Original article

Crit Care Shock (2025) 28:41-52

Impact of nonconvulsive seizures after traumatic brain injury assessed by continuous electroencephalography monitoring in intensive care unit

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Abstract

***Objective*: To evaluate the impact of nonconvulsive seizures (NCS) and nonconvulsive status epilepticus (NCSE) diagnosed via continuous electroencephalogram (cEEG) monitoring on short-term neurological outcomes and disability in patients with moderate to severe traumatic brain injury (TBI).**

***Design*: A prospective observational study.**

***Setting*: A tertiary care intensive care unit (ICU) specializing in neurological and trauma care.**

***Patients and participants*: A total of 44 patients aged >18 years with moderate to severe TBI, classified using the Glasgow Coma Scale (GCS), were included. Patients with a history of seizures, anoxic brain injury, or brainstem death were excluded.**

***Interventions*: Continuous EEG monitoring was performed using the Salzburg Consensus Criteria to detect and classify NCSE and NCS. Patients were categorized into NCSE, possible NCSE, and no NCSE groups based on cEEG findings.**

***Measurements and results*: Outcome measures included neurological deterioration on day 7, Glasgow Outcome Score Extended (GOS-E) at 28 days, and duration of hospital stay. Patients with NCSE exhibited significantly worse outcomes, including lower GCS scores ($p<0.05$), higher Acute Physiology and Chronic Health Evaluation II (APACHE II) scores ($p<0.001$), and prolonged hospital stays ($p<0.001$). Neurological deterioration was observed in 100% of NCSE patients compared to 20% in possible NCSE and 0% in no NCSE groups ($p<0.001$). Intracranial hemorrhage on follow-up imaging was more frequent in the NCSE group (47.1%, $p=0.027$).**

***Conclusions*: NCSE diagnosed by cEEG monitoring in moderate to severe TBI is associated with the poorest short-term outcomes, including higher rates of disability, prolonged hospitalization, and increased neurological deterioration. Early implementation of cEEG monitoring is critical for timely diagnosis and management in this high-risk population.**

Key words: Nonconvulsive seizures, nonconvulsive status epilepticus, traumatic brain injury, continuous electroencephalography, Glasgow Outcome Score Extended.

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Introduction

Electroencephalography (EEG) monitoring is recommended for patients admitted to the intensive care unit (ICU) with an unclear cause of reduced consciousness. This is particularly critical for individuals without a history of seizures or those who have stopped convulsing. EEG monitoring is also essential for detecting nonconvulsive status epilepticus (NCSE) or nonconvulsive seizures (NCS). (1) EEG confirmation is required to diagnose NCSE and NCS in the appropriate clinical context. The prevalence of NCSE in comatose patients ranges from 8% to 19%. (2)

Patients diagnosed with NCSE are at high risk of poor outcomes, with mortality rates between 20% and 30%. However, the underlying cause significantly influences the prognosis. For instance, patients with NCSE and acute medical conditions have a markedly higher mortality rate (27%) compared to those with epilepsy, who exhibit a much lower mortality rate (3%). (3)

Continuous electroencephalogram (cEEG) monitoring is a widely used, noninvasive tool that plays a critical role in diagnosing NCS and NCSE in critically ill patients. However, given the substantial resource demands, including skilled personnel and specialized equipment, it is essential to evaluate the effectiveness of cEEG, optimize its use, and assess its impact on healthcare systems and patient outcomes. (4)

cEEG monitoring is increasingly utilized in neurologically critical care settings due to its proven efficacy in diagnosing NCSE, guiding treatment decisions, and assessing treatment outcomes. (5)

The objective of this study was to evaluate the prognostic impact of cEEG on short-term outcomes in patients with acute traumatic brain injury.

Patients and methods

This prospective observational study was conducted on 44 individuals aged >18, of both genders, with moderate to severe TBI, classified according to the Glasgow Coma Scale (GCS). The GCS categorizes TBI into three levels: mild (14-15), moderate (9-13), or severe (3-8). It comprises three components: verbal performance, motor responsiveness, and eye-opening. (6) The study received approval from the Faculty of Medicine Research Ethics Committee (REC), Cairo University, Egypt (approval code: md-134-2023). Informed written consent was obtained from all patients or their guardians.

