a TBI.

ETHICAL CLEARANCE

CONFLICT OF INTEREST

There were no conflicts of interest in this study.

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AUTHOR CONTRIBUTIONS

All of authors are contributed to the study from the study framework, data gathering, data analysis, until reporting the result of study

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