



Research article

Altered mental status in moderate-severe traumatic brain injury in Indonesia: the clinical manifestation and EEG features of non-convulsive status epilepticus

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ABSTRACT

Introduction: Moderate-to-severe traumatic brain injury (msTBI) can cause non-convulsive status epilepticus (NCSE). Electroencephalography (EEG) is employed as a diagnostic tool due to the non-specificity of clinical symptoms. This study aimed to identify clinical and EEG features related to NCSE in patients with msTBI.

Methods: This was a cross-sectional study. Suspected NCSE in msTBI was examined using EEG data collected in consecutive patients from January 2017 to December 2019 at Dr. Cipto Mangunkusumo Hospital, Jakarta. Diagnoses of NCSE were made based on clinical manifestations and EEG features using the modified Salzburg Consensus Criteria for NCSE (mSCNC).

Results: Of the 39 msTBI patients, 19 were diagnosed with NCSE; only two fulfilled the definitive criteria, and the remaining were possible NCSE. Delirium and perceptual impairment were only found in NCSE, while psychomotor agitation was higher (12.8% vs. 5.1% in NCSE vs. non-NCSE). The most common EEG feature was rhythmic activity (>0.5 Hz) without fluctuation, which improved with anti-epileptic drug administration. The Glasgow Coma Scale (GCS) score at onset and at hospitalisation discharge was significantly lower in patients with NCSE. The lesions in NCSE mostly originated from the temporal lobe. Injury to the temporal lobe had a significant relationship with NCSE occurrence ($p = 0.036$, odds ratio 11.45 [95% confidence interval 1.17–111.6]).

Discussion: Post-traumatic NCSE can manifest as an alteration in mental status that could lead to missed diagnosis. In this study, delirium, perceptual impairment, and psychomotor agitation were confirmed as NCSE using EEG. The most common discharge originated from the injured temporal lobe, and this site was a significant factor associated with NCSE in patients with msTBI.

Conclusion: NCSE can be found in msTBI cases with clinical manifestations of altered mental status, psychomotor agitation, and hallucination. An injured temporal lobe was a susceptible site for the development of NCSE.

1. Introduction

Post-traumatic seizure is the most common complication in 20%–35% of all traumatic head injuries, and manifests as convulsive or non-convulsive seizures [1, 2, 3]. Post-traumatic seizures are caused by a series of primary or secondary injury processes and are classified as immediate (≤ 24 h), early (≤ 7 days), or late onset (> 7 days). Approximately 22% of the patients with moderate-to-severe traumatic brain injury (msTBI) were found to experience seizures, 52% of whom experienced non-convulsive seizures [4]. Non-convulsive seizures were found in 23.5% of patients with msTBI, and 5.9% of these cases evolved into non-convulsive status epilepticus (NCSE) [2].

NCSE is a non-motor seizure that manifests as a deterioration of consciousness, alteration of mental status, automatism, behavioural changes, or hallucination [5]. Therefore, this condition may be overlooked by many clinicians. Electroencephalography (EEG) tests are important for determining epileptic seizures and foci. The modified Salzburg consensus criteria for non-convulsive status epilepticus (mSCNC) is a validated guideline criterion for the diagnosis of NCSE [6]. Delay in diagnosis is associated with the occurrence of prolonged seizures and higher morbidity and mortality rates.

Few studies have been published on NCSE and msTBI in Asia, especially in Indonesia. This study aimed to identify the clinical manifestations and EEG features associated with NCSE in msTBI.

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2. Material and methods

This was a cross-sectional study conducted on patients with msTBI at the Dr. Cipto Mangunkusumo General Hospital. Clinical and EEG data were collected for patients with msTBI who were suspected of having NCSE from 2017 to 2019. Clinical manifestations that were highly suspicious of NCSE symptoms in patients with msTBI were altered mental status, psychomotor agitation, illusion/hallucination, cognitive impairment, and behavioural or thought content changes. These symptoms were suspected to be of NCSE if they occurred in msTBI patients who had already exhibited improvement of consciousness and laboratory parameters (e.g., leukocytes, imaging on brain computed tomography [CT] scan).

The inclusion criteria were as follows: patients with msTBI, age >18 years, those who had undergone a brain CT scan and EEG test, and for whom informed consent from the family could be obtained. The exclusion criteria were unstable vital signs, history of intracranial tumour, infection, stroke, metabolic impairment, history of epilepsy or mental illness, and brain trauma involving the brainstem.