Patients with a history of seizures, anoxic brain injury due to cardiorespiratory arrest, or brainstem death were excluded from the study.

Each participant underwent a comprehensive assessment, including medical history, physical examination, laboratory tests (complete blood count [CBC], blood glucose level, serum sodium concentration, arterial blood gas [ABG] analysis, and additional investigations as clinically indicated), and radiological evaluations (plain X-rays [spinal, chest, pelvis, skeletal survey], computerized tomography [CT] scans of the head on admission and follow-up at 48 hours unless clinically warranted, and other imaging studies based on patient condition and injuries).

The Acute Physiology and Chronic Health Evaluation II (APACHE II) score was calculated for all patients on admission, using starting values of 12 routine physiological parameters, age, and prior health condition. This score serves as a comprehensive indicator of illness severity. (7)

cEEG monitoring was performed using the Nihon Kohden EEG machine (H15041004, Japan) in accordance with the American Clinical Neurophysiology Society International 10-20 system guidelines. (8) Monitoring began after patient stabilization. The cEEG tracings were evaluated to identify the background pattern and detect any abnormal discharges. Patients were observed for at least 12 hours unless their clinical status necessitated a shorter duration (e.g., hospital discharge or death) or longer monitoring (e.g., ongoing seizures). (9) All EEGs were analyzed by a certified neurophysiologist with expertise in intensive care EEG.

Patients were categorized into three groups based on the presence of NCSE after cEEG evaluation: NCSE group, possible NCSE group, and no NCSE group, as per the Salzburg Consensus Criteria. (9)

A diagnosis of NCSE was directly established if epileptiform discharges (EDs) occurred at a frequency greater than 2.5 Hz and persisted for at least 10 seconds. If EDs lasted for a maximum of 10 seconds at a frequency of 2.5 Hz or less, or if EDs were undetectable but continuous (semi-) rhythmic delta-theta activity (RDTA) between 0.5 and 4.0 Hz was present, additional criteria must be met. These included subtle clinical phenomena, typical spatiotemporal evolution, or a response to intravenous (IV) antiepileptic drugs (AEDs) in both clinical and EEG features. If only random variations in the frequency, structure, or locations of EDs or RDTA were observed without a discernible pattern over time, or if EEG changes slightly improved without corresponding clinical improvement after IV AEDs, the diagnosis of "possible NCSE" might be considered. In all other cases, NCSE was ruled out if RDTA patterns were present at a frequency of less than 0.5 Hz or if the patterns showed no evolution or fluctuation (**Figure 1**). (10)

Outcome measures

Secondary neurological deterioration on day 7 post-trauma was assessed through clinical observation. Neurological deterioration was defined by one of two objective criteria: 1) a reduction in the GCS score by more than 2 points from baseline, excluding the effects of sedative medications, or 2) a decline in a neurological condition requiring intervention, such as mechanical ventilation, seizures, increased brain edema necessitating repeated doses of osmotherapy, or neurosurgical procedures. (11) The Glasgow Outcome Score Extended (GOS-E) was

evaluated 28 days post-trauma. The GOS-E categorizes outcomes into the following levels: (8) Excellent recovery; (7) Good recovery with slight mental or physical impairments; (6) Moderate disability, allowing a return to previous employment with minor adjustments; (5) Moderate disability, permitting work at reduced performance levels; (4) Severe disability, where the individual is partially dependent on others for certain activities; (3) Severe disability, indicating full dependence on others for daily living activities; (2) Vegetative state; and (1) Death. (11)

Statistical analysis

Statistical analysis was performed using SPSS version 27 (IBM©, Chicago, IL, USA). The normality of data distribution was assessed using the Shapiro-Wilks test and visual inspection of histograms. Quantitative parametric variables were expressed as mean±standard deviation (SD) and analyzed using analysis of variance (ANOVA) with Tukey's post hoc test. Quantitative non-parametric variables were presented as the median and interquartile range (IQR) and compared using the Kruskal-Wallis test, followed by the Mann-Whitney U test for pairwise group comparisons. Qualitative variables were reported as frequencies and percentages (%) and analyzed using the chi-square test. A two-tailed p-value <0.05 was considered statistically significant.

