Acta Neurologica Indonesia
Department of Neurology, Faculty of Medicine Universitas Indonesia
ISSN 3032-6303

Case Report

Case Series: Risk and Clinical Manifestation of Non-Convulsive Status Epilepticus After Traumatic Brain Injury

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ABSTRACT

Traumatic brain injury occurs due to impacts on the head or penetrating injuries that result in damage to the brain. Traumatic brain injury can lead to complications, including post-traumatic seizures. Seizures following traumatic brain injury (TBI) present a complex interplay of various risk factors and determinants that significantly impact clinical outcomes and patient management, especially in non-convulsive status epilepticus. Non-convulsive status epilepticus (NCSE) is an underrecognized complication following traumatic brain injury (TBI), with potentially severe consequences for patient outcomes. Factors contributing to NCSE development include level of consciousness, injury severity, and cortical involvement. Clinical presentation is often subtle, ranging from altered mental status to focal neurological deficits. Early recognition through EEG monitoring is crucial, guiding targeted antiepileptic therapy to mitigate neuronal injury and improve outcomes Understanding these risk factors and clinical manifestations are crucial for effective surveillance, early intervention, and tailored treatment strategies aimed at mitigating the burden of seizures and improving the long-term prognosis of individuals with TBI.

Keywords: Non-Convulsive Status Epilepticus; Traumatic brain Injury; Clinical evaluation; Imaging

INTRODUCTION

Traumatic brain injury (TBI) stands as a persistent public health issue worldwide, impacting millions annually due to diverse causes like accidents, falls, assaults, and sports-related incidents. Traumatic brain injuries stem from impacts to the head or penetrating wounds that inflict harm on the brain. Typically, these injuries occur more frequently among younger individuals, with males exhibiting the highest prevalence. The causes of such injuries vary, encompassing falls, traffic accidents, physical assaults, and sports-related mishaps. Notably, traumatic brain injuries rank among the most common forms of trauma leading to disability and mortality in individuals aged between 1 and 45 years.[1,2]

While immediate consequences of TBI are typically evident and addressed, such as physical impairments and cognitive deficits,

attention to long-term outcomes, notably the onset of late seizures, becomes imperative. Post-traumatic seizures (PTS) can manifest weeks, months, or even years following the initial injury, significantly affecting individuals' well-being, and posing challenges to healthcare providers. These seizures can escalate the risk of secondary brain damage and pose challenges during critical recovery periods. Moreover, specific damage to the temporal lobe region might disrupt neural networks, heightening susceptibility to seizures or post-traumatic seizure occurrences (PTS). It's important to note that the onset of post-traumatic seizures can exhibit variability, spanning from early post-traumatic seizures (occurring within 7 days) to late post-traumatic seizures (manifesting after 7 days) following the traumatic event.[2-4] While convulsive status epilepticus (CSE) has been extensively studied in the context of TBI, non-convulsive status epilepticus (NCSE) remains an underrecognized entity with potentially severe consequences. This review aims to elucidate the risk factors and clinical manifestations of NCSE following TBI.

CASE ILLUSTRATION

Case 1

A 54-year-old woman experienced decreased consciousness for ten days after being involved in a motorcycle accident where she collided with a wall. Not wearing a helmet, she sustained a head injury, leading to loss of consciousness. Upon arrival at the hospital, she displayed confusion, incoherent speech, and hallucinations. Headaches primarily focused on the right eye and forehead, persisted despite medication, and a CT scan revealed brain bleeding. She was then transferred to a hospital with better-equipped facilities but continued to suffer from disorientation and recurring headaches.

After 8 days of treatment at the second hospital, the patient developed worsening vision and eye damage, leading to a referral to the Hospital. Upon arrival, her condition persisted with disorientation, the tendency to sleep, incoherent speech, and pain around the right eye. At RSCM, she continued to exhibit excessive sleepiness and difficulty in communication. Lab results were normal, but a non-contrast CT scan

revealed epidural hematoma. She was scheduled for an electroencephalogram. The patient had no history of significant medical conditions, malignancy, or substance use.

Vital signs and general status examination were normal. Glasgow Coma Scale assessment indicated disorientation (E3M6V4). Pupil examination showed difficulty with the right eye, with a round left pupil of 4mm diameter, fast-reacting direct light reflex, and an unassessable consensual light reflex with a cloudy cornea. Meningeal irritation tests were negative for neck stiffness, Laseque's sign, or Kernig's sign. Cranial nerve examination showed no palsy, and motor examination displayed no paralysis. The reflex examination was normal. Sensory and autonomic assessment couldn't be evaluated.

