EVALUATION OF TRAUMATIC BRAIN INJURIES DUE TO ALCOHOL INTOXICATION: STUDY IN CENTRAL HOSPITAL IN WEST JAVA

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ABSTRACT

Background: *Alcohol intoxication is one of the predisposing factors for the incidence of traumatic brain injury (TBI). At present, there are no hospital-based specific data that provide TBI prevalence rates due to alcohol intoxication, especially in West Java.* ***Objective:*** *This study aims to make descriptive observations about the incidence of TBI due to alcohol intoxication in one of the central referral hospitals in West Java.* ***Method:*** *A retrospective cross-sectional study was carried out involving 10,662 TBI patients recorded in the emergency department database and the Department of Neurosurgery daily case database in the period 2012 to 2018. All data relating to the two items were analyzed. Alcohol intoxication is known from a history of autoanamnesis, alloanamnesis, and the subjective identification of the examiner of alcohol odor on the breath in TBI patients who enter the emergency room. Statistical cross-tabulations were performed to present clinical and demographic phenomena in TBI cases due to alcohol intoxication.* ***Results:*** *The analysis showed that the prevalence of alcohol intoxication in TBI cases was 4.77% (N = 507), all of which were due to motor vehicle accidents. Mean age of 26.44 ± 9.32 years (median 24 and range 12-64). The predominant age in TBI cases due to alcohol intoxication was in the age range of 20-30 years (44.2%), and male sex 92.7%. Most cases occurred in May (12%) and on Saturdays. Based on the consciousness assessment, initial Glasgow Coma Scale in emergency department were 14 (35.9%) and classified as mild TBI (69.2%). Subarachnoid hemorrhage and cerebral contusion are predominantly lesions found on CT scans of patients (2.2%), anterior skull base fractures of 8.1%, and linear fractures (5.1%) are the dominant extracranial lesions. Soft tissue lacerations are found in 16% of all cases. Craniotomy surgery was performed in 3.6% of patients for epidural, intracerebral, and subdural hemorrhage lesion evacuation as well as correction of depressed skull fractures. About 45% were treated for a minimum of three days to observe changes in their consciousness status before discharge.* ***Conclusion:*** *Alcohol consumption before the head injury in motor vehicle accidents has a relationship with various complications. Alcohol intoxication is a complex issue, and more research needs to be done to create comprehensive prevention programs and regulations.*

***Keywords**: Alcohol, Intoxication, traumatic brain injury, prevalence*

ABSTRAK

Latar Belakang: Intoksikasi alkohol merupakan salah satu faktor predisposisi kejadian cedera otak traumatika (COT). Saat ini belum ada data spesifik berbasis rumah sakit yang menyediakan angka prevalensi COT akibat intoksikasi alkohol, khususnya di Jawa Barat. **Tujuan:** Penelitian ini bertujuan untuk melakukan observasi deskriptif tentang kejadian TBI akibat intoksikasi alkohol di salah satu rumah sakit rujukan pusat di Jawa Barat. **Metode:** Penelitian potong lintang retrospektif dilakukan dengan melibatkan 10.662 pasien COT yang tercatat di basis data instalasi gawat darurat dan basis data laporan kasus harian Departemen Bedah Saraf pada periode 2012 hingga 2018. Semua data terkait kedua hal tersbut dianalisa. Intoksikasi alkohol diketahui dari riwayat *autoanamnesis*, *alloanamnesis* dan identifikasi subjektif pemeriksa terhadap bau napas alkohol pada pasien COT yang masuk ke IGD. Tabulasi silang secara statistik dilakukan untuk menyajikan fenomena klinis dan demografis pada kasus COT akibat intoksikasi alkohol. **Hasil:** Hasil analisis menunjukkan bahwa prevalensi intoksikasi alkohol pada kasus COT adalah 4,77% (N=507), semuanya karena kecelakaan kendaraan bermotor. Rerata usia 26,44 ± 9,32 tahun (median 24 dan rentang 12-64). Dominasi usia pada kasus COT akibat intoksikasi alkohol adalah pada rentang usia 20-30 tahun (44,2%); dan jenis kelamin laki-laki 92,7%. Kasus terbanyak terjadi pada bulan Mei (12%) dan pada hari Sabtu. Berdasarkan penilaian kesadaran, pasien yang masuk ke IGD memiliki skor GCS 14 (35,9%) dan dikelompokan sebagai COT ringan (69,2%). Perdarahan subaraknoid dan kontusio serebral merupakan dominasi lesi yang ditemukan pada *CT scan* pasien (2,2%), fraktur basis tengkorak anterior sebanyak 8,1% dan fraktur linier 5,1% merupakan lesi ekstrakranium yang dominan. Laserasi jaringan lunak ditemukan sebanyak 16% dari keseluruhan kasus. Dilakukan tindakan operasi kraniotomi pada 3,6% pasien untuk evakuasi lesi perdarahan epidural, intraserebral dan subdural maupun koreksi fraktur depresi kranium Sekitar 45% dirawat selama minimal tiga hari untuk observasi perubahan status kesadarannya sebelum dipulangkan. **Kesimpulan:** Konsumsi alkohol sebelum cedera kepala pada kecelakaan kendaraan bermotor memiliki hubungan dengan berbagai komplikasi. Intoksikasi alkohol merupakan isu yang kompleks dan perlu dilakukan lebih banyak penelitian untuk membuat program dan regulasi pencegahan yang komprehensif.