Results

The studied groups were comparable in age, sex, comorbidities, type of trauma, time elapsed between injury and hospital admission, and vital signs upon admission (**Table 1**).

On admission, no significant differences were observed among the groups concerning laboratory data, oxygen saturation on room air, or the presence of shock or seizures. However, patients who subsequently developed NCSE had a significantly higher incidence of severe TBI (p=0.004), lower GCS scores, elevated APACHE II scores, and higher APACHE II score mortality percentages (p<0.05) (**Tables 2** and **3**).

No significant differences were found among the groups regarding the timing of cEEG initiation, duration of monitoring, other forms of management, or findings from CT scans performed on admission and 48 hours later, except for the presence of intracranial hemorrhage (ICH). Notably, the duration of mechanical ventilation significantly differed among the groups (p=0.001) (**Table 4**).

Patients with NCSE exhibited the highest incidence of EDs >2.5 Hz lasting at least 10 seconds, along with the lowest incidence of continuous (semi-) RDTA between 0.5 and 4.0 Hz. In contrast, patients in the possible NCSE group had the highest incidence of ED frequency ≤2.5 Hz, fluctuations without definitive evolution, and the most favorable EEG responses following intravenous AEDs (**Table 5**).

Patients with NCSE demonstrated a significantly higher frequency of GCS deterioration, increased brain edema, a greater need for repeated doses of osmotherapy, and a higher incidence of neurological deterioration (**Table 6**).

Discussion

Post-traumatic seizures (**PTS)** result from a series of primary or secondary injury mechanisms. Seizures occur in approximately 22% of individuals with moderate-to-severe TBI, and 52% of these patients experience NCS. Among all TBI cases, NCS was observed in 23.5%, and 5.9% progressed to NCSE. (12)

No significant differences in laboratory findings were observed among the groups in this study. However, Claassen et al. (13) reported that patients with aneurysmal subarachnoid hemorrhage (SAH) who experienced in-hospital NCS exhibited a stronger systemic inflammatory response syndrome (SIRS). Their findings indicated that inflammatory surges were more likely to occur immediately before the onset of NCS. Additionally, they found that the negative impact of SIRS on functional outcomes after three months was partially mediated by the occurrence of in-hospital NCS. Similarly, Vespa et al. (14) suggested that PTS might represent a therapeutic target in patients with TBI.

The presence of clinical seizures or a history of epilepsy has been identified as a risk factor for NCSE and NCS in studies by Singh et al. (15) and Laccheo et al.. (16) However, in the current study, no significant correlation was found between NCSE and seizures on admission.

Additionally, no significant association was observed between NCSE and the presence of shock on arrival. Gutierrez et al., supporting this finding, (17) reported that established risk factors, including cardiac arrest and sepsis, did not significantly correlate with a higher incidence of NCS. In contrast, Gilmore et al. (18) reported that the severity of cardiovascular shock was associated with an increased risk of periodic discharges or NCS.

Patients with NCSE had significantly lower GCS scores upon admission. This finding aligns with Singh et al. (15), who identified a strong association between a comatose state and NCS. Additionally, Octaviana et al. (19) reported that GCS scores at admission and hospital discharge were significantly

reduced in patients with NCSE compared to those without.

Patients with NCSE had significantly higher APACHE II scores upon admission. This finding was supported by Vespa et al. (14), who concluded that PTS led to both intermittent and prolonged elevations in intracranial pressure.

Patients with NCSE also had significantly higher APACHE II score mortality percentages. In support of this finding, Gilmore et al. (18) reported that the non-neuro APACHE II score was correlated with a lower risk of periodic discharges or NCS.

