COURSE OF EPILEPTIC SEIZURES IN PATIENTS AFTER TRAUMATIC BRAIN INJURY: CLINICAL OBSERVATIONS OF 23 CASES

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Abstract:

Background: Post-traumatic epilepsy (PTE) is one of the most disabling long-term complications of traumatic brain injury (TBI). Despite progress in acute neurotrauma care, epileptic seizures remain a major cause of morbidity, drug resistance, and impaired quality of life among survivors.

Objective: To analyze the course, clinical features, and outcomes of epileptic seizures in patients with TBI.

Methods: A total of 23 patients with post-traumatic seizures were observed for a mean follow-up of 24 months. Clinical data, electroencephalography (EEG), and neuroimaging findings were assessed. Seizures were classified according to the International League Against Epilepsy (ILAE 2017). Treatment outcomes and the rate of drug-resistant epilepsy (DRE) were evaluated.

Results: Early post-traumatic seizures occurred in 30.4% of patients, while late seizures predominated (69.6%). Generalized tonic–clonic seizures were the most frequent type (51.2%). EEG abnormalities were present in 78.3% of cases, most often localized to the temporal lobes. Cortical contusions and hematomas were the leading imaging correlates. Levetiracetam was the most commonly prescribed antiepileptic drug (60.8%). Seizure control was achieved in 65.2% of patients, whereas 34.8% developed DRE. Psychiatric comorbidities (depression and anxiety) were reported in 26.1%.

Conclusion: Epileptic seizures after TBI are predominantly late in onset and frequently associated with temporal lobe lesions. Approximately one-third of patients develop drug resistance, underscoring the need for early risk stratification, individualized therapy, and preventive strategies to reduce the burden of PTE.

Keywords: traumatic brain injury; post-traumatic epilepsy; epileptic seizures; EEG; neuroimaging; drug-resistant epilepsy; quality of life

ТЕЧЕНИЕ ЭПИЛЕПТИЧЕСКИХ ПРИПАДКОВ У БОЛЬНЫХ ПОСЛЕ ЧЕРЕПНО-МОЗГОВОЙ ТРАВМЫ: КЛИНИЧЕСКИЕ НАБЛЮДЕНИЯ 23 СЛУЧАЕВ

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Аннотация:

Введение: Посттравматическая эпилепсия (ПТЭ) является одним из наиболее тяжелых и инвалидизирующих последствий черепно-мозговой травмы (ЧМТ). Несмотря на достижения в лечении острой травмы, эпилептические припадки остаются значимой причиной заболеваемости, фармакорезистентности и снижения качества жизни.

Цель: Изучить течение, клинические особенности и исходы эпилептических припадков у пациентов с ЧМТ.

Материалы и методы: В исследование включены 23 пациента с посттравматическими припадками. Средний период наблюдения составил 24 месяца. Анализировались клинические данные, ЭЭГ и нейровизуализация. Припадки классифицировались согласно ILAE (2017). Оценивались результаты лечения и частота развития фармакорезистентной эпилепсии (ФРЭ).

Результаты: Ранние припадки отмечены у 30,4% пациентов, поздние — у 69,6%. Наиболее частым типом были генерализованные тонико-клонические припадки (51,2%). ЭЭГ-изменения выявлены у 78,3% больных, преимущественно в височных отделах. По данным МРТ и КТ чаще регистрировались кортикальные ушибы и гематомы. Наиболее назначаемым препаратом был леветирацетам (60,8%). Контроль припадков достигнут у 65,2% пациентов, у 34,8% сформировалась ФРЭ. Психические расстройства (депрессия, тревожность) выявлены у 26,1%.

Заключение: Эпилептические припадки после ЧМТ преимущественно имеют позднее начало и ассоциированы с поражением височных долей. У трети больных формируется ФРЭ, что подчеркивает необходимость ранней стратификации риска, индивидуализированного лечения и профилактических мероприятий.

Ключевые слова: черепно-мозговая травма; посттравматическая эпилепсия; эпилептические припадки; ЭЭГ; нейровизуализация; фармакорезистентная эпилепсия; качество жизни

TRAVMATIK BOSH MIYA SHIKASTLANISHIDAN KEYINGI EPILEPTIK TUTQANOQLAR KECHISHI: 23 TA KLINIK HOLAT KUZATUVI

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Annotatsiya:

Kirish: Posttravmatik epilepsiya (PTE) bosh miya shikastlanishining (BMS) eng og‘ir va nogironlikka olib keluvchi asoratlaridan biridir. Neyrotravma bo‘yicha zamonaviy davolash choralariga qaramay, epileptik xurujlar bemorlar orasida yuqori kasallanish, dori vositalariga chidamlilik va hayot sifatining pasayishiga sabab bo‘lmoqda.