***Kata kunci:** Alkohol, Intoksikasi, cedera otak traumatika, prevalensi*

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INTRODUCTION

Traumatic brain injury (TBI) is still one of the global health problems. The data

shows that in the United States, every year, 50,000 patients die from TBI. The main mechanisms causing TBI are falls and motor vehicle accidents, which contribute at least 1.4 million cases of TBI per year. It has an impact on economic spending due to TBI reaching 9-10 million dollars per year to finance the acute phase of care and rehabilitation of TBI patients.[1] Thus indirectly, this TBI case has an impact on the country's considerable economic expenditure.

Based on 2006 data, alcohol intoxication accounted for 32% of fatal motor vehicle accidents in the USA, half of which died before being taken to hospital. In general, about 35-81% of TBI patients are due to alcohol intoxication.[1] The incidence of TBI in Europe reaches 83.3 per 100,000 patients and specifically in the UK, mortality due to TBI reaches 6-10 per 100,000 population per year.[2]

Intoxication due to alcohol consumption is a strong predisposition to TBI.[2] The patient's prognosis TBI due to alcohol intoxication depends on the degree of complications of the patient and the pattern of intoxication that occurs.[2] Besides being related to the prognosis and complications that occur, other difficulties are related to the establishment of the diagnosis itself. This is because TBI due to alcohol requires an immediate assessment and intervention as early as possible to assess the actual damage that occurred or changes in consciousness "vague" due to alcohol consumption.

The absence of specific hospital-based data in Indonesia related to alcohol intoxication and the incidence of TBI requires special attention because alcohol intoxication is one of the complex problems that is directly related to the long-term effects of brain injury and is likely to occur in the younger generation as a future capital for developing nation. This study aims to determine the prevalence of TBI due to alcohol intoxication that occurred at the central referral hospital in West Java.

RESEARCH METHOD

This study used a retrospective cross-sectional design that analyzed secondary data from 10,622 traumatic brain injury patients who entered the hospital emergency department (ED) from January 1st 2012 to December 31st 2018. The data used were obtained from a database of daily reports of patients in the Department of Neurosurgery according to the patient's medical record at admission and initial management when the patient is in the hospital emergency room. All TBI cases included in the period were selected according to the inclusion and exclusion criteria set. As many as 507 TBI cases were obtained with a history of alcohol intoxication prior to a motor vehicle

accident and resulted in TBI in the study subjects. The inclusion criteria of the study were TBI patients, had initial GCS values while in the emergency room, had a history of alcohol consumption before experiencing trauma, computed tomography scanning (CT Scan) of the head as indicated. The exclusion criteria are TBI patients with a history of alcohol consumption who died before arriving at the hospital emergency room and TBI patients who, after being in the emergency room, then went home of their own volition or refused all types of actions or examinations.