This patient underwent diagnostic tests including a non-contrast head CT scan and an electroencephalogram (EEG). Here are the results of the head CT scan without contrast with a single window. In the first head CT scan, epidural hematoma was observed in the right frontal region, and subarachnoid hemorrhage in the left temporal and occipital regions (Figure 1).

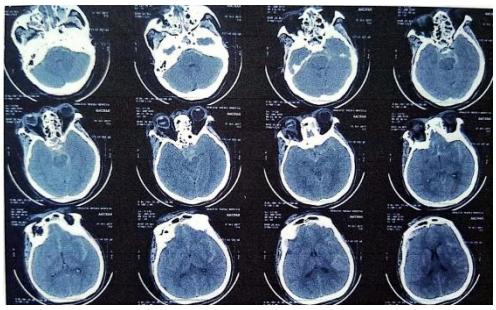


Figure 1. The first Head CT scan

Then, a head CT scan without contrast with bone window 10 days after the initial examination revealed epidural bleeding in the right frontal region, subarachnoid bleeding in the left parietal-temporal region, hypodense area in the white matter of the left parietal lobe, pneumocephalus in the left frontal region, multiple fractures in the right frontal bone, nasal bone, anterior and posterior walls of the right maxillary sinus, right sphenoid wing, right ethmoid wall, inferolateral wall and roof of the right orbit, right zygomatic bone, and left maxillary bone (Figure 2).

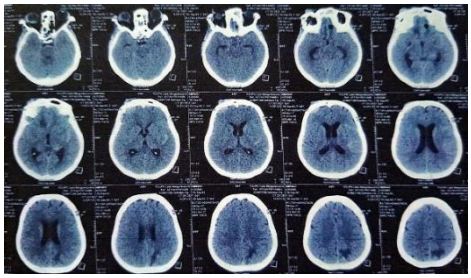


Figure 2. The second Head CT scan in Hospital

The third head CT scan without contrast was performed one week later, showing decreased epidural bleeding in the right frontal region and subarachnoid bleeding in the temporal lobe, suspected cerebral edema, fractures in the cranial and right maxillary bones, and bleeding in the vitreous of the right eye bulb (Figure 3).

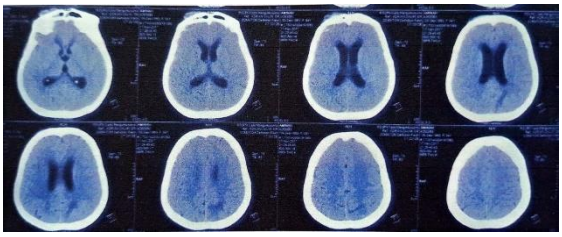


Figure 3. The third Head CT scan in Hospital

The results of the electroencephalogram (EEG) following intravenous administration of 10mg diazepam revealed abnormal EEG findings characterized by generalized slowing accompanied by multifocal epileptiform discharges in the bilateral frontal and temporal regions. There were bilateral TIRDA FIRDA patterns that responded to diazepam. These findings are consistent with non-convulsive status epilepticus.