Demographic and clinical data were collected from the medical records. The Glasgow Coma Scale (GCS) was assessed twice: on the first day of hospitalisation and on the day the patients were discharged. A laboratory workup was performed to identify predisposing factors that could affect the clinical manifestations, such as impairment of renal or hepatic function, electrolyte imbalance, blood gas analysis disorder, and/or glucose impairment. The EEG test with a duration of 30–60 min was performed using a 32 channel EasyIII-Cadwell® EEG machine. The scalp electrodes were placed using the 10–20 international system. During the test, the level of consciousness was assessed by asking patients to perform five tasks: (1) ‘Say your name’, (2) ‘Repeat 1,2,3’, (3) ‘Raise your arms’,

(4) ‘Open your eyes’, and (5) ‘Look at the examiner’. If no response was received from the patient, the procedure was repeated after providing strong tactile stimulation. Non-convulsive status epilepticus was determined by clinical manifestations and EEG recordings based on the mSCNC [6]. Since continuous EEG (cEEG) could not be performed in our hospital due to practical limitations, the EEG and clinical improvement following administration of anti-seizure medication (ASM) was determined by administering diazepam 0.2 mg/kg intravenously (IV) during the EEG test. Diazepam was given for patients whose EEG showed one of the following atypical features: (a) epileptic discharge (ED) <2.5 Hz, (b) rhythmic delta-theta activity with frequency >0.5 Hz with or without fluctuation. EEG and clinical improvement were observed for the subsequent 30 min. As patients were assessed for NCSE based on the EEG (NCSE group), they were routinely treated with maintenance ASM (i.e., phenytoin, valproic acid). The EEG test was performed every week in the NCSE group until the EEG showed improvement, or patients could have been discharged from the hospital.

EEG recording results were interpreted by EEG board-certified neurologists (FO, AB). Data were analysed using SPSS version 23.0. Statistical significance was determined at $p < 0.05$, with a 95% confidence interval (CI). Ethical approval was obtained from the Universitas Indonesia–Dr. Cipto Mangunkusumo General Hospital number 947/UN2.F1/ETIK/PPM.00.02/2019.

3. Results

A total of 39 participants met the inclusion criteria. The mean age was 40.8 ± 16.2 years, with a male to female sex ratio of 3:1. The EEG examination was performed on the median day 10 (range 2–46 days) after trauma onset, and the median length of stay was 19 days (range, 7–68