In this study, the measurement of blood alcohol concentration (BAC) was not carried out on the examination of the patient's blood serum or examination of a whole blood analysis (gas chromatography) or breath analysis to determine alcohol content. History of alcohol consumption is known only based on autoanamnesis, alloanamnesa, or an objective examination of the patient's alcohol-odor breath when first examined.

Assessment of awareness status was done using the Glasgow Coma Scale (GCS) by the neurosurgeon at the emergency room when the patient first entered. Observation of consciousness status was carried out while the patient was managed in the emergency room and re-assessment of awareness status. A computed tomography scan of the head (CT scan) is performed according to indications of consciousness status, damage to the anatomical structure of the head and face or other indications that indicate a CT scan of the head when the patient is managed in the emergency room such as seizures, vomiting, or a history of lucid intervals prior to hospital. TBI is classified as mild, moderate, and severe according to GCS assessment. TBI is mild if GCS 14-15, moderate if GCS 9-13 and severe if GCS 3-8.

We used a statistical analysis using cross-tabulation and calculation of the prevalence of alcohol intoxication associated with TBI. All patient demographic data, including age, gender, age group, and the results of clinical and radiological examinations of patients, were analyzed. The results of the analysis that are statistically observed are interesting case phenomena presented in graphs and tables.

RESULT

The results of this study indicate that in the period of January 1st 2012 to December 31st 2018, there were 10,622 cases of TBI that were managed at the Emergency Department (ED) of the hospital where the study was conducted. In accordance with this number, 507 cases of TBI were found, accompanied by a history of alcohol intoxication before a two-wheeled motor

vehicle accident in the vicinity of West Java and was referred to the emergency room. The mean age of the patients was 26.44 ± 9.33 years (median 24 years; age range 12 - 64 years).

Table 1. Comparison of the Impact of TBI due to alcohol intoxication in a motor vehicle accident in West Java

Description	Drivers	Passengers
TBI:		
Mild	324 (63.9%)	27 (5.3%)
Moderate	53 (10.5%)	93 (18.3%)
Severe	0	10 (2%)
Extra-cerebral Lesions		
None	294 (58.0%)	94 (18.5%)
Anterior basilar skull fracture	33 (6.5%)	8 (1.6%)
Middle basilar skull fracture	10 (2.0%)	8 (1.6%)
Anterior and middle basilar skull fracture	2 (0.4%)	2 (0.4%)
Depressed skull fracture	11 (2.2%)	15 (3.0%)
Anterior-middle basilar skull fracture and linear fracture	7 (1.4%)	3 (0.6%)
Depressed skull fracture and basilar skull fracture	3 (0.6%)	4 (0.8%)
Others	5 (1.0%)	8 (1.6%)
Intra-cranial lesions		
None	344 (67.9%)	105 (20.7%)
Epidural hemorrhage	0	5 (1.0%)
Subarachnoid hemorrhage	6 (1.2%)	4 (0.8%)
Subarachnoid hemorrhage + Cerebral contusions	4 (0.8%)	7 (1.4%)
Cerebral contusions	4 (0.8%)	7 (1.4%)
Epidural hemorrhage + Pneumocephalus	1 (0.2%)	1 (0.2%)
Subdural hemorrhage+ Cerebral contusions + Subarachnoid hemorrhage	0	4 (0.8%)
Subarachnoid hemorrhage + Intraventricular hemorrhage	1 (0.2%)	1 (0.2%)
Intracerebral hemorrhage	1 (0.2%)	1 (0.2%)
Intracerebral hemorrhage + Intraventricular hemorrhage	1 (0.2%)	1 (0.2%)
Epidural Hemorrhage + Cerebral contusions	1 (0.2%)	1 (0.2%)
Others	3 (0.6%)	4 (0.8%)
Other multiple injuries accompanying TBI:		
None	228 (45.0%)	93 (18.3%)
Facial skin laceration	66 (13.0%)	15 (3.0%)
Facial bone fracture	22 (4.3%)	13 (2.6%)
Limb fracture	30 (5.9%)	10 (2.0%)
Clavicle fracture	5 (1.0%)	3 (0.6%)
Traumatic optic neuropathy	7 (1.4%)	4 (0.8%)
Lung contusions	4 (0.8%)	2 (0.4%)
Abdominal trauma	3 (0.6%)	2 (0.4%)