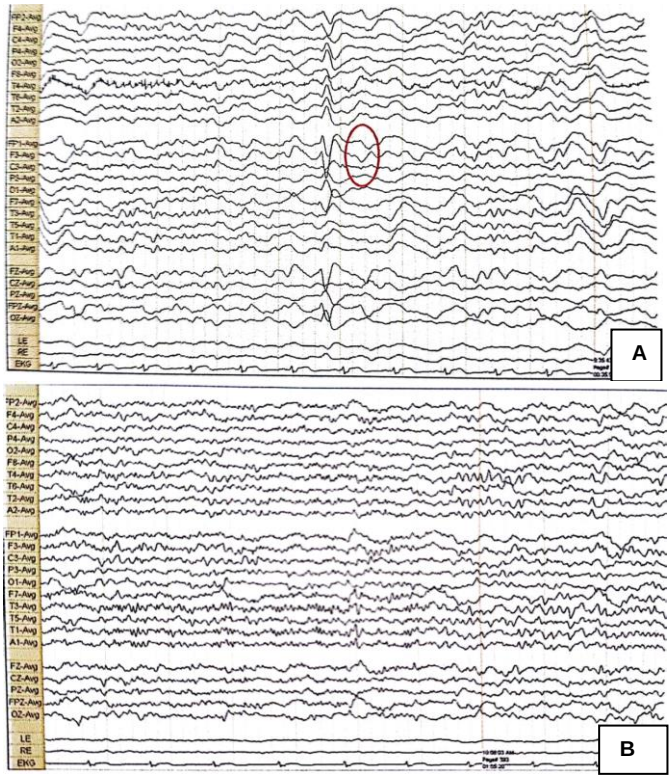


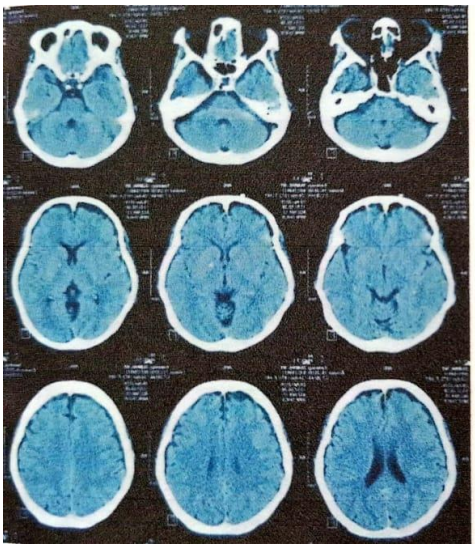
Figure 4. EEG in Hospital (A); EEG after diazepam 10mg (B)

Case 2

A 45-year-old woman presented to the Hospital with complaints of hallucinations and talking to herself for 3 days before admission. Six days earlier, she was hit by a motorcycle from the right side while crossing the street, hitting her head on the asphalt, and losing consciousness for 2 minutes. She was brought to Hospital conscious but vomiting three times. On the third day of treatment, she experienced hallucinations, including seeing animals, hearing voices, and talking to herself, becoming more

aggressive. A head CT scan showed normal results. She was prescribed aripiprazole 5mg once daily and olanzapine 5mg in the morning and 3mg at night and discharged. However, at home, she became increasingly aggressive, leading her family to take her to the emergency department at the Hospital. On arrival, she appeared restless, irritable, and disruptive, requiring restraint. She complained of pain on the right side of her face and has trouble sleeping but denied fever, double vision, blurred vision, seizures, numbness, or weakness. Vital signs were normal. General examination revealed edema of the right eyelid, periorbital edema, hematoma, and tenderness on the right periorbital and zygoma areas. Neurological examination showed a Glasgow Coma Scale score of E4M6V6, anisocoric pupils measuring 5mm/3mm, normal light reflexes, cloudy refractive media, and otherwise normal findings.

The result of the non-contrast head CT scan showed multiple fractures of the inferior wall of the right orbit with displacement of fracture fragments into the inferior aspect, accompanied by herniation of right orbital fat into the right maxillary sinus, consistent with a blowout fracture. There were comminuted fractures of the lateral wall of the right orbit, multiple linear fractures of the right zygomatic bone, right sphenoid wing, and right maxilla. Additionally, there was opacification noted in the right maxillary sinus (Figure 5).



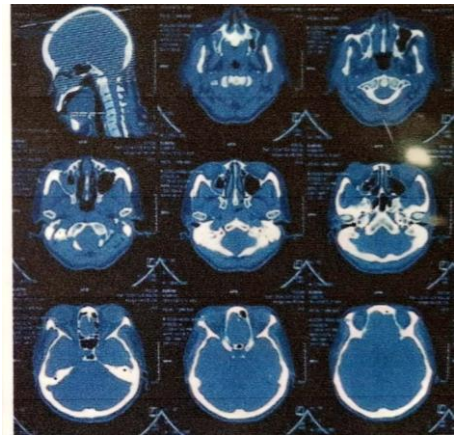


Figure 5. Head CT scan in hospital

The patient also underwent an EEG examination to evaluate for epileptic activity that might be causing cognitive impairment and sleep disturbances. The EEG examination revealed intermittent rhythmic slow delta waves appearing at F8-T6-T4-T2-A2, spreading to FP2-F4-C4. Following diazepam injection, there was a change in the baseline rhythm to alpha waves of 9-10 Hz, accompanied by the disappearance of slow waves. This led to the diagnosis of non-convulsive status epilepticus.