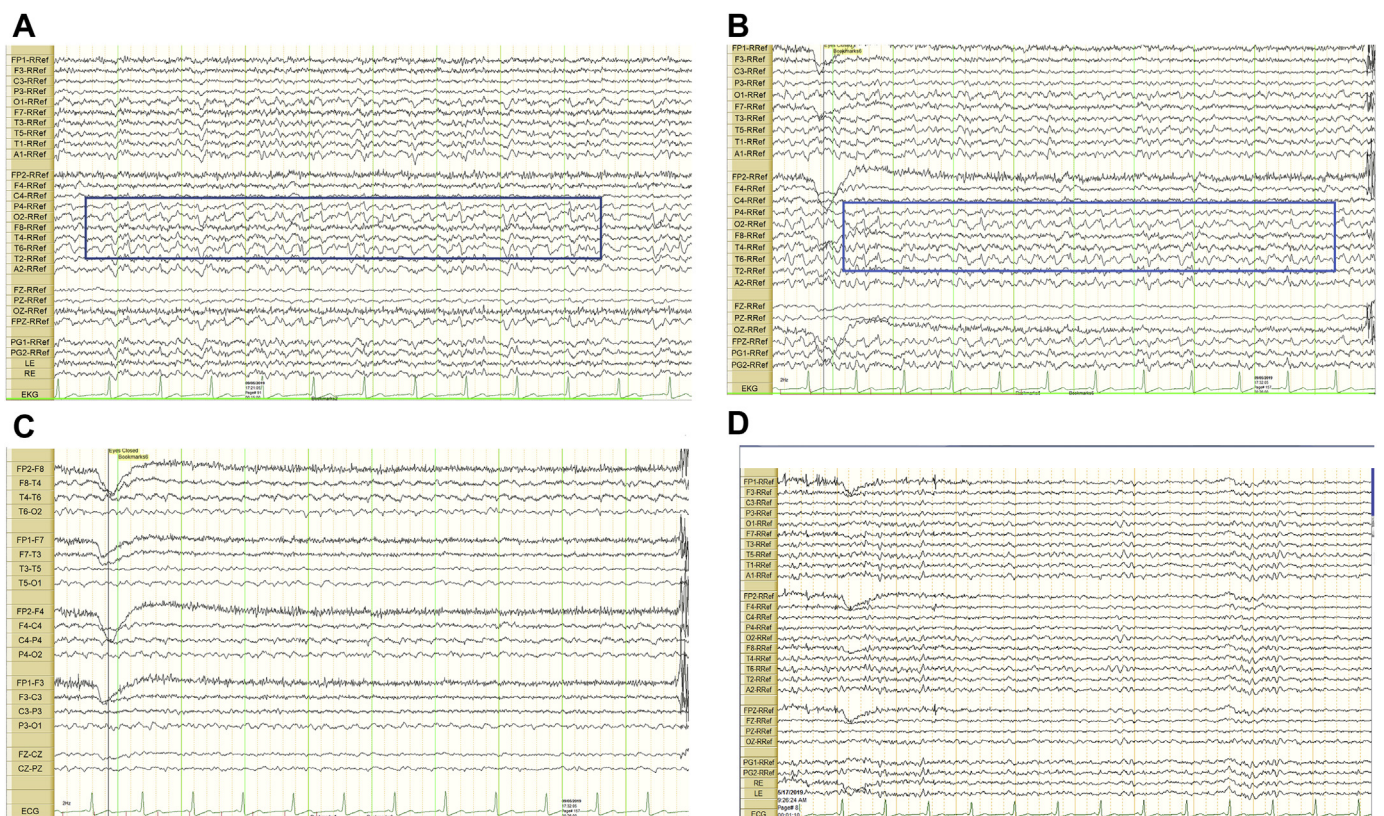


Figure 1. Epileptic discharge >2.5 Hz of more than 10 s. The EEG of a 46-year-old female patient who had visual hallucinations on the second day of hospitalisation due to a traffic accident. Rhythmic spike discharges were continuously evident for >10 s at right parieto-occipital lobe indicated by the blue box in the monopolar montage (A and B) and bipolar montage (C). On the third day, after treatment with phenytoin 300 mg/day, the hallucination was resolved and EEG 1 week later showed improvement (D). Phenytoin was continued for the next 1 month. EEG, electroencephalography.

Table 1. Demography, clinical, and radiological characteristics of the patients (n = 39).

Variables	n (%)
Age (year) [mean (SD)]	40.8 (16.2)
Sex	
Male	29 (74.4)
Female	10 (25.6)
Time interval between trauma onset and EEG test (days) [median (range)]	10 (2–46)
Duration of hospitalisation (days, [median (range)])	19 (7–68)
Trauma mechanism	
Traffic accident	32 (82.1)
Non-traffic accident	5 (12.8)
Unknown	2 (5.1)
GCS at onset (median (min-max))	12 (3–14)
Neurological deficit	
Altered consciousness	18 (46.2)
Delirium	2 (5.1)
Cognitive impairment	3 (7.7)
Psychomotor agitation	7 (17.9)
Perceptual impairment (illusion, hallucination)	2 (5.1)
Behavioural or thought content changes	1 (2.6)
Skull fracture	16 (41)
Convulsive seizure at onset	1 (2.6)
Anti-seizure medication (n = 19)	
Phenytoin	2 (10.5)
Valproic acid	16 (84.2)
Vaproic acid + Phenytoin	1 (5.2)
GCS at discharged (n = 38, median (min-max))	15 (11–15)
Died	1 (2.6)
Neurological deficit at discharged (n = 38)	
Not present	19 (50)
Present	19 (50)
Altered consciousness	1 (2.6)
Cognitive impairment	15 (39.5)
Behavioural or thought content changes	3 (7.9)

EEG = electroencephalography.

days). Traffic accidents were the most common causes (82.1%). The clinical manifestations varied greatly, with the most frequent being altered consciousness (46.2%), followed by psychomotor agitation, cognitive impairment, delirium, and perceptual impairment (illusion, hallucination). The common sites of skull fracture were the sphenoid wing and frontal region. The time interval between the onset of trauma and EEG tests varied widely (2–46 days). Patients with mTBI could be observed and were suspected of having NCSE symptoms if they already gained improvement of consciousness (e.g. spontaneous eye opening on GCS assessment), improvement in laboratory parameters (e.g. leukocytes, imaging on brain CT scan), and no other metabolic conditions could explain the fluctuating altered consciousness or agitation that they experienced. The only patient who underwent the EEG test on day-2 of onset presented with visual hallucinations (the EEG is shown in Figure 1). The demographic, clinical, and radiological characteristics of the study participants are presented in Table 1.

The common sites of TBI in this study were the frontal (79.5%), parietal (59%), and temporal (53.8%) lobes. Subarachnoid haemorrhage was the most common intracranial lesion (43.6%), followed by contusion (41%) and subdural haemorrhage (SDH) (35.9%) (Table 2).

In total, 19 of 39 (48.7%) patients were diagnosed with NCSE based on the mSCNC criteria. The EEG characteristics of the study subjects are presented in Table 3.

Non-convulsive seizures were determined based on EEG in 19 of the 39 recruited participants, as described in the mSCNC criteria (Table 3).