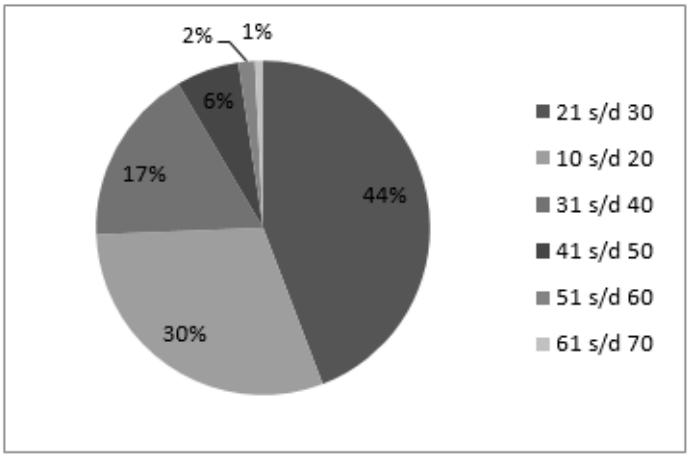

Figure 1. Age-group TBI patients due to alcohol intoxication that has a motor vehicle accident (Large to small clockwise).

TBI and alcohol intoxication were dominated by male sex as many as 470 (92.7%) patients and 37 women (7.3%). The dominant age group in TBI due to alcohol intoxication is age 21-30 years as many as 224 (44.2%) cases and aged 10-20 years as many as 153 (30.2%) cases (Figure 1). When the patient was taken to the ED of the hospital, the initial GCS assessed were 351 (69.2%) mild TBI (GCS 14-15), 146 (28.8%) moderate TBI (GCS 9-13) and severe TBI (GCS ≤8) 10 (2%) cases.

The predominance of TBI due to alcohol intoxication was on Saturday (26.6%) (Figure 3), and in May (12%) (Figure 4). The incidence of TBI due to alcohol intoxication also results in intracranial, extracranial, and even multi-traumatic lesions involving other body compartments (thorax, abdomen, and extremities) [See Table 1].

The majority of TBI sufferers due to alcohol intoxication are involved in two-wheeled motor vehicle accidents; 365 riders (72%) and 130 passengers (28%). TBI due to alcohol intoxication accounted for at least 29 (5.7%) cases of single intra-cranial lesions (epidural bleeding only, sub-dural bleeding, intracerebral hemorrhage, subarachnoid hemorrhage, and others); 21 (4.14%) multiple lesions, 1.38% triplet lesions and 0.19% quartet lesions in one patient (Figure 2). The predominance of extracranial lesions (on the skull and around the face) is 5.1% linear fractures on the cranial bones, 2.5% depressed fractures on the cranial bones. Tear injuries on the face reached 15%, followed by fractures on the facial bone 7.88%. Involving body compartments other than TBI due to alcohol intoxication are limb fractures (5.91%), clavicle fractures (2.16%), pulmonary contusions, and abdominal trauma,

respectively 1.18% and 0.98%. Another damage specific to TBI related to alcohol intoxication is the base fracture of the anterior skull fossa is 8.08%, and the media fossa is 3.5%, and a combination of both 0.78% (Table 1).

