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RISK FACTORS OF HIGH INTRACRANIAL PRESSURE AND THE RELATED OUTCOMES IN SUBJECTS WITH SEVERE HEAD INJURY IN SANGLAH GENERAL HOSPITAL DENPASAR

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ABSTRACT

Background: Severe head injury management target is to prevent the secondary brain injury characterized by deterioration in the outcome. High intracranial pressure (ICP) and low cerebral perfusion pressure (CPP) could cause unfavourable outcomes which is influenced by many factors, such as hypoxia and haemorrhage lesions pictured on head CT scan. This study analyzes various risk factors that can lead to increased ICP and the influence of high ICP on the outcome.

Methods: This study is a prospective cohort, involving 42 consecutive subjects with severe head injury patients from June to October 2016. The subjects underwent examination for blood pressure, blood gas analysis, and head CT scan. ICP monitoring was then performed and the outcome was assessed using the Glasgow Outcome Scale score when the patient was discharged. In this study, risk factors such as hypoxia, hypotension, and subarachnoid haemorrhage (SAH) were analyzed. Statistical analysis was performed with SPSS 27 with a confidence interval of 95%.

Result: There were two risk factors that significantly influenced the increase of ICP, which were hypotension (RR 0.27; 95CI 0.095-0.775; p<0.001) and hypoxia (RR 0.125; 95CI 0.034-0.457; p<0.001). High ICP value ≥ 20 mmHg was associated with an unfavourable outcome (RR 2.28; 95CI 1.31-3.98; p<0.001).

Conclusion: Hypoxia and hypotension were two risk factors that significantly influenced the increase of ICP, where high ICP caused the unfavourable outcome.

Keywords: *severe head injury, secondary brain injury, hypoxia, hypotension, SAH, ICP monitoring, outcome.*
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INTRODUCTION

Traumatic brain injury (TBI), one of the most common findings in the emergency department, included 3.4% from all cases, with an incidence around 450 cases per 100,000 population. This is also the most common cause of death in young adults, predominantly men.[1,2] Mortality due to TBI in Indonesia ranged from 6.2 to 11.2. [3] The incidence of TBI in Sanglah Hospital Denpasar was estimated to be more than 2000 cases per year, where 30% was moderate head injury and severe head injury.[4]

Traffic accidents were common causes of TBI, followed by persecution, and incidence falls from heights. The outcomes were also worse, which mostly needed long-term supportive rehabilitation.[5] Every year 1.6 million people in America suffer from a severe head injury, resulting in 52,000 deaths and 80,000 permanent neurological deficits. The high number of deaths due to head trauma was not only determined by the level of severity but also the accuracy and fast response to treatment. Accurate and fast treatment for head trauma can reduce mortality and disability.[3,6]

The target management of patients with severe head injury is to prevent secondary brain injury, this can be achieved by monitoring the intracranial pressure (ICP) and cerebral perfusion pressure (CPP) regularly. High ICP and low CPP were both related to worse final outcomes. High ICP is influenced by various factors such as hypoxia and type of lesion on head CT scan, while the low CPP is directly affected by hypotension and high ICP. This study aims to analyze the various risk factors that can lead to increasing of ICP and the related worse outcome.

METHOD

This is a prospective cohort study involving 42 patients with severe head injury and selected consecutively from June to October 2016. This study has been approved by the Local Ethical Committee in Udayana University. All subjects included in this study have been informed and consented to participate in this study.

There are three risk factors analyzed in this study, hypotension, hypoxia, and subarachnoid haemorrhage. Therefore, the subjects underwent manual blood pressure checking to obtain mean arterial blood pressure (MAP), blood gas analysis examination for hypoxia detection, and head CT scan to check any possibility of subarachnoid haemorrhage. The medical records were assessed

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for the gender and age information. Finally, intracranial pressure data was recorded. In this study, the author took 20 mmHg as ICP cut off point for high and low ICP pressure. The final outcome of the subjects were determined by the Glasgow Outcome Scale (GOS) determined upon discharge from the hospital.

All data was tabulated and presented in the table. The relationship between high ICP values with the final outcome was analyzed by using bivariate analysis with confidence interval (CI) 95%.