Table 2. Pathological characteristics based on head CT scans (n = 39).

Variables	n (%)
Number of lesions	
Not present	3 (7.7)
1 lesion	3 (7.7)
>1 lesion	33 (84.6)
Location of lesion	
Without lesion	3 (7.7)
Frontal	31 (79.5)
Temporal	21 (53.8)
Parietal	23 (59)
Number of lesion pathology	
1 pathology	15 (38.5)
>1 pathology	24 (61.5)
Type of lesion pathology	
None	3 (7.7)
ICH	11 (28.2)
Contusion	16 (41)
EDH	8 (20.5)
SDH	14 (35.9)
SAH	17 (43.6)

ICH = intracranial haemorrhage; EDH = epidural haemorrhage; SDH = subdural haemorrhage; SAH = subarachnoid haemorrhage; CT, computed tomography.

NCSE based on mSCNCs was classified as definite or possible NCSE. Definite NCSE was determined if the EEG showed ED (>2.5 Hz) for more than a 10 s epoch, rhythmic activity (>0.5 Hz) with spatiotemporal evolution, or subtle ictal clinical phenomena. ED > 2.5 Hz was observed in one patient who had a visual hallucination on the second day of hospitalisation (Figure 1). She was treated with an antipsychotic drug before EEG was performed. As NCSE was diagnosed based on EEG, she was treated with phenytoin 300mg/day, and the hallucinations disappeared. Rhythmic delta activities (>0.5 Hz) with spatiotemporal evolution in the left temporal lobe were observed in one patient with disorientation and agitation (Figure 2). Rhythmic activity (>0.5 Hz) with fluctuations that disappeared after administration of an AED was observed in two patients (Figure 3). Most participants showed rhythmic activity (>0.5 Hz) without fluctuation, which improved with ASM administration (Figure 4). The details of the EEG features of NCSE are described in Table 3.

Table 3. EEG Characteristics of subjects with NCSE according to the mSCNC (n = 19).

EEG Features	n (%)
A. Definite NCSE	
A.1. Epileptic discharges >2.5 Hz for more than 10-s epoch	1 (2.6)
A.2. Typical ictal spatiotemporal evolution of	
(2a) Epileptic discharge	
(2b) Rhythmic activity (>0.5 Hz)	1 (2.6)
A.3. Subtle ictal clinical phenomena with:	
(3a) Epileptic discharge	0
(3b) Rhythmic activity (>0.5 Hz)	1 (2.6)
B. Possible NCSE	
B.1. Responsive with ASM	
(1a) Epileptic discharge ≤2.5 Hz with fluctuation	0
(1b) Rhythmic activity (>0.5 Hz) with fluctuation	2 (7.4)
(1c) Rhythmic activity (>0.5 Hz) without fluctuation	15 (55.6)
C. More than 1 criterion	1 (2.6)

mSCNC = modified Salzburg criterion for non-convulsive status epilepticus; EEG = electroencephalography; ASM = anti-seizure medication; NCSE = Non-convulsive status epilepticus.