The results of this study also showed that there were 3.6% of cases that underwent craniotomy for indications of intracranium lesions (confirmation of CT head scan results) and/or extracranial lesions. Of the 18 cases that underwent craniotomy surgery, 9 cases were due to epidural hemorrhage, 4 cases were due to subdural bleeding (accompanied by cerebral contusions, and subarachnoid hemorrhage), the rest were due to complications from epidural bleeding and pneumocephalus (2 cases); intracerebral hemorrhage (2 cases); 1 case was due to multiple lesions ranging from epidural, subdural, subarachnoid hemorrhage and intracerebral hemorrhage in the same patient (Table 2). A total of 228 patients (45%) of cases had to undergo treatment for at least three days in the hospital; the rest were sent home for a doctor's permission or went back on their own. The prediction of financing on the management of TBI patients due to alcohol intoxication per day (± 3.5 million Rupiah) has increased sharply 10-fold if coupled with operative measures (detailed data not presented).

DISCUSSION

Alcohol intoxication is a major predisposing factor in traumatic brain injury.[2,6,10] Alcohol concentration was detected in 35-50% of patients diagnosed with TBI who had previously consumed alcohol.[6.8] Alcohol consumption is also known to be a risk factor for death in TBI patients.[7]

The results of this study indicate that in the period 2012-2018, there were 507 cases of head injuries (mild, moderate, and severe) caused by alcohol intoxication before a motor vehicle accident. Of these, surface lesion damage was recorded at 23.47%, intracranium damage (various types of lesions) of 11.44%; and even 36.69% of multiple damages to parts of the body other than the head. As many as 3.55% of cases (either as drivers or as passengers) underwent emergency craniotomy for the evacuation of various types of lesions ranging from epidural bleeding, subdural bleeding, cerebral contusions, subarachnoid hemorrhage, intracerebral hemorrhage or even intra-ventricular hemorrhage.

It was also known that when the patient first arrived at the emergency room, the patient's consciousness status based on GCS obtained a mild TBI (GCS 14-15) of 351 (69.2%) (Table 2). However, this initial GCS assessment is not easy to do because it must be sorted out really whether the cause of GCS decline is purely due to TBI or the

influence of alcohol content. Therefore, the Shahin[2] study recommends that the best GCS assessment be carried out 6 hours after the incident. GCS assessment before 6 hours does not help in management, and the best recommendation is to check blood alcohol concentration. When TBI is mild, the signs of alcohol intoxication will be the same as for TBI patients without a history of alcohol consumption, and this will complicate the diagnosis.[2,4]

The dominance of cases at the age of 21-30 years (44%) in this study has the same trend as the results of the Bernier study (2016).[10] In the 10-year retrospective study, it was noted that the dominance of TBI cases due to alcohol intoxication which had a dominant motor vehicle accident at age 18-30 years with a case incidence reaching 65% of all TBI cases. In that study, the main mechanism of TBI was a motor vehicle accident after alcohol consumption by 54.26%; while the results of this study show the figure reached 100% due to motor vehicle accidents.

The results of this study also showed that the trend in the incidence of TBI cases due to alcohol intoxication in these seven years was dominated by cases that occurred on Saturdays and the highest in May. It cannot be explained fundamentally why this happened. The Bernier study also noted a unique phenomenon in 18 years of alcohol-related head injury studies that the highest incidence of TBI was at 24.00 midnight to 04.00 dawn.[10] The only possible explanation is that this pattern reflects the context in which individuals prefer alcohol consumption at that time. Saturday's dominance could also be related to the social events of the residents of the study site, especially the related age generation (21-30 years) in passing the time off from routine activities on Saturdays.

After a primary injury to the brain, subsequent sequelae injury follows such as edema, hematoma, increased intracranial pressure, decreased cerebral perfusion, or decreased cerebral oxygenation. All of these conditions will affect the effects of long-term complications in TBI patients due to alcohol intoxication.[4] This study did not factually record the incidence of secondary injuries following primary TBI.
In general, the management of patients with alcohol intoxication with TBI requires rapid transportation to the hospital, early intubation, adequate resuscitation, early CT head scan, rapid evacuation of intracranial lesions (such as EDH or SDH) and intensive care for indicated patients. With this step, it will help reduce mortality in patients with severe TBI due to alcohol intoxication. The results of this study noted that there were 18 cases of severe TBI related to alcohol

intoxication and had to undergo operative craniotomy. The state of this severe TBI is unknown, whether it is a direct result of the TBI itself or delays and errors in the initial management of patients outside the hospital. There are no specific data related to this problem.