Maqsad: BMS kechirgan bemorlarda epileptik xurujlarning kechishi, klinik xususiyatlari va natijalarini tahlil qilish.

Materiallar va usullar: Posttravmatik xurujlar bilan og‘rigan 23 bemor o‘rganildi. O‘rtacha kuzatuv muddati 24 oy bo‘ldi. Klinik ma’lumotlar, EEG va neyroimaging natijalari baholandi. Tutqanoqlar ILAE (2017) tasnifi asosida guruhlandi. Davolash samarasi va dori chidamli epilepsiya (DChE) darajasi aniqlangan.

Natijalar: Erta tutqanoqlar 30,4% bemorlarda kuzatildi, kech tutqanoqlar esa 69,6% hollarda qayd etildi. Eng ko‘p uchraydigan tur — generalizatsiyalashgan toniko-klonik tutqanoqlar (51,2%). EEG o‘zgarishlari 78,3% hollarda aniqlanib, asosan chakka sohalarida qayd etildi. MRT va KTda kortikal lat yeyilishlari va gematomalar ustunlik qildi. Eng ko‘p buyurilgan dori — levetirasetam (60,8%). Tutqanoqlarni nazorat qilish 65,2% bemorlarda erishildi, 34,8% hollarda esa DChE rivojlandi. Psixik buzilishlar (depressiya, xavotir) 26,1% bemorlarda kuzatildi.

Xulosa: BMSdan keyingi epileptik tutqanoqlar ko‘pincha kech paydo bo‘ladi va chakka sohalarining shikastlanishi bilan bog‘liq. Har uchinchi bemorda DChE shakllanadi, bu esa erta xavf stratifikatsiyasi, individual yondashuv va profilaktik choralar zarurligini ko‘rsatadi.

Kalit so‘zlar: bosh miya shikastlanishi; posttravmatik epilepsiya; epileptik tutqanoqlar; EEG; neyroimaging; dori chidamli epilepsiya; hayot sifati

INTRODUCTION

Traumatic brain injury (TBI) represents one of the leading causes of morbidity, mortality, and long-term disability worldwide, with an annual incidence estimated to exceed 69 million cases globally [Maas et al. 2017]. Beyond the immediate neurological consequences, TBI is strongly associated with the development of late complications, among which post-traumatic epileptic seizures remain one of the most disabling. Epileptic seizures following brain injury not only impair neurological recovery but also exert a profound impact on psychosocial functioning, quality of life, and overall survival [Annegers et al. 1998; Lowenstein 2009]. Post-traumatic epilepsy (PTE) is defined as recurrent unprovoked seizures occurring after TBI and is considered the most common cause of symptomatic epilepsy in young adults [Pitkänen & Immonen 2014].

Epidemiological data suggest that approximately 10–25% of patients with severe TBI will eventually develop epilepsy, although the exact risk varies according to injury severity, type of brain lesion, and genetic susceptibility [Englander et al. 2003; Christensen 2009]. The natural history of post-traumatic seizures is typically divided into early seizures, occurring within the first week after trauma, and late seizures, manifesting after this interval. This distinction has important prognostic and therapeutic implications: early seizures are considered an acute symptomatic response to structural or metabolic derangements, whereas late seizures signify the establishment of epileptogenic networks and carry a substantially higher risk of chronic epilepsy [Frey 2003; Fisher et al. 2014].

From a pathophysiological standpoint, the mechanisms underlying epileptogenesis after TBI are multifactorial and remain incompletely understood. Experimental and clinical studies implicate a complex interplay between excitotoxic neuronal injury, gliosis, synaptic reorganization, blood–brain barrier disruption, neuroinflammation, and altered expression of ion channels and neurotransmitter receptors [Pitkänen & Engel 2014; Walker & White 2017]. These processes converge to generate a hyperexcitable neural environment capable of producing recurrent spontaneous seizures. Importantly, epileptogenesis may unfold over months or years, explaining why PTE often emerges as a delayed complication [Gupta et al. 2014].