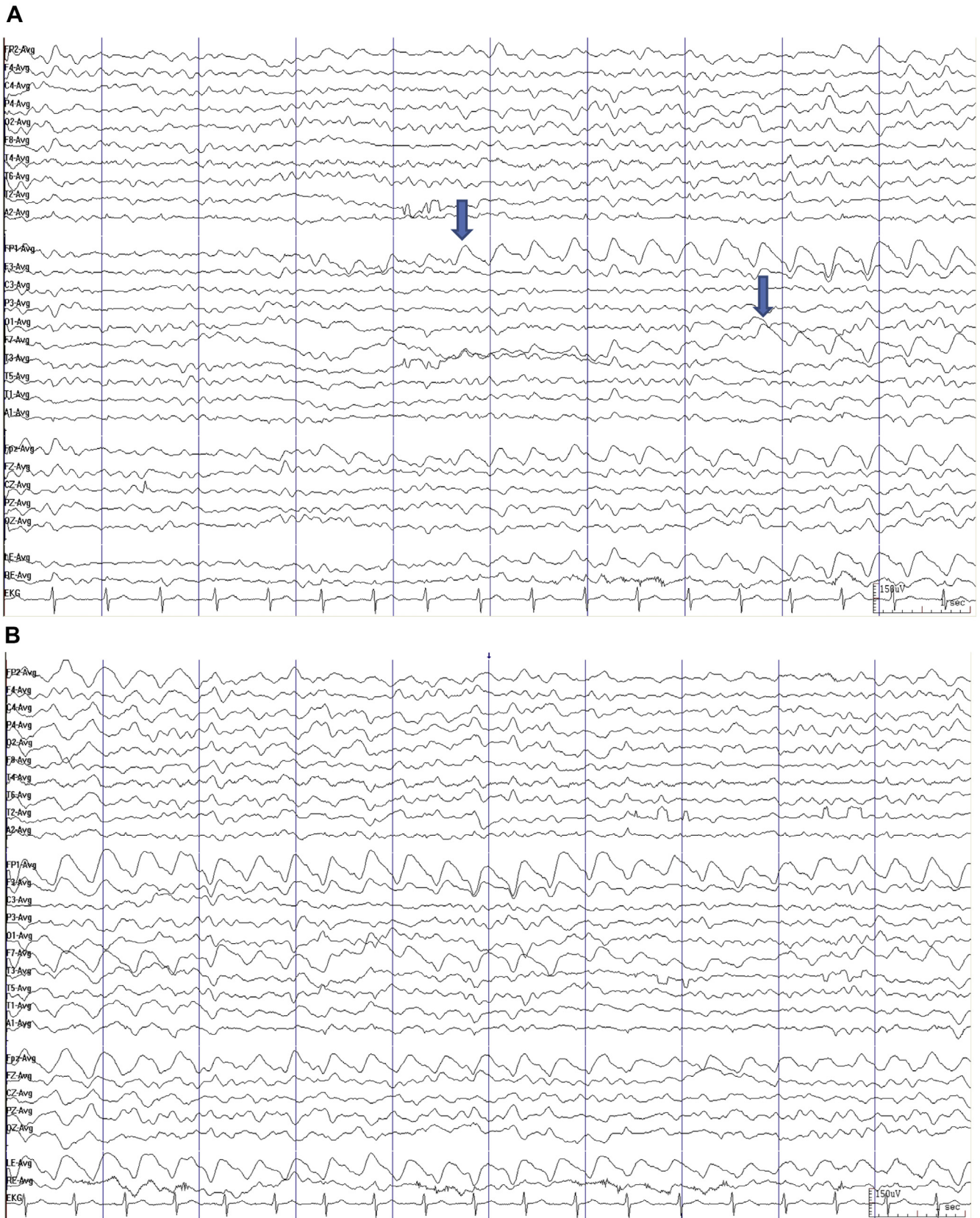


Figure 2. Typical Ictal Rhythmic Spatiotemporal Evolution. EEG of a 22-year-old male patient with disorientation and motoric agitation during hospitalisation due to a traffic accident. Delta rhythmic waves >0.5 Hz were evident on the left fronto-polar (FP1), which evolved in frequency and morphology, with propagation to adjacent areas (F7) indicated by blue arrows. EEG, electroencephalography (A continued to B).