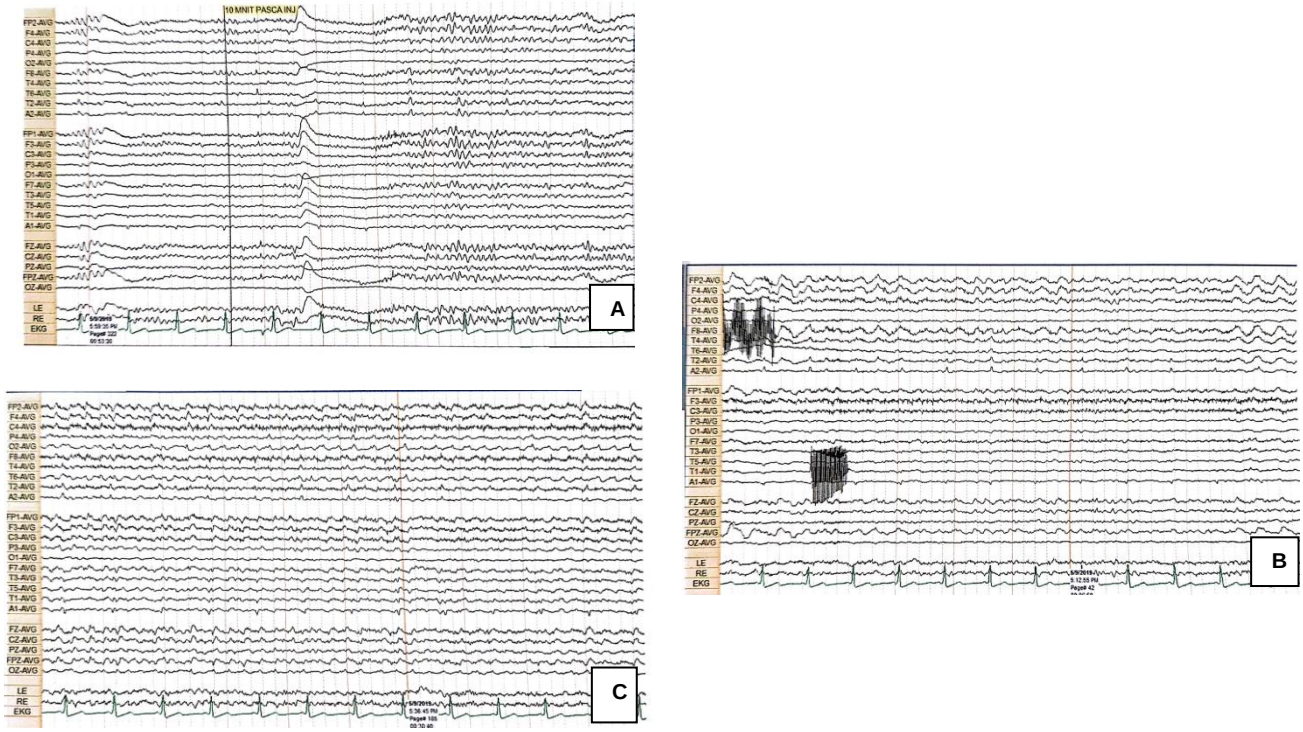


Figure 6. Intermittent slow wave activity (A), spike waves observed at T4-O2-P4 (B), absence of ictal waves, and slowing with an alpha baseline rhythm (C).

Seizures After Traumatic Brain Injury

In the United States, there are more than 2.5 million cases of traumatic brain injury (TBI) reported annually, with around 12% resulting in hospitalization or death. Post-traumatic seizures can occur at any time after a TBI. These seizures are classified based on when they occur after the injury: immediate seizures happen within 24 hours, early seizures occur between 24 hours and 7 days, and late seizures happen more than 7 days after the TBI. Late post-traumatic seizures (PTS), often referred to as post-traumatic epilepsy (PTE), are characterized by persistent changes in the brain's functioning due to biochemical processes and epileptic mechanisms triggered by the initial injury, leading to eventual clinical seizures. In severe TBI cases, the likelihood of experiencing late PTS is 10.0% five years after the injury, with early PTS occurring in 2.6% of cases.[5]