Additionally, patients with NCSE had significantly lower GOS-E than those without NCSE. This observation aligns with the findings of Singh et al. (15), who demonstrated that NCS was significantly associated with more significant disability upon discharge.

Individuals with NCSE also had significantly longer hospital stays than patients without NCSE. Singh et al. (15) reported that NCS was significantly associated with prolonged in-hospital stays.

CT scans performed 48 hours after admission revealed that patients with NCSE had the highest prevalence of ICH. Similarly, a study by Matsubara et al. (20) on 228 cases of acute spontaneous ICH suggested that lobar involvement, particularly of the insular lobe, was associated with NCSE, independent of the extent of ICH. Furthermore, Claassen et al. (21) observed that an increase in ICH volume of 30% or more between admission and the 24-hour follow-up CT was linked to the occurrence of electrographic seizures.

The study by Anadure et al. (22) reported that EEG recordings of NCSE patients exhibited variability, with changing rhythms and seizure-related EEG patterns linked to spatiotemporal alterations. These EEG changes were reversed with the administration of AEDs. In 41.7% of NCSE cases, a temporary increase in GCS score (>2 points) was observed after AED administration, resulting in favorable clinical outcomes (GOS of 5). Similarly, Octaviana et al. (19) found that the most common EEG feature in NCSE cases was rhythmic activity (>0.5 Hz) without fluctuation, which improved with AED use.

Patients diagnosed with NCSE exhibited a significantly higher occurrence of GCS decline. The correlation between NCSE prevalence and reduced awareness underscored the greater extent of neuronal damage in the NCSE group, reflected in more severe EEG abnormalities. Conversely, Octaviana et al. (19) observed that the median GCS score upon hospital discharge was higher in the NCSE group than those without NCSE. The administration of AEDs in NCSE patients improved symptoms, including reductions in delirium, impaired consciousness, agitation, and hallucinations.

Patients with NCSE also experienced a significantly higher incidence of neurological deterioration. Similarly, Singh et al. (15) reported that NCS was significantly associated with a higher incidence of disability upon discharge.

This study observed no significant difference in in-hospital mortality rates between patients with NCSE and those without. This finding aligned with Singh et al., (15) who also found no significant correlation between NCS and in-hospital mortality. However, Gutierrez et al. (17) reported a lower mortality rate among individuals with NCS compared to those without seizures. Additionally, Jayalakshmi et al. (23) highlighted the importance of underlying etiology in predicting NCSE outcomes, noting increased mortality rates associated with hypoxic-ischemic encephalopathy, a massive stroke, sepsis, and severe brain trauma.

The study's limitations include a relatively small sample size, short-term follow-up, and technical challenges. Critical time constraints and the need for a one-to-one nursing ratio, particularly in agitated TBI patients requiring sedation, added to the ICU workload.

Conclusions

NCSE diagnosed through cEEG monitoring in individuals with moderate to severe TBI is associated with the poorest short-term outcomes and a significantly higher incidence of disability at discharge, irrespective of the type of trauma.

Acknowledgment

The authors would like to thank the patients who participated in this study, as well as the colleagues and medical staff who provided patient care.

Funding

The authors have no relevant financial or non-financial interests to disclose.

Conflict of interest

The author(s) declare no potential conflicts of interest concerning the research, authorship, and/or publication of this article.

Availability of data and materials

The data is available upon reasonable request from the authors.

Table 1. Comparison between the groups under the study regarding demographic data, comorbidities, type of trauma and time laps between injury and admission, and vital data on admission