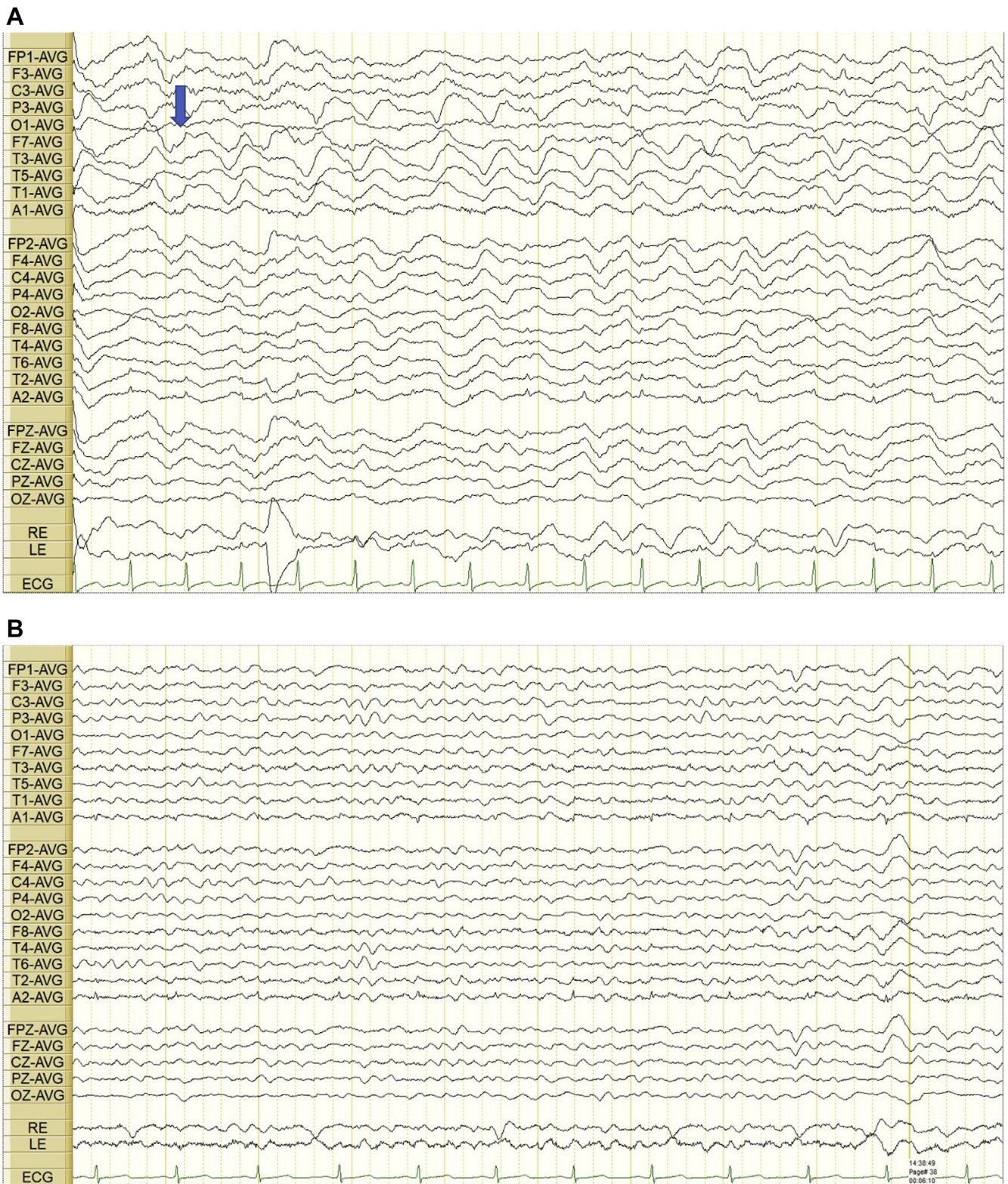


Figure 3. The fluctuating Rhythmic Activity (>0.5 Hz). EEG of a 62-year-old male patient with psychomotor agitation due to traffic accident. Rhythmic delta waves >0.5 Hz were seen in the left temporal lobe (blue arrows) with fluctuating changes in frequency and amplitude (A) that disappeared within 20 min as the anti-seizure medication, Diazepam 10 mg intravenously, was administered intravenously (B). EEG, electroencephalography.

In total, 19 participants had NCSE, and 20 did not. A comparison of the demographic and clinical characteristics is presented in Table 4. The GCS score was significantly lower in the NCSE group than in the non-NCSE group at onset and at hospital discharge. Delirium and hallucinations were only seen in the NCSE group, and

these improved during hospitalisation following ASM administration. Cognitive impairment was seen in nine subjects in the NCSE group and in six in the non-NCSE group when assessed at hospital discharge. The temporal lobe was more affected in the NCSE group (30.8%).