Understanding the mechanisms underlying late seizures is critical, as they can significantly impact an individual's quality of life and pose risks of secondary brain injury. Risk factors for late seizures following TBI are multifactorial, including injury severity, type of injury, age at the time of injury, presence of intracranial hemorrhage, contusions, and genetic predisposition. Furthermore, the temporal pattern of late seizures varies, with some occurring shortly after the injury while others may have a delayed onset. Early identification of individuals at risk and implementation of appropriate surveillance and preventive measures are essential for mitigating the burden of late seizures and improving long-term outcomes in TBI survivors.[6]

Risk Factors of Seizures After Traumatic Brain Injury

Several studies have revealed the incidence of post-traumatic seizures. Thapa A, et al.'s (2010) cohort study reported a post-traumatic seizure occurrence of 11.4%. The incidence of immediate, early, and late-onset post-traumatic seizures was 6.5%, 2.1%, and 2.7%, respectively. In this study, they demonstrated factors such as being in the pediatric age group (under 10 years old), being female, experiencing falls from height, enduring delayed loss of

consciousness, suffering from amnesia beyond 30 minutes, having comorbidities, and history of epilepsy. Delayed loss of consciousness amplifies the risk of PTS by 8 times, and patients with brain edema are 1.7 times more likely to develop PTS. The initial GCS score is correlated with the development of PTS, with each component of GCS serving as a predictor of seizure occurrence. Notably, a GCS score of less than 9 significantly heightens the risk of developing PTS, with a 2.3 times greater risk compared to those with a GCS score higher than 13.[7]

Another study by Najafi MR, et al. (2015) indicated a post-traumatic seizure occurrence of 6.33%. Among subjects, the incidence of early post-traumatic seizures and late post-traumatic seizures was 1.95% and 4.38%, respectively. The factor of severe head injury correlates with the occurrence of late post-traumatic seizures (PTS). The severity of trauma and the Glasgow Coma Scale (GCS) emerge as pivotal risk factors influencing the incidence of seizures in patients afflicted by traumatic brain injury (TBI). This correlation underscores the critical role played by the extent of trauma in predisposing individuals to post-traumatic seizures (PTS). Patients experiencing severe head injuries, indicated by a lower GCS score upon presentation, demonstrate a heightened susceptibility to PTS. Notably, the GCS score serves as a reliable indicator of neurological impairment following TBI, with lower scores indicative of more severe injury and increased risk of adverse outcomes such as seizures. Consequently, meticulous assessment and monitoring of trauma severity, as reflected by the GCS score, are imperative in identifying patients at elevated risk of developing post-traumatic seizures, facilitating timely interventions, and improving management strategies in TBI cases.[8]

Englander J, et al.'s (2003) study reported an incidence rate of 10% and post-traumatic seizures (PTS) correlate with patients who have contusion and subdural hematoma (SDH). This study conducted a prospective, observational study involving 647 subjects, with a follow-up period of 24 months following traumatic brain injury (TBI) incidents. It was revealed that within

24 months post-TBI, 66 subjects (10%) experienced late posttraumatic seizures.[9]

Several characteristics offer insight into the risk factors and probabilities associated with late post-traumatic seizures. Patients who experience early post-traumatic seizures face a 26.2% risk of developing subsequent late seizures. Moreover, individuals with a Glasgow Coma Scale (GCS) score ranging from 9 to 12 demonstrate a higher incidence of late post-traumatic seizures, with a probability of 24.3%. Notably, the presence of brain compression, specifically cisternal compression exceeding 5mm, is associated with a 25.8% likelihood of late seizure occurrence. Within 24 months, individuals with an initial Glasgow Coma Scale (GCS) score of 9-12 are at a 2.8 times higher risk of experiencing late seizures. Similarly, those with basal fracture compression exceeding 5mm are found to be at 2.21 times higher risk within the same period.[9]

Assessing the likelihood of late post-traumatic seizures via CT scans is essential. This method involves evaluating intracerebral conditions and contusions detected by CT scans. Multiple cortical contusions carry a probability of 25.2%, while subcortical contusions are associated with a probability of 33.4%. Furthermore, bilateral contusions in the frontal, temporal, or parietal regions also impact the likelihood of occurrence. Nevertheless, the highest probability is observed with bilateral parietal contusions, presenting a 66% likelihood of post-traumatic seizure occurrence. Over a span of 24 months, the likelihood of late seizures rises to 4.97 times for multiple cortical contusions and 2.79 times for multiple subcortical contusions. Bilateral contusions escalate the risk by 5 times, with bilateral parietal contusions demonstrating the highest risk at 5.35 for late seizure occurrence.[9]