Risk factors for PTE include the severity of the initial injury, presence of intracranial hematomas, cortical contusions, penetrating head injuries, depressed skull fractures, prolonged loss of consciousness, and early seizures themselves [Annegers et al. 1998; Haltiner et al. 1997]. Modern neuroimaging and electrophysiological methods have expanded our ability to identify structural and functional predictors of epileptogenesis. Lesions involving the temporal and frontal lobes, diffuse axonal injury, and hippocampal sclerosis have been consistently associated with higher epileptogenic potential [Engel et al. 2013]. In addition, genetic and molecular factors—such as polymorphisms in genes encoding for glutamate receptors, GABAergic proteins, or multidrug transporters—may contribute to individual vulnerability to epilepsy after trauma [Kwan et al. 2011].

The clinical spectrum of post-traumatic seizures is highly heterogeneous. Patients may present with focal aware seizures, focal seizures with impaired awareness, or secondarily generalized tonic–clonic seizures. The semiology often reflects the location of the traumatic lesion, but diffuse injuries can produce multifocal or generalized seizure patterns [Fisher et al. 2017]. Recurrent seizures frequently complicate neurological rehabilitation, delay return to work, and

increase the risk of psychiatric comorbidities such as depression and anxiety [Herman 2002]. Moreover, uncontrolled seizures are associated with increased risk of sudden unexpected death in epilepsy (SUDEP) [Devinsky 2016].

The therapeutic management of PTE remains a major clinical challenge. Although prophylactic administration of antiepileptic drugs (AEDs) such as phenytoin or levetiracetam can reduce the incidence of early seizures, randomized controlled trials have consistently shown no benefit in preventing late epilepsy [Temkin 2009]. Consequently, long-term prophylaxis is not routinely recommended, and treatment is generally initiated only after the occurrence of unprovoked seizures. Even then, a significant subset of patients develop drug-resistant epilepsy, defined by the International League Against Epilepsy (ILAE) as failure of adequate trials of two tolerated, appropriately chosen AEDs [Kwan et al. 2010]. This underscores the urgent need for improved preventive strategies and disease-modifying therapies aimed at halting epileptogenesis rather than merely suppressing seizures.

The burden of post-traumatic epilepsy extends beyond individual morbidity to encompass substantial socioeconomic consequences. Individuals with PTE often require prolonged medical care, experience reduced employability, and face stigmatization. In resource-limited settings, delayed access to specialized neurological services further exacerbates the prognosis [Beghi 2020]. Epidemiological studies in regions with high rates of road traffic accidents, military conflicts, and occupational injuries highlight the importance of integrating epilepsy prevention into broader trauma care and rehabilitation programs [DeGiorgio et al. 2019].

Despite decades of research, important gaps remain in our understanding of the natural history of epileptic seizures after TBI. Key unanswered questions include: which patients are most likely to progress from early seizures to chronic epilepsy; what biological markers can reliably predict epileptogenesis; and which interventions may alter the course of disease. Recent advances in neuroimaging, electrophysiology, and biomarker discovery offer new avenues for risk stratification and early intervention [Pitkänen et al. 2016]. Furthermore, experimental neuroprotective strategies targeting inflammation, oxidative stress, and synaptic remodeling hold promise for preventing or mitigating epilepsy in the aftermath of TBI [Walker et al. 2018].

Given the profound impact of epileptic seizures on the lives of patients with TBI, a comprehensive understanding of their epidemiology, pathogenesis, clinical manifestations, and management strategies is essential. This article aims to analyze the course of epileptic seizures in patients who have sustained TBI, synthesizing current knowledge from both clinical and experimental research. By delineating risk factors, temporal patterns, and prognostic implications, we seek to contribute to the ongoing effort to optimize diagnostic, therapeutic, and preventive approaches to this complex and challenging condition.

The aim of this study is to examine the course of epileptic seizures in patients after traumatic brain injury (TBI). It focuses on identifying temporal patterns and clinical features of post-traumatic seizures, assessing their impact on recovery and quality of life, and evaluating current treatment approaches in order to improve risk stratification and preventive strategies.

MATERIALS AND METHODS

This observational clinical study included **23 patients** diagnosed with post-traumatic epileptic seizures who were admitted to the neurology and neurosurgery departments between 2020 and 2024. All patients had a documented history of traumatic brain injury (TBI), confirmed by clinical examination and neuroimaging (CT or MRI). The primary aim was to evaluate the

temporal course, clinical characteristics, and treatment outcomes of epileptic seizures in this cohort.

Inclusion criteria were: (1) confirmed diagnosis of TBI; (2) at least one epileptic seizure occurring after the injury; (3) availability of complete clinical and imaging records. Exclusion criteria included prior history of epilepsy, metabolic encephalopathy, or seizures attributable to toxic or infectious causes.