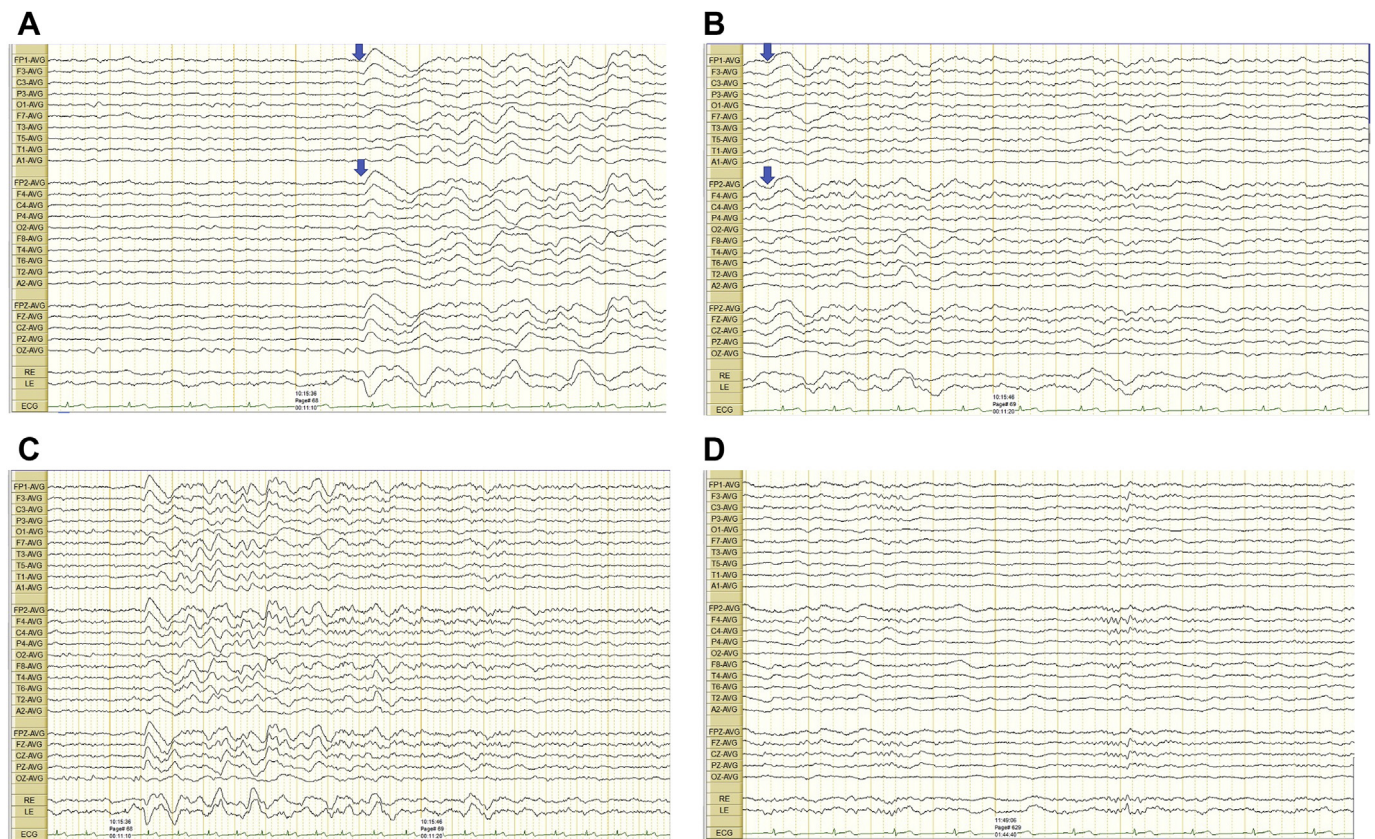


Figure 4. Rhythmic Activity without Fluctuation (>0.5 Hz). EEG of a 32-year-old male patient with decreased consciousness due to a traffic accident. Rhythmic delta activity >0.5 Hz (blue arrows) was seen in the bilateral fronto-temporal region without fluctuation (A continued to B [paper speed 30 mm/s]; same epoch in C [paper speed 15 mm/s]), which disappeared as anti-seizure medication (Diazepam 10 mg; intravenously) was administered (D). EEG, electroencephalography.

Variables with $p < 0.25$, in bivariate analysis, were included in the multivariate analysis using logistic regression. Table 5 shows that a lesion in the temporal lobe was an independently significantly associated factor for NCSE in msTBI ($p = 0.036$, odds ratio 11.45 [95% CI 1.17–111.6]).

4. Discussion

All participants were of reproductive age, with males more commonly affected than females. The trend towards a higher proportion of males was consistent with previous studies.² Based on the mechanism of trauma, motorcycle and pedestrian accidents were the most frequent causes (53.8% and 17.9%, respectively), in agreement with a study by Faried et al., which reported frequencies of 65.4% and 16.3% [7].

Based on the location of the injury, the frontal lobe was the most commonly affected (79.5%), followed by the parietal lobe (59%) and the temporal lobe (53.8%). This result differs from a study by Vespa et al., which found that the temporal and frontal lobes were the most involved locations in msTBI.⁵ Similarly, Gupta et al. examined 123 post-traumatic epilepsy patients and found that the temporal lobe was the most common site of lesion (57%), followed by the frontal lobe (25%), parietal lobe (3%), and occipital lobe (3%) [8, 10].

The GCS scores of the NCSE patients were significantly lower in our study than in previously published studies. Overall, four patients with GCS scores of 3–8 had NCSE, whereas no one in the non-NCSE group had GCS ≤ 8 . The association between the occurrence of NCSE and decreased consciousness provided an overview of the higher degree of neuronal injury in the NCSE group which leads to worse EEG. Claasen et al. reported that altered mental status, particularly coma, was an independent risk factor for NCSE [9].

The clinical manifestations of NCSE in the present study varied considerably. Decreased consciousness was the most common symptom

(47.4%), followed by psychomotor agitation (26.3%), perceptual impairment (hallucination, illusion; 10.5%), and delirium (10.5%). Sutter et al. reported that approximately 43% of NCSE patients presented with altered and decreased consciousness [10]. The clinical manifestations found in the present study were closely associated with temporal lobe involvement.

This study showed that temporal lobe involvement was independently associated with NCSE. Lesions originating from the temporal lobe are associated with many neuropsychiatric signs and symptoms, such as agitation, delirium, and hallucinations. The temporal lobe plays an important role in awakening and consciousness [11]. As regions of the temporal lobe, the hippocampus and dentate gyrus are the most susceptible to injury, either due to direct mechanical injury or post-traumatic seizures [12].