Furthermore, factors such as ventriculostomy, penetration of bone and metal fragments, evacuated subdural hematomas (SDH), and multiple surgeries also contribute to the risk of late post-traumatic seizures (PTS). Ventriculostomy presents a 1.96-fold increased risk of late seizure occurrence, followed by a 3.94-fold elevated risk associated with

penetration of bone and metal fragments. Patients undergoing multiple surgeries face a 2.99-fold risk of experiencing seizures in the future. Additionally, evacuated subdural hematomas (SDH) entail a 2.72-fold risk. In this scenario, late seizures are prone to manifest within a 24-month timeframe.[9]

The following is a table of traumatic brain injury characteristics and the probability of late post-traumatic seizure from the study by Englander et al.[9]

Table 1. Characteristics and the probability of late post-traumatic seizure[9]

Variable	Sample Size n(%)	Individuals with PTS	Cumulative Probability (%)	Breslow Rank P value	Relative Risk at 24 mo
Cortical contusions: occurence				<.0001	
None	244(38)	10	5.9		1.00
Single	177(27)	10	8.2		1.38
Multiple	226(35)	46	25.2		4.97
Cortical contusions: (any location)				<.0001	
None	244(38)	10	5.9		1.00
Unilateral	258(40)	26	13.6		2.45
Bilateral	145(22)	30	25.4		5.05
Frontal contusions				<.0001	
No frontal contusio	382(59)	21	7.4		1.00
Unilateral	159(25)	23	20.1		2.63
Bilateral	106(16)	22	25.6		3.78
Temporal contusions				.0136	
No temporal contusio	449(69)	36	11.0		1.00
Unilateral	153(24)	20	15.9		1.63
Bilateral	45(7)	10	31.2		2.77
Parietal contusions				<.0001	
No parietal contusio	526(81)	43	10.9		1.00
Unilateral	105(16)	16	19.1		1.86
Bilateral	16(2)	7	66.0		5.35
Subcortical contusions				.0161	
None	567(88)	53	12.5		1.00
Single	57(9)	7	15.5		1.31
Multiple	23(3)	6	33.4		2.79
Ventriculostomy				.0065	
No	593(92)	56	12.6		1.00
Yes	54(8)	10	24.6		1.96
Fragment penetration				.0055	
None	621(96)	63	13.5		1.00
Bone	16(2)	0	0		-
Metal	5(1)	1	25.0		1.97
Bone+metal	5(1)	3	62.5		3.94

SDH				.0008	
None	369(57)	28	9.5		1.00
No evacuation	184(28)	19	15.3		1.36
Evacuation	92(15)	19	27.8		2.72
Operations				.0151	
No	383(59)	31	11.1		1.00
Single	225(35)	27	14.7		1.48
Multiple	33(5)	8	36.5		2.99

Risk factors for post-traumatic seizures (PTS) include falls from height, severe head injury (GCS<9), and certain medical conditions such as hypertension, diabetes mellitus, chronic respiratory disorders, cerebrovascular diseases, and kidney failure at the time of injury. Poor outcomes on the Glasgow Outcome Scale (GOS) also correlate with a higher risk of PTS, along with incidents of behavioral disturbances in patients with PTS after traumatic brain injury. Patients with severe head injury have a significantly higher risk of developing PTS compared to those with mild to moderate head injury. Although there is no significant relationship between age, gender, severity of head injury, and GCS score with the occurrence of early PTS and late PTS, late PTS is more common, particularly in patients with GCS 3-8. Brain contusions, especially multiple ones in cortical and subcortical areas, significantly increase the likelihood of late PTS.[7-9]

Non-Convulsive Status Epilepticus

Non-convulsive status epilepticus (NCSE) manifests as continuous or repetitive seizure activity, devoid of overt convulsions or tonic-clonic movements. Its presentation often includes altered consciousness, confusion, subtle behavioral changes, or other neurological symptoms, contrasting the dramatic convulsions characteristic of convulsive seizures. Diagnosing NCSE can be challenging due to its subtle symptoms, which may resemble other conditions like delirium or psychiatric disorders.