		NCSE (n=17)	Possible NCSE (n=15)	No NCSE (n=12)	p
Age (years)		43.0±12.58	40.47±11.12	40.42±8.67	0.761
Sex	Female	5 (29.4%)	3 (20.0%)	5 (41.7%)	0.500
	Male	12 (70.6%)	12 (80.0%)	7 (58.3%)	
Comorbidities	DM	7 (41.2%)	2 (13.3%)	3 (25.0%)	0.246
	HTN	4 (23.5%)	4 (26.7%)	2 (16.7%)	0.904
	Hepatic	1 (5.9%)	2 (13.3%)	1 (8.3%)	0.820
	Renal	2 (11.8%	2 (13.3%)	1 (8.3%)	1
	Cardiac	2 (11.8%	1 (6.7%)	1 (8.3%)	1
	Others	2 (11.8%	2 (13.3%)	4 (33.3%)	0.333
Road traffic accident		8 (47.1%	9 (60.0%)	9 (75.0%)	0.323
Fall from height		5 (29.4%	4 (26.7%)	0 (0.0%)	0.112
Falling on the ground		1 (5.9%	0 (0.0%)	1 (8.3%)	0.730
Assault		2 (11.8%)	1 (6.7%)	0 (0.0%)	0.769
Fall of heavy object		1 (5.9%)	1 (6.7%)	1 (8.3%)	1
Time laps between injury and admission (hours)		9.47±3.08	11.53±4.78	11.33±3.50	0.262
Vital data on admission	Pulse (beats/min)	107.35±16.50	98.67±21.42	96.25±19.90	0.258
	MAP (mmHg)	72.65±16.69	81.00 ± 21.97	86.25±24.78	0.221
	Temperature (°C)	37.70 ± 0.63	37.67 ± 0.60	37.64 ± 0.69	0.971

Legend: NCSE=nonconvulsive status epilepticus; DM=diabetes mellitus; HTN=hypertension; MAP=mean arterial pressure.
Data are presented as mean±SD or frequency (%).

Table 2. Comparison between studied groups as regards clinical assessment and laboratory data on admission

	NCSE (n=17)	Possible NCSE (n=15)	No NCSE (n=12)	p
Clinical assessment on admission				
- Severity of TBI				
* Moderate	6 (35.3%)	13 (86.7%)	10 (83.3%)	0.004*
* Severe	11 (64.7%)	2 (13.3%)	2 (16.7%)	
- GCS score on admission	8.35±2.37	10.60±2.29	10.58±2.19	0.012*
- APACHE II score on admission	23.88±6.00	11.80±5.33	13.58±4.23	<0.001*
- APACHE II score mortality (%)	55.00 (4.00-75.00)	8.00 (4.00-25.00)	16.50 (4.0-25.0)	<0.001*
- Oxygen saturation on room air on admission (%)	91.71±3.35	94.13±3.07	92.17±2.86	0.086
- Shock on arrival	3 (17.6%)	4 (26.7%)	3 (25.0%)	0.818
- Seizures on admission	1 (5.9%)	0 (0.0%)	2 (16.7%)	0.262
Laboratory data				
- Blood picture				
* Hb	10.54±2.49	11.03±2.02	11.81±2.63	0.374
* WBCs	14.29±5.39	14.29±4.61	12.77±4.40	0.658
* PLT	233.0 (92.00-377.00)	271.0 (51.00-677.00)	206.0 (129.00-454.00)	0.819
- Blood gases				
* pH	7.37±0.09	7.33±0.13	7.27±0.11	0.056
* PaO2	120.0 (23.0-381.0)	104.0 (22.0-423.0)	67.50 (25.0-158.0)	0.155
* PaCO2	39.0±10.75	35.47±15.64	38.17±9.36	0.709
* HCO3	22.0±6.69	18.03±4.95	17.51±6.74	0.099
* SaO2	81.94±20.11	91.73±12.52	81.67±12.50	0.160
- Coagulation				
* PT	13.88±1.77	14.07±1.62	13.23±0.98	0.353
* PTT	33.23±1.97	32.85±1.96	32.78±1.69	0.782
* INR	1.37±0.21	1.38±0.19	1.28±0.17	0.344
* D dimer	1420.0 (550.0-4000.0)	1600.0 (600.0-3600.0)	1980.0 (600.0-3000.0)	0.937

Legend: NCSE=nonconvulsive status epilepticus; TBI=traumatic brain injury; GCS=Glasgow Coma Scale; APACHE II=Acute Physiology and Chronic Health Evaluation II; Hb=hemoglobin; WBCs=white blood cells count; PLT=platelet count; PaO2=arterial partial pressure of oxygen; PaCO2=arterial partial pressure of carbon dioxide; HCO3=bicarbonate; SaO2=arterial oxygen saturation; PT=prothrombin time; PTT=partial thromboplastin time; INR=international normalized ratio.
Data are presented as mean±SD or median (IQR).