The increase in the median GCS score at hospital discharge was higher in the NCSE group than in the non-NCSE group. Symptoms such as decreased consciousness, delirium, agitation, and hallucinations improved in the NCSE group after administration of ASM. Responsiveness to ASM is one sign that can confirm the diagnosis of NCSE. Despite the more improvement of GCS score in NCSE group, it was still significantly lower than in the non-NCSE group when patients were discharged, and the motor and verbal components of the GCS were the most affected. This finding is thought to reflect the prolonged electrographic seizures that occurred in the NCSE group. The EEG after traumatic brain injury (TBI) will improve over time along with improvement of clinical symptom. The diffuse slowing of the EEG in the acute phase of TBI will resolve by 3 months up to 1 year of the head trauma [13]. In this study, the clinical improvement could be due to resolving brain injury over time and resolving NCSE by ASM as well. As diagnosis of NCSE has been made, treating the non-convulsive seizure helps improvement of brain injury. Further prospective studies are needed to determine the other outcome

Table 4. Comparison of demographic, clinical, and EEG characteristics between the NCSE and Non-NCSE groups (n = 39).

Variables	n (%)		p value	95% CI OR (Min-Max)
	NCSE (n = 19)	Non-NCSE (n = 20)		
Age (years) (mean (SD))	42.1 (14.6)	39.6 (18.1)	0.628 ^a	
Male sex	15 (78.9)	14 (70)	0.394 ^b	
Female sex	4 (10.3)	6 (15.4)		
Length of stay (days) (median (range))	24 (13–68)	14 (7–47)		
Mechanism of trauma				
Traffic accident	17 (89.5)	15 (75)		
GCS at onset (median (min-max))	10 (3–14)	13.5 (9–14)	0.004 ^d	
Neurological deficit at onset	18 (94.7)	15 (75)	0.151 ^c	2.59 (0.7–9.64)
Altered consciousness	9/18	9/15	0.513 ^b	
Delirium	2/18	0		
Cognitive impairment	0	3/15		
Psychomotor agitation	5/18	2/15		
Hallucination	2/18	0		
Behavioural changes or thought content changes	0	1/15		
Skull fracture	10 (52.6)	6 (30)		
Immediate seizure	0	1 (5)		
Outcome				
- GCS at discharged (n = 38), (median (range))	14 (11–15)	15 (13–15)	0.03 ^d	
- Died	1	0		
Neurological deficit at discharge	11 (57.9)	8 (40)		
Altered consciousness	0/11	1/8		
Cognitive impairment	9/11	6/8		
Behavioural or thought content changes	2/11	1/8		
Location of pathology (n = 36)				
Temporal	12 (63.2)	9 (45)	0.158 ^c	2.67 (0.67–10.6)
Extratemporal	5 (26.3)	10 (50)		

EEG = electroencephalography; GCS = Glasgow Coma Scale; NCSE = Non-convulsive status epilepticus.

^a Unpaired t-test.

^b Fisher's exact test.

^c Chi-square test.

^d Mann-Whitney test, p 2-tailed.

Table 5. Logistic regression multivariate analysis of the site of the lesion variable.

Variable	Coefficient	p value	95% CI OR (Min-Max)
Temporal site	2.438	0.036	11.45 (1.17–111.6) R ² = 0.485

OR = Odds Ratio; 95% CI = confidence interval.

variables in the long-term evaluation (e.g., cognitive function, neuropsychiatry assessment).

The number of NCSE cases in msTBI was estimated to exceed that obtained in the present study. Many msTBI cases with suspected NCSE were treated in intensive care units that were not covered by EEG facilities in our hospital. Our findings should increase awareness that NCSE must be highly suspected in msTBI patients with altered mental status accompanied by temporal lobe lesions.

The limitation of our study was that the onset of NCSE could not be determined since cEEG could not be performed. The NCSE symptoms could only be observed in msTBI patients who had already gained improvement in consciousness, but there were no other conditions that could explain the fluctuating altered consciousness and agitation. Therefore, the interval between onset and the EEG test varied widely.

5. Conclusion

The most commonly observed clinical manifestations in patients with msTBI in terms of NCSE were altered mental status, including psychomotor agitation, hallucination, and delirium. Temporal lobe involvement was a substantial factor associated with the occurrence of NCSE in msTBI.

Declarations

Author contribution statement

Fitri Octaviana: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Jeffri Harisman: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data.

Winnugroho Wiratman: Conceived and designed the experiments; Wrote the paper.

Astri Budikayanti: Conceived and designed the experiments; Performed the experiments; Wrote the paper.

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Data availability statement

Data included in article/supplementary material/referenced in article.

Declaration of interests statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

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