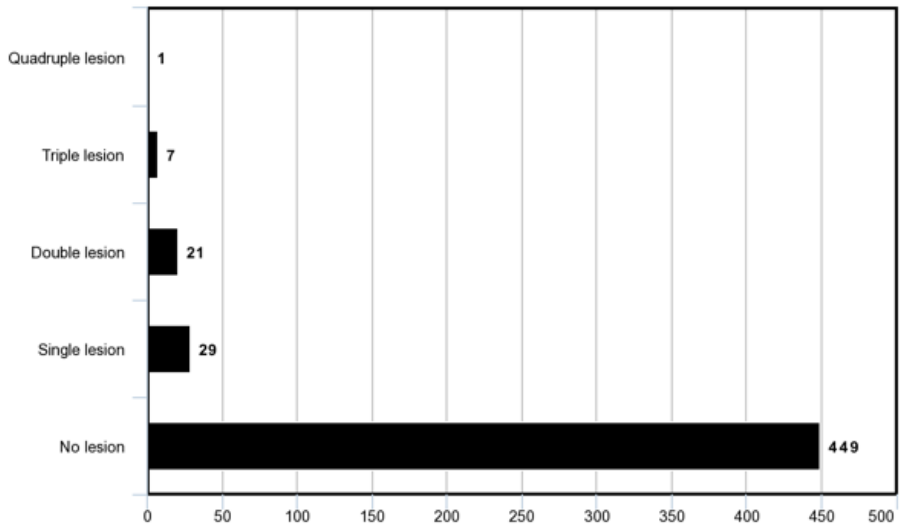

Figure 2. Number of intracranial lesions on TBI due to alcohol intoxication (persons)

Alcohol intoxication has an important role in the incidence of TBI.[1,2] In various clinical studies, it is known that patients who experience TBI due to alcohol intoxication are usually treated and require mechanical intubation to support respiratory failure that is experienced.[3] Other complications are poor clinical outcomes, especially related to levels of very high blood alcohol or those classified as chronic alcohol users.[3] This study did not identify the number of alcohol users classified as chronic alcohol consumers.

This study also did not measure alcohol levels quantitatively even though many patients who came classified as severe TBI. However, in the Opreanu (et al) study at low and moderate doses (< 1 g/kgBW or 100 mg/dL, around 0.1%), alcohol consumption causes impaired motor deficits and mild cognitive impairment. Whereas at high doses (> 3 g/kgBW or 200 mg/dL) causes mainly respiratory failure, increased lactic acid in the brain, and decreased blood flow. This study also did not measure alcohol levels quantitatively even though many patients who come classified as severe TBI. Other damage mechanisms that occur at high doses are hemodynamic changes, including increased mean arterial pressure (MAP) and cerebral blood flow (CBF). Both of these will end in an increase in high intracranial pressure and brain edema to changes in neurobehavior function.

The effect of alcohol on TBI occurs very quickly; it can be physiological effects such as respiratory depression or decreased cerebral perfusion or also biomolecular changes that occur in changes in neuronal receptor function and membrane function.[3] Alcohol also causes osmotic pressure shifts, so cells become swollen. Changes in enzymatic binding in the cell membrane through abnormal free radical reactions caused by the presence of alcohol will have an impact on increasing the acceptability of damage to the cell membrane. The latest evidence in laboratory studies proves that alcohol will cause potentiation effects on TBI by changing cerebral perfusion.