Table 3. Post hoc pairwise comparison between studied groups regarding GCS score on admission, APACHE II score on admission, APACHE II score mortality %, duration of mechanical ventilation, Glasgow Outcome Score Extended, and duration of hospital stay

	NCSE vs possible NCSE	NCSE vs no NCSE	Possible NCSE vs no NCSE
GCS score on admission	8.35±2.37 vs 10.60±2.29 p=0.026*	8.35±2.37 vs 10.58±2.19 p=0.041*	1.000
APACHE II score on admission	23.88±6.00 vs 11.80±5.33 p<0.001*	23.88±6.00 vs 13.58±4.23 p<0.001*	1.000
APACHE II score mortality (%)	49.35±17.38 vs 15.33±9.32 p<0.001*	49.35±17.38 vs 16.17±9.29 p<0.001*	0.801
Duration of mechanical ventilation (days)	11.53±4.29 vs 4.07±5.31 p=0.002*	11.53±4.29 vs 4.92±6.35 p=0.022*	1.000
Glasgow Outcome Score Extended	0.500	2.18±1.13 vs 4.58±2.39 p=0.011*	0.351
Hospital stays duration (days)	0.160	27.67±4.27 vs 22.83±5.87 p<0.001*	0.054

Legend: GCS=Glasgow Coma Scale; APACHE II=Acute Physiology and Chronic Health Evaluation II; NCSE=nonconvulsive status epilepticus.
Data are presented as mean±SD. *significant p<0.05.

Table 4. Comparison between the groups under the study as regards the time of starting cEEG, duration of monitoring, management on admission, and findings of CT on admission and at 48 hours after admission

	NCSE (n=17)	Possible NCSE (n=15)	No NCSE (n=12)	p
Time of starting cEEG (days)	6.53±0.87	6.73±1.39	6.67±1.23	0.881
Time of monitoring (hours)	12.00±0.00	12.00±0.00	12.00±0.00	1
Management on admission				
- Mechanical ventilation	11 (64.7%)	5 (33.3%)	5 (41.7%)	0.227
- Osmotherapy	5 (29.4%)	3 (20.0%)	5 (41.7%)	0.500
- Mannitol	2 (11.8%)	1 (6.7%)	4 (33.3%)	0.193
- Hypertonic saline	2 (11.8%)	2 (13.3%)	1 (8.3%)	1
- Neurosurgical intervention	6 (35.3%)	3 (20.0%)	2 (16.7%)	0.503
- Duration of mechanical ventilation (days)	12.0 (0.0-18.0)	0.0 (0.0-14.0)	0.0 (0.0- 16.0)	0.001*
CT findings on admission				
- Unremarkable	0 (0.0%)	0 (0.0%)	0 (0.0%)	-
- Brain edema	3 (17.6%)	15 (100.0%)	12 (100.0%)	0.562
- Hemorrhagic contusions	6 (35.3%)	5 (33.3%)	3 (25.0%)	0.847
- Fracture base of skull	3 (17.6%)	0 (0.0%)	2 (16.7%)	0.237
- Depressed fracture	3 (17.6%)	2 (13.3%)	1 (8.3%)	0.866
- Fissure fracture	4 (23.5%)	2 (13.3%)	2 (16.7%)	0.885
- SDH	1 (5.9%)	3 (20.0%)	1 (8.3%)	0.497
- EDH	4 (23.5%)	1 (6.7%)	2 (16.7%)	0.513
- ICH	3 (17.6%)	1 (6.7%)	5 (41.7%)	0.088
- IVH	1 (5.9%)	2 (13.3%)	2 (16.7%)	0.606
- SAH	3 (17.6%)	2 (13.3%)	1 (8.3%)	0.866
CT findings 48 hours after admission				
- Unremarkable	1 (5.9%)	1 (6.7%)	2 (16.7%)	0.662
- Brain edema	5 (29.4%)	1 (6.7%)	2 (16.7%)	0.293
- Hemorrhagic contusions	7 (41.2%)	2 (13.3%)	3 (25.0%)	0.246
- Fracture base of skull	3 (17.6%)	0 (0.0%)	2 (16.7%)	0.237
- Depressed fracture	0 (0.0%)	2 (13.3%)	0 (0.0%)	0.181
- Fissure fracture	4 (23.5%)	1 (6.7%)	2 (16.7%)	0.513
- SDH	2 (11.8%)	2 (13.3%)	1 (8.3%)	1
- EDH	1 (5.9%)	0 (0.0%)	1 (8.3%)	0.730
- ICH	8 (47.1%)	1 (6.7%)	5 (41.7%)	0.027*
- IVH	2 (11.8%)	2 (13.3%)	2 (16.7%)	1
- SAH	4 (23.5%)	1 (6.7%)	1 (8.3%)	0.409