Clinical Characteristics. The study population consisted of **23 patients** (15 males, 8 females), with a mean age of **34.7 ± 12.5 years** (range 18–59 years). The majority of cases were associated with **severe TBI** (56.5%), while moderate injuries accounted for 30.4% and mild injuries for 13.1%.

The leading mechanisms of trauma were road traffic accidents (52.1%), falls from height (26.0%), and domestic or occupational injuries (21.9%). The mean duration of follow-up was **24 months**.

Table 1. Baseline Demographic and Clinical Characteristics of Patients (n = 23)

Variable	Value
Total patients	23
Age (years), mean ± SD	34.7 ± 12.5 (range 18–59)
Sex (male/female)	15 / 8
Mechanism of trauma	Road traffic: 12 (52.1%) Falls: 6 (26.0%) Domestic/occupational: 5 (21.9%)
Severity of TBI	Severe: 13 (56.5%) Moderate: 7 (30.4%) Mild: 3 (13.1%)
Mean follow-up (months)	24

Seizure Classification and Temporal Distribution

A total of **41 seizures** were documented across the 23 patients during follow-up. Based on ILAE criteria, seizures were classified as focal aware, focal impaired awareness, and generalized tonic–clonic seizures.

Early post-traumatic seizures (EPTS), occurring within 7 days after injury, were observed in 7 patients (30.4%). Late post-traumatic seizures (LPTS), emerging after 7 days, were detected in 16 patients (69.6%).

Among seizure types, generalized tonic–clonic seizures predominated (51.2%), followed by focal impaired awareness seizures (31.7%), and focal aware seizures (17.1%).

Table 2. Distribution of Seizures in 23 Patients

Category	n (%)
Patients with early seizures	7 (30.4%)
Patients with late seizures	16 (69.6%)
Seizure type	
– Generalized tonic–clonic	21 (51.2%)
– Focal impaired awareness	13 (31.7%)
– Focal aware	7 (17.1%)

Diagnostic Evaluation. All patients underwent **electroencephalography (EEG)** and **neuroimaging**. EEG abnormalities were identified in 18 patients (78.3%), most frequently temporal or frontal epileptiform discharges. Neuroimaging revealed cortical contusions in 12

patients, intracranial hematomas in 8, and diffuse axonal injury in 3. Lesions involving the temporal lobes were strongly associated with the occurrence of late seizures.

Therapeutic Management. All patients received **antiepileptic drug (AED) therapy** after the first seizure. **Levetiracetam** was prescribed in 14 cases (60.8%), **Valproic acid** in 6 cases (26.0%), **Phenytoin** in 3 cases (13.2%).

During follow-up, seizure control was achieved in 15 patients (65.2%), while 8 patients (34.8%) experienced recurrent seizures despite therapy, fulfilling criteria for **drug-resistant epilepsy (DRE)**.

Table 3. Treatment and Outcomes

Variable	n (%)
AED prescribed	
– Levetiracetam	14 (60.8%)
– Valproic acid	6 (26.0%)
– Phenytoin	3 (13.2%)
Seizure control achieved	15 (65.2%)
Drug-resistant epilepsy (DRE)	8 (34.8%)

RESULTS

A total of **23 patients** with post-traumatic epileptic seizures were included. The mean age was **34.7 ± 12.5 years** (range 18–59), with a male predominance (15 men, 65.2%). The majority of cases resulted from **road traffic accidents** (52.1%), followed by **falls** (26.0%) and **domestic/occupational injuries** (21.9%). According to Glasgow Coma Scale (GCS) criteria, 13 patients (56.5%) sustained severe TBI, 7 (30.4%) moderate, and 3 (13.1%) mild injuries.

Table 4. Baseline Demographic and Clinical Characteristics of Patients (n = 23)

Variable	Value
Total patients	23
Age (years), mean ± SD	34.7 ± 12.5 (18–59)
Sex (male/female)	15 / 8
Mechanism of trauma	Road traffic: 12 (52.1%) Falls: 6 (26.0%) Domestic/occupational: 5 (21.9%)
Severity of TBI	Severe: 13 (56.5%) Moderate: 7 (30.4%) Mild: 3 (13.1%)
Mean follow-up (months)	24

Seizure Onset and Classification

A total of **41 seizures** were observed during follow-up. **Early post-traumatic seizures (EPTS)** occurred in 7 patients (30.4%). **Late post-traumatic seizures (LPTS)** developed in 16 patients (69.6%).

By seizure type (ILAE 2017 classification): **Generalized tonic–clonic seizures (GTCS):** 21 events (51.2%). **Focal impaired awareness seizures:** 13 events (31.7%). **Focal aware seizures:** 7 events (17.1%).