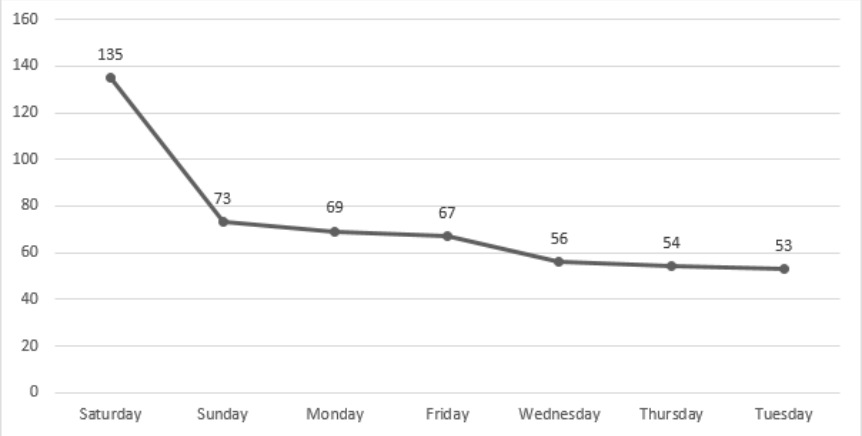

Figure 3. The dominant distribution of TBI due to alcohol intoxication (persons)

Table 2. Types of TBI Impact Lesions due to alcohol intoxication by operative measures

	Num.
TBI Classification:	
Mild	0
Moderate	16
Severe	2
Intracranial Lesions	
Epidural hemorrhage	9
Subdural hemorrhage	4
Epidural hemorrhage + Pneumocephalus	2
Intracerebral hemorrhage	2
Epidural + Subdural + Subarachnoid + Intracerebral hemorrhage	1
Extracranial Lesions	
Depressed skull fracture	7
Multiple lesions belong to intracranial lesions	11
Other multiple injuries accompanying TBI	
Facial bone fracture	3
Limb fracture	3
Multiple lesions belong to intracranial lesions	12

Speaking in terms of biomolecular, a high alcohol consumption activates the inflammatory response in the brain by activating microglia and pro-inflammatory compounds, which in clinical appearance can be assessed by observing changes in cognitive deficits and signs of general inflammation.[5] Cognitive deficits in TBI patients are related to more alcohol intoxication in TBI moderate and severe.[9] This cognitive deficit can last for several years and has a negative impact on the reintegration of patients after TBI treatment (to school, work or playground).[9]

The use of intubation and mechanical ventilation with oxygen support in the intensive care room certainly has an impact on increasing health financing, which is becoming increasingly expensive. In this study, there were at least ten patients with severe TBI classification (GCS ≤ 8), thus requiring mechanical intubation and respiratory support. All these patients are motorized vehicle passengers. In the case of TBI, the result may be due to the lower level of alertness and the reflex response of the body compared to the driver in the event of a motor accident.[8,10]

Primary serious damage can occur due to two main phenomena in the nerves, namely necrosis or degeneration of nerve tissue and swollen brain tissue, which also ends in nerve tissue necrosis. In the next stage, patients with alcohol intoxication and TBI will have a risk of secondary brain damage through a mechanism of decreasing cerebral blood flow, and alcohol metabolism products which decrease the stability of capillary membrane and ultimately cause widespread damage.[4,6]

Long-term consequences as a result of alcohol intoxication before TBI during TBI and after TBI are very difficult to predict.[8] This is especially true for TBI patients who are classified as mild because often the long-term evaluation will only involve moderate and severe TBI patients.[8] Some of the problems found related to the management of alcohol intoxication patients who experience TBI in the first-line emergency team are: (1) not many nurses or emergency physicians have awareness in early recognizing patients who have decreased consciousness due to alcohol intoxication along with the accompanying intra-cranial lesions; (2) there is no practical written clinical guideline available for physicians or health-care workers in the ED regarding case recording, early management and data collection of TBI patients with alcohol intoxication.[2] The patients analyzed in this study also included patients from previous hospital referrals who did initial management of the patient.