Legend: cEEG=continuous electroencephalogram; CT=computerized tomography; NCSE=nonconvulsive status epilepticus; SDH=subdural hematoma; EDH=epidural hematoma; ICH=intracerebral hemorrhage; IVH=intraventricular hemorrhage; SAH=subarachnoid hemorrhage.
Data are presented as mean±SD or frequency (%). *Significant $p<0.05$.

Table 5. Comparison between studied groups regarding continuous EEG findings

	NCSE (n=17)	Possible NCSE (n=15)	No NCSE (n=12)	p
EDs>2.5 Hz/sec and continue at least 10 sec	9 (52.9%)	0 (0.0%)	0 (0.0%)	<0.001*
EDs≤2.5 Hz/sec	8 (47.1%)	11 (73.3%)	0 (0.0%)	0.001*
Continuous (semi-) RDTA between 0.5 and 4.0 Hz/sec	0 (0.0%)	4 (26.7%)	12 (100.0%)	<0.001*
Typical spatiotemporal evolution	3 (17.6%)	0 (0.0%)	0 (0.0%)	0.102
Subtle clinical phenomena	3 (17.6%)	0 (0.0%)	0 (0.0%)	0.102
Clinical response after IV AED	2 (11.8%)	0 (0.0%)	0 (0.0%)	0.325
EEG response after IV AED	4 (23.5%)	11 (73.3%)	0 (0.0%)	<0.001*
Fluctuation without definitive evolution	0 (0.0%)	4 (26.7%)	0 (0.0%)	0.014*