RESULT

This study includes 12 female (28.57%) and 30 male subjects (71.43%) with a mean age of 41 ± 18.035 years (age range 15-65 years) (Table 1). A total of 21 patients (50%) had hypotension and 21 patients had no hypotension (50%) with an average mean arterial pressure (MAP) 87.56 ± 26.05 mmHg. Most subjects had hypoxia condition (66.67%) and the proportion of hypotension was similar in both groups. Most subjects suffered from subarachnoid haemorrhage (SAH) (59.52%) (Table 1). High intracranial pressure (≥20 mmHg) was seen in 26 patients (61.9%) leading to 78.6% of subjects with unfavourable GCS scores (Table 1).

Table 1 Characteristics of subjects

Characteristic	Result
Gender	
Man	30 (71.43%)
Woman	12 (28.57%)
Aged	
15-25 y.o	13 (30.95%)
25-45 y.o	10 (23.81%)
45-65 y.o	19 (45.24%)
Risk factor	
Hypotension (+) (MAP ≤ 70 mmHg)	21 (50%)
Hypotension (-) (MAP > 70 mmHg)	21 (50%)
Hypoxia (+)	28 (66.67%)
Hypoxia (-)	14 (33.33%)
SAH (+)	25 (59.52%)
SAH (-)	17 (40.48%)
Average ICP pressure	
High ICP (≥20 mmHg)	26 (61.9%)
Low ICP (<20 mmHg)	16 (38.1%)
Temporary GOS	
Favourable (GOS 4-5)	9 (21.43%)
Unfavourable (GOS 1-3)	33 (78.57%)

From the bivariate analysis, we found two factors that significantly affected the risk of ICP increased, they were hypotension (RR 0.27; 95CI 0.095-0.775; p<0.001) and hypoxia (RR 0.125; 95CI 0.034-0.457; p<0.001). (Table 2). It was significant that high ICP value ≥ 20 mmHg related to the unfavourable outcome based on GOS scale parameter (P: 0.000; RR: 2.286; 95% CI: 1.31 to 3.98) (Table 3).

DISCUSSION

Cerebral blood flow <60 mL/100g/min causes decreased protein synthesis of brain tissue. At a later stage, the brain will enter the area penumbra where the process of accumulation of lactic acid as a result of glycolysis begains leading to cytotoxic oedema and later an increase of ICP. If CBF has decrease <20 mL/100g/min, it will lead to irreversible brain cell damage.[7] Therefore, therapeutic targets for severe head injury focus on preventing secondary brain injury, by maintaining adequate CPP, decreasing ICP, and increasing MAP. In this condition, hypotension can decrease cerebral blood flow (CBF), reducing blood supply to the brain that will lead to ischemia, as well as hypoxia.

Hypotension (systolic blood pressure <90 mm Hg) is one of five major predictors of the final outcome. A single episode of hypotension is associated with increased morbidity and mortality compared to patients without hypotension. The relative risk of death increases in the case of 2-3 episodes of a patient with hypotension, so the repetition of episodes of hypotension is closely related to mortality.[8]

Hypoxia describes the brain tissue ischemia, in which the oxygen supply to the brain is decreased (PaO2 <40 mmHg). In hypoxic conditions, there is a failure of energy production, oxidative phosphorylation is inhibited resulting in decreased production of ATP, the failure of active transport of Na^+/K^+ ATPase lead sodium enter intracell (sodium influx) and potassium diffuses out from intracell (potassium efflux), resulting in depolarization of the cell membrane and the opening of Ca^{2+} channels causing massive calcium influx. Intracell calcium levels will be excessive, causing activation of phospholipase and proteolysis, and hydrolysis of membrane phospholipids and an increase in free fatty acids, resulting in irreversible damage to the cell membrane. Damage to the cell membrane will lead to cerebral oedema and increased ICP.[7,9]

Patients with a severe head injury who have experienced hypoxemia are significantly associated with an increased risk of mortality and morbidity. Patients with the saturation of oxygen <60% have a 50% mortality rate and all who survived suffered from a severe disability. The duration of hypoxemia is an independent predictor of mortality