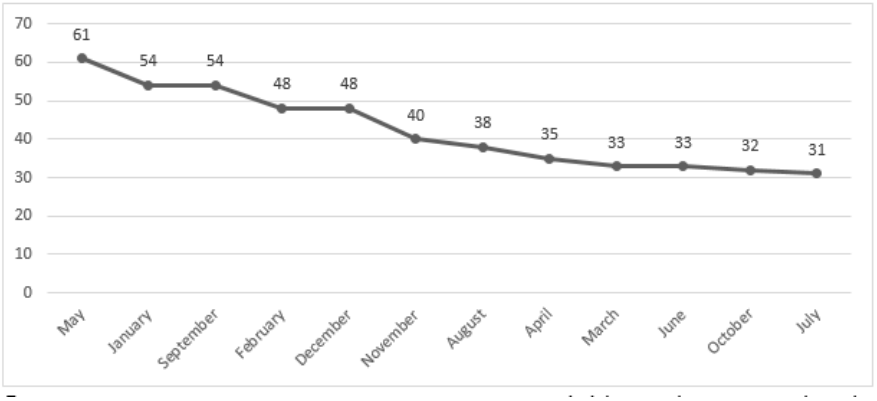

Figure 4. The distribution of TBI due to alcohol intoxication in a year

Although this study is the result of a study at a local institution in Indonesia, this hospital is one of the regional referral hospitals in West Java, so that the characteristics of patients analyzed can represent the population of TBI patients that occur due to alcohol consumption and intoxication. Some drawbacks in this study are that the concentration of alcohol in the blood of the patient is not measured, so it is difficult to draw a clinical correlation between alcohol levels, the risk of TBI, and clinical abnormalities that may arise in patients. Subjective examinations through the scent of alcohol are not included in routine clinical procedures, although they help lead to the diagnosis of risk factors. The subjective examinations will inevitably lead to this problem: when TBI patients who consume locally produced alcoholic drinks are odorless and are not accompanied by another person when the TBI patient is brought to the emergency department, it ultimately complicates the history taking.

CONCLUSION

Alcohol intoxication has the potential to cause TBI in motor vehicle accident victims. The results of recording cases of TBI due to alcohol intoxication are significant to contribute to the establishment of various policies and regulations related to alcohol consumption and measures to prevent excessive alcohol consumption.

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REFERENCES

1. Oprenau RC, Donald K, and Marc B. Influence of alkohol on mortality

in traumatic brain injury. J Am Coll Surg 2010, 210 (6): 997-1007.

2. Shahin H and Claudia R. Alkohol and the head-injured patient. Trauma, 2012; DOI:10.1177/1460408611434382
3. Cunningham RM, Ronald FM, Elizabeth MH, and Brian JZ. The effects of alcohol on head injury in the motor vehicle crash victim. Alkohol & Alcholism, 2002, 37(3): 236-240.
4. Miller TW and Eunell BG. Head injury in the presence of alcohol intoxication. Int J Trauma Nurs, 1997, 3:50-55.
5. Weil ZM, Corrigan JD, Karelina K. Alcohol use disorder and traumatic brain injury. Alcohol Res: Current Rev. 2018;39(2):171-180.
6. Mathias JL, Osborn AJ. Impact of day-of-injury alcohol consumption on outcomes after traumatic brain injury: A meta-analysis. Neuropsychological Rehab. 2018;28(6):997-1018.
7. Lin H, Lin T, Soo K, Chen C, Kuo L, Lin Y, et al. The effect of alcohol intoxication on mortality of blunt head injury. BioMed Res Int. 2014; DOI:10.1155/2014/619231.
8. Silverberg ND, Panenka W, Iverson GL, Brubacher JR, Shewchuck JR, Heran MKS, et al. Alcohol consumption does not impede recovery from mild to moderate traumatic brain injury. JINS. 2016;22:816-827.
9. Herrold AA, Sander AM, Wilson KV, Scimeca LM, Cobia DJ, Breiter HC. Dual diagnosis of traumatic brain injury and alcohol use disorder: characterizing clinical and neurobiological underpinnings. Curr Addict Rep. 2015;2:273-284.
10. Bernier RA, Hillary FG. Trends in alcohol use during moderate and severe traumatic brain injury: 18 years of neurotrauma in Pennsylvania. Brain Injury. 2016;30(4):414-421