Typically, EEG monitoring is necessary to confirm the diagnosis, revealing abnormal electrical activity in the brain indicative of ongoing seizure activity. Treatment typically entails administering anti-epileptic

medications to halt the seizures and stabilize the patient's condition.

The clinical manifestations of NCSE can vary. Patients may experience classic clinical manifestations, such as complex partial seizures or simple partial seizures. In simple partial seizures, symptoms correspond to the location of seizure onset. In temporal lobe epilepsy, hallucinations and language disturbances may occur. Frontal lobe epilepsy may manifest as motor seizures. Additionally, parietal lobe epilepsy can lead to sensory disturbances, while occipital lobe epilepsy can cause visual disturbances. Moreover, patients may also experience alterations in consciousness, cognitive impairment, apnea, autonomic dysfunction, myoclonus, and organ dysfunction.[10]

The EEG criteria utilized for the diagnosis of NCSE may differ depending on the presence or absence of epileptic encephalopathy. In cases without known epileptic encephalopathy, the presence of epileptiform discharges surpassing 2.5 Hz or rhythmic delta/theta activity (> 0.5 Hz), coupled with observable clinical improvement after intravenous anti-seizure medications, subtle ictal phenomena coinciding with specific EEG patterns, or typical spatiotemporal evolution, serve as indicators. Conversely, in instances of NCSE with epileptic encephalopathy, the condition may be characterized by frequent or continuous generalized spike-wave discharges exhibiting heightened profusion or frequency compared to baseline EEG, alongside alterations in the clinical state, or observed clinical/EEG amelioration following intravenous benzodiazepines.[11]

The initial management for NCSE involves administering benzodiazepines orally, intramuscularly, or intravenously. Other medication options include phenytoin, valproic acid, or levetiracetam. If the patient's condition does not improve with medication, intubation with sedation becomes necessary.[11]

DISCUSSION

The case involves a 54-year-old woman who presented with a prolonged decrease in consciousness following a traffic accident. The

accident involved a collision while she was a passenger on a motorcycle, resulting in a head injury and subsequent loss of consciousness for 60-120 minutes. Upon hospital admission, she exhibited contact and communication abilities but was disoriented by people, places, and time. Initial assessment using the Glasgow Coma Scale (GCS) indicated Moderate Head Injury. CT scan findings revealed minimal epidural hematoma and subarachnoid hemorrhage, explaining her persistent altered consciousness over the initial ten days of hospitalization.

The patient displayed symptoms consistent with delirium, including fluctuating consciousness, daytime sleepiness, nighttime restlessness, and disorientation. Diagnostic criteria for delirium outlined in ICD-10 and DSM-5 were met, with evidence of altered consciousness, attention deficits, disorientation, short-term memory impairment, sleep-wake cycle disturbances, and underlying structural brain damage.

After eight days, the patient's consciousness worsened, characterized by increased sleepiness during both daytime and nighttime, with difficulty in awakening. Despite negative metabolic laboratory findings, a follow-up CT scan showed improvement in the epidural hematoma and subarachnoid hemorrhage, prompting consideration of nonconvulsive status epilepticus (NCSE).

Confirmation of NCSE was obtained through EEG, and the diazepam challenge resulted in improvement. Afterward, the patient received anti-epilepsy drug (AED) therapy with valproic acid at a loading dose of 1x800mg followed by a maintenance dose of 2x400mg. Two days after the administration of AED, the patient began to show improvement in consciousness. The patient became easier to awaken, with longer periods of wakefulness compared to before, started to make contact when spoken to, but still showed disorientation to time, people, and place. Therefore, it can be said that the worsening of consciousness changes in the patient was caused by the presence of NCSE (Non-Convulsive Status Epilepticus).