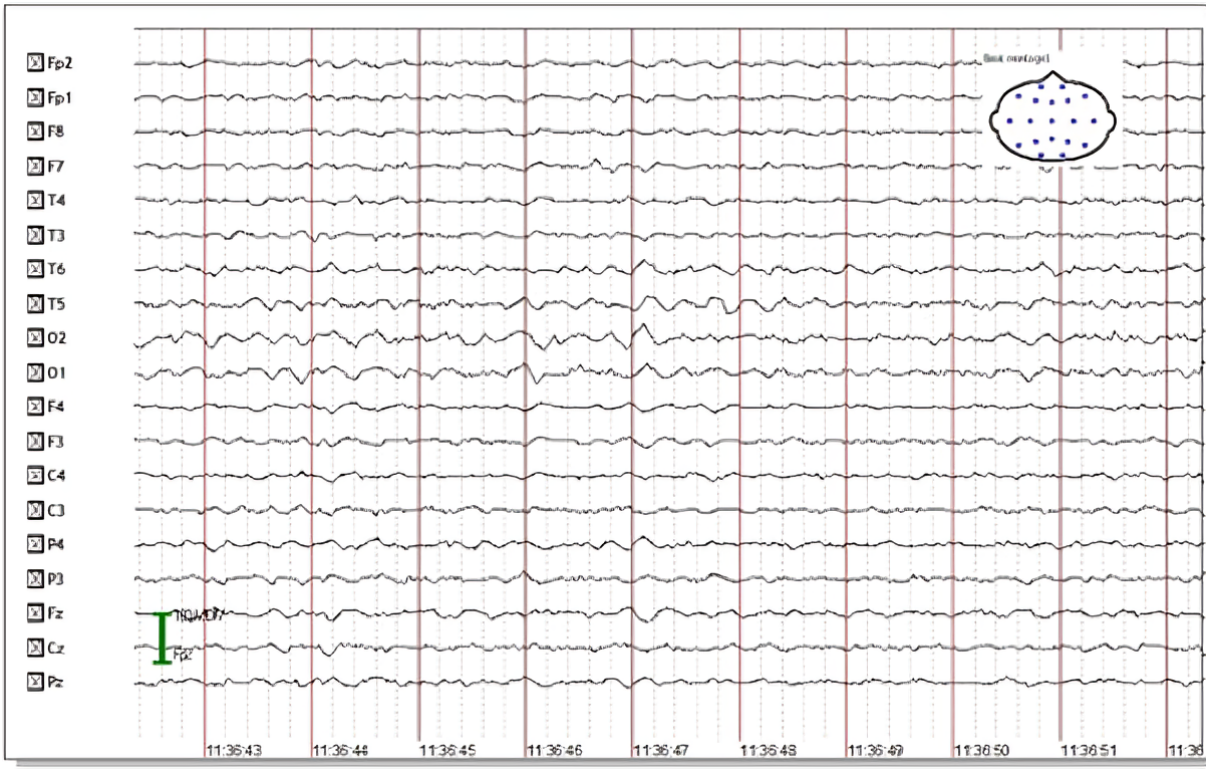
Legend: EEG=electroencephalogram; NCSE=nonconvulsive status epilepticus; Eds=epileptiform discharges; RDTA=rhythmic delta-theta activity; IV=intravenous; AED=antiepileptic drug.
Data are presented as mean±SD or frequency (%). *Significant p<0.05.

Table 6. Comparison between studied groups regarding patients' outcome on day 7, Glasgow Outcome Score Extended, and duration of hospital stay

	NCSE (n=17)	Possible NCSE (n=15)	No NCSE (n=12)	p
Deterioration of GCS	9 (52.9%)	1 (6.7%)	0 (0.0%)	0.001*
Mechanical ventilation	5 (29.4%)	1 (6.7%)	0 (0.0%)	0.072
Seizures	0 (0.0%)	0 (0.0%)	0 (0.0%)	-
Increase brain edema	7 (41.2%)	2 (13.3%)	0 (0.0%)	0.024*
Repeated doses of osmotherapy	7 (41.2%)	2 (13.3%)	0 (0.0%)	0.024*
Mannitol	5 (29.4%)	1 (6.7%)	0 (0.0%)	0.072
Hypertonic saline	2 (11.8%)	1 (6.7%)	0 (0.0%)	0.769
Neurosurgical intervention	4 (23.5%)	1 (6.7%)	0 (0.0%)	0.196
Neurological deterioration	17 (100.0%)	3 (20.0%)	0 (0.0%)	<0.001*
In-hospital mortality	6 (35.3%)	4 (26.7%)	2 (16.7%)	0.578
Glasgow Outcome Score Extended	2.0(1.0-4.0)	3.0(1.0-6.0)	5.0 (1.0-8.0)	0.014*
Hospital stays duration (days)	31.24±5.09	27.67±4.27	22.83±5.87	<0.001*

Legend: NCSE=nonconvulsive status epilepticus; GCS=Glasgow Coma Scale.
Data are presented as mean±SD or frequency. *Significant p<0.05.

Figure 1. EEG trace of trauma patient presented with DLC showed continuous semi-rhythmic delta activity, CT brain showed brain contusion, and the patient had subtle clinical phenomena, "twitches of the eyelids"



Legend: EEG=electroencephalogram; DLC=disturbed level of consciousness; CT=computerized tomography.

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