Table 2 Relative risk each Component Risk Factors Against Increased ICP

Risk Factor	ICP ≥20 mmHg (n = 26)	ICP < 20 mmHg (n = 16)	Total	P	RR	95% CI
Hypotension						
Yes	18	3	21	0.001*	0.271	0.095 – 0.775
No	8	13	21			
	26	16	42			
Hypoxia						
Yes	26	2	28	0.000*	0.125	0.034 – 0.457
No	0	14	14			
	26	16	42			
SAH						
Yes	17	8	25	0.324*	0.765	0.435 – 1.344
No	9	8	17			
	26	16	42			

*Chi-square

Table 3 Correlation ICP and the final outcome

	Final outcome (*GOS*)					95% CI	
	Favourable (n = 9)	Unfavourable (n = 33)	Total	P	RR	Lower	Upper
ICP ≥ 20 mmHg	0	26	26	0.000*	2.286	1.311	3.984
ICP < 20 mmHg	9	7	16				
	9	33	42				

*Chi-square. Significant if p < 0.05.

(SaO2 ≤ 90%; median duration of hypoxemia between 11.5 to 20 minutes).[8]

High ICP (≥ 20mmHg) produces the worst final outcome. A study showed that the increase of ICP associated with an increased odds ratio (OR) of deaths in which OR: 3.5 (95% CI: 1.7-7.3) for ICP 20-40 mmHg, and OR 6.9 for the value of ICP > 40 mmHg (95% CI: 3.9-12.4).[10] Our research shows that the value of ICP ≥20 mmHg produces the worst final outcome/unfavourable (RR 0.038L 95CI 0.006-0.263; p<0.001), where the majority of patients with increased ICP also suffered from hypotension (MAP ≤70 mm Hg). In comparison, from the 28 patients with hypoxia, 26 patients (92.86%) had high ICP (Table 2).

The decrease in CPP occurred in 80% of patients with severe head injury who experienced an increase of ICP> 20 mmHg during the first 5 days post-trauma, but more responsive to the decrease the CPP is a low value of MAP (MAP <80 mmHg).[11] The mortality rate in patients with a severe head injury 39% increased in group patient with MAP <60 mmHg (Mann-Whitney U-test, P <0.0001).[12] Other studies have shown that 100% mortality occurred in severe head injury subjects with MAP <70 mmHg in the first 24 hours after trauma.[13,14]

In this study, some subjects showed overlapping of risk factors, where subjects with hypotension also experienced hypoxia (54.55%). This group also has high-value ICP (≥20 mmHg) and had an unfavourable final outcome. Hypoxia and hypotension affect each other and led to an ICP increase. This is also evidenced in another study which showed that hypoxia increased the mortality rate up to 6% and hypotension up to 33%, while the combination of hypoxia and hypotension increased mortality up to 48%.[12]

Subarachnoid haemorrhage is one of the predictors of poor outcome in patients with a severe head injury. The existence of SAH on head CT scan indicated the occurrence of vasospasm, which causes cerebral oedema. The final outcome of patients with traumatic SAH is significantly worse than patients without SAH. An unfavourable outcome is seen in 60% of patients with SAH, compared with patients without SAH (P <0.001).[15] In our study, only 17 of 25 patients (68%) with SAH had an increased ICP, and the results of statistical tests showed no significant relationship between SAH and an increased ICP (P: 0.324; RR: 0.765; 95% CI: 0.435 -1.344). This is due to our samples with SAH mostly being grade 1 and 2 (according to Fisher Scale for

Grading SAH on Admission CT Scan).[16] From another SAH grouping in which group 1 limited to traumatic SAH and group 2 for traumatic SAH in other parts of the brain. From group above, the end result is poorly seen at 2 groups (36.7% vs. 7.4%; OR: 8:56; 95% CI: 1.11-66.20; p = 0.0396), so it can be concluded that the extent and location of SAH affect the improvement of ICP and final outcome.[17] Both studies showed that SAH was associated with poor outcome.

CONCLUSION

Hypoxia and hypotension were two risk factors that significantly influenced the increase of ICP, where high ICP caused the unfavourable outcome.

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