This differs from the second case where a 45-year-old woman

experienced hallucinations post-trauma. Hallucinations involve perceiving objects or events through the senses without external stimuli. They can stem from various factors such as psychotic disorders, delirium, seizures, focal disturbances, or visual impairments. In this case, hallucinations could be attributed to delirium or focal disturbances since psychiatric disorders and migraines were ruled out from history, and visual impairments were excluded from physical examination. The patient experienced complex visual hallucinations. Functional MRI revealed increased activity in the visual association areas (areas 18 and 19), particularly in the ventral extra striate cortex/V4 (area 19), with some activity noted in temporal and anterior cingulate areas, indicating structural disturbances cannot be ruled out. In addition, the patient also experienced auditory hallucinations, which could be attributed to various factors such as delirium, epilepsy, infarction, and brainstem disorders.[12,13]

Additionally, the patient exhibited restless agitation supporting the diagnosis of delirium on the third day of treatment. Hallucinations in delirium occur in visual and auditory cases. According to ICD-10, delirium is identified by alterations in consciousness and attention. This typically presents clinically with cognitive impairment, psychomotor changes, disrupted sleep patterns, and emotional disturbances. Cognitive impairment may manifest as perceptual disturbances or psychosis, while emotional disturbances may present as agitation or apathy. Meanwhile, according to DSM-5, delirium criteria include acute attentional disturbances and alterations in consciousness with cognitive impairment attributable to medical conditions.[14,15]

Delirium management constitutes emergent therapy based on the patient's delirium category. Delirium is categorized as mild, moderate, or severe. It is considered mild when the patient experiences attentional disturbances but remains cooperative, moderate when they disturb the surroundings but do not pose a danger to themselves or others, and severe when they become dangerous. In this case, the patient was categorized as having moderate delirium, exhibiting understanding but remaining restless

and uncooperative for examination, thus receiving 5mg intramuscular haloperidol. This aligns with delirium management involving psychotherapy and, when needed, pharmacological therapy. Pharmacological therapy choices for post-traumatic delirium are varied to suit the patient's needs.[16]

Delirium in trauma patients can have multiple causes, stemming either from the trauma itself or other medical conditions. In trauma cases, delirium can occur due to processes inducing molecular, biochemical, and cellular changes because of neuronal damage. This event correlates with secondary brain injury following trauma, which can occur from hours to days after the onset of the event. The three dominant pathophysiology contributing to delirium are neuroinflammation, failure of network connectivity, and structural damage.[17]

The patient underwent EEG, revealing intermittent rhythmic delta waves at F8-T6-T4-T2-A2, occasionally spreading to FP2-F4-C4, which changed to alpha waves after diazepam injection. Reviewing the EEG showed ictal waves at T4-O2-P4, indicating non-convulsive status epilepticus. Post-trauma seizures occur in 20-35% of cases, with non-convulsive status epilepticus in 8.8% of moderate-severe head injuries. Early or late seizures, occurring within the first week or after 7 days post-trauma, respectively, pose risks of recurrent episodes. The most common causes include skull fractures, penetrating injuries, contusions, and bleeding. Non-convulsive status epilepticus shares pathophysiology with convulsive status epilepticus but inhibits motor activity, involving neurotransmitter imbalances and inhibitory pathways. Diagnosis is challenging due to varied symptoms, including confusion, coma, lethargy, and agitation. EEG confirms non-convulsive status epilepticus, treated with benzodiazepines followed by phenytoin or alternatives if necessary. Following phenytoin therapy, the patient's sleep disturbances resolved, strengthening the suspicion of delirium secondary to non-convulsive status epilepticus. Antiepileptic therapy improved EEG findings, indicating a favorable prognosis for the patient.[18]

From the two cases above, clinical manifestations of non-convulsive status epilepticus in post-head injury patients can be observed. The patient's risk factors include brain contusion that can occur in the frontal or temporal areas, which can trigger seizures. Neurological status assessment is important to be conducted as an initial assessment, such as assessing the patient's GCS. In addition, a head CT scan is also necessary and should be done as early as possible before the patient's condition worsens. EEG examination is one of the important tools in confirming the diagnosis of the patient to differentiate whether the patient is experiencing delirium or non-convulsive seizures.

CONCLUSION

Non-convulsive status epilepticus (NCSE) following traumatic brain injury (TBI) presents a complex clinical challenge. This neurological complication underscores the intricate interplay between brain trauma and epileptic activity, posing significant risks to patient outcomes and recovery trajectories. The varied clinical manifestations of NCSE, ranging from subtle cognitive impairment to overt neurological deficits, necessitate a nuanced understanding and prompt recognition by healthcare providers. Timely diagnosis and intervention are paramount to mitigate the risk of long-term neurological sequelae and optimize patient management strategies. Overall, a comprehensive understanding of the risk factors and clinical manifestations is crucial in navigating the complexities of NCSE post-TBI, thereby enhancing prognostication and optimizing patient care pathways.

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