Table 5. Seizure Distribution in 23 Patients

Category	n (%)
Patients with early seizures	7 (30.4%)
Patients with late seizures	16 (69.6%)

Seizure type	
– Generalized tonic–clonic	21 (51.2%)
– Focal impaired awareness	13 (31.7%)
– Focal aware	7 (17.1%)

Electroencephalography (EEG): Abnormal epileptiform discharges were found in **18 patients (78.3%)**, with most localized to the **temporal (44.4%)** and **frontal (27.8%)** lobes. Multifocal abnormalities were seen in 4 patients (17.4%).

Neuroimaging (CT/MRI): Cortical contusions: 12 patients (52.1%). Intracranial hematomas: 8 patients (34.7%). Diffuse axonal injury: 3 patients (13.2%).

Lesions involving the **temporal lobes** were strongly associated with late seizures, observed in 10 out of 16 patients with LPTS.

All patients received antiepileptic drug (AED) therapy following their first seizure: **Levetiracetam** was prescribed in 14 cases (60.8%), **Valproic acid** in 6 cases (26.0%), **Phenytoin** in 3 cases (13.2%).

Seizure control was achieved in **15 patients (65.2%)**, while **8 patients (34.8%)** developed **drug-resistant epilepsy (DRE)**.

Table 6. Treatment and Seizure Outcomes

Variable	n (%)
AED prescribed	
– Levetiracetam	14 (60.8%)
– Valproic acid	6 (26.0%)
– Phenytoin	3 (13.2%)
Seizure control achieved	15 (65.2%)
Drug-resistant epilepsy (DRE)	8 (34.8%)

Psychiatric comorbidities (anxiety or depression) were diagnosed in 6 patients (26.1%). No cases of sudden unexpected death in epilepsy (SUDEP) were reported during follow-up.

DISCUSSION

This study provides a clinical overview of epileptic seizures following TBI in a cohort of 23 patients. The predominance of late seizures (70%) aligns with prior research demonstrating that late-onset events are the strongest predictor of chronic post-traumatic epilepsy (PTE) [Annegers et al., 1998; Englander et al., 2003]. The high frequency of generalized tonic–clonic seizures in our cohort mirrors international data and underlines the impact of diffuse cortical dysfunction in post-traumatic epileptogenesis [Lowenstein, 2009].

Pathophysiological mechanisms likely include excitotoxicity, blood–brain barrier disruption, neuroinflammation, and maladaptive synaptic reorganization [Pitkänen & Engel, 2014]. The strong association between temporal lobe lesions and seizure development corroborates experimental findings that mesial temporal structures are particularly vulnerable to epileptogenesis [Gupta et al., 2014].

Therapeutic implications are significant. While AEDs provided satisfactory seizure control in two-thirds of patients, one-third developed DRE. This proportion is consistent with broader epilepsy literature, where 30–40% of patients remain refractory despite modern pharmacotherapy [Kwan et al., 2010]. Our data reinforce the need for early risk stratification, close follow-up, and consideration of surgical or neuromodulatory interventions in drug-resistant cases.

Impact on recovery: Patients with late seizures required prolonged rehabilitation and showed higher rates of psychiatric comorbidities, echoing reports that epilepsy worsens post-TBI outcomes by impairing cognitive and psychosocial recovery [Herman, 2002].

Study limitations: The small sample size, retrospective elements, and relatively short follow-up (24 months) limit generalizability. Nonetheless, the findings highlight critical trends relevant to neurosurgical and neurological practice in similar cohorts.

Future directions should include biomarker-driven prediction models, advanced neuroimaging studies, and multicenter prospective trials aimed at developing disease-modifying interventions to prevent epileptogenesis after TBI.

CONCLUSION

Epileptic seizures remain one of the most significant and disabling complications of traumatic brain injury. In our cohort of 23 patients, late post-traumatic seizures predominated and were strongly associated with cortical and temporal lobe lesions. Generalized tonic–clonic seizures were the most frequent clinical presentation, and approximately one-third of patients developed drug-resistant epilepsy despite appropriate pharmacological therapy. These findings emphasize the importance of early recognition of high-risk patients, comprehensive neurodiagnostic evaluation, and individualized management strategies. Post-traumatic epilepsy not only complicates neurological recovery but also contributes to psychiatric comorbidities and reduced quality of life. Future studies should focus on developing predictive biomarkers and preventive interventions to reduce the burden of epilepsy among TBI survivors.

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