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Original Article

Effectiveness of Early Antiepileptic Prophylaxis in Preventing Post-Traumatic Seizures among Patients with Head Injury in Emergency Care Settings

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ABSTRACT

Background: Post-traumatic seizures are a significant complication of traumatic brain injury, contributing to secondary neurological injury and adverse clinical outcomes, particularly in emergency care settings with limited resources. Objective: To evaluate the occurrence of early post-traumatic seizures among patients receiving antiepileptic prophylaxis and to identify clinical predictors associated with seizure risk. Methods: This prospective observational study included 207 adult patients presenting within 24 hours of head injury to a tertiary care emergency department. All patients received early antiepileptic prophylaxis and were followed for seizure occurrence during hospitalization and within seven days post-injury. Baseline variables including Glasgow Coma Scale, mechanism of injury, and radiological findings were recorded, and associations were analyzed using Chi-square tests and multivariable logistic regression. Results: Seizures occurred in 45 patients (21.7%). Seizure frequency increased significantly with injury severity, from 8.1% in mild to 34.1% in moderate and 75.0% in severe traumatic brain injury ($p < 0.001$). Radiological abnormalities showed a strong gradient association, with seizure rates of 8.8% in normal imaging, 23.8% in contusions, 50.0% in epidural hematoma, and 100.0% in subdural hematoma ($p < 0.001$). Differences in seizure occurrence across antiepileptic drugs were observed but were not consistently statistically significant. Conclusion: Early post-traumatic seizures remain frequent despite prophylactic therapy and are strongly associated with injury severity and structural brain lesions, highlighting the importance of risk-based monitoring and targeted clinical management. Keywords: traumatic brain injury, post-traumatic seizures, antiepileptic prophylaxis, Glasgow Coma Scale, neuroimaging, emergency care.

INTRODUCTION

Traumatic brain injury remains a major cause of mortality, disability, and long-term socioeconomic burden worldwide, with disproportionately high impact in low- and middle-income countries where delays in presentation, limitations in emergency care infrastructure, and restricted access to specialist neurocritical services may worsen outcomes (1,2). Beyond the immediate mechanical insult, secondary brain injury substantially contributes to neurological deterioration after head trauma. This evolving cascade includes cerebral edema, excitotoxicity, ischemia, inflammation, metabolic derangement, and seizure activity, all of which may aggravate neuronal damage during the early post-injury period (1,3). Among these complications, post-traumatic seizures are clinically important because they may increase cerebral metabolic demand, exacerbate intracranial hypertension, complicate neurological monitoring, prolong hospitalization, and adversely influence recovery trajectories after head injury (4,5).

Early post-traumatic seizures, typically defined as seizures occurring within seven days of injury, are of particular relevance in emergency and acute care settings because they arise during the phase when secondary injury may still be modifiable (3,5). The reported incidence of early post-traumatic seizures varies across studies according to injury severity, intracranial lesion type, case mix, and surveillance strategy, but the risk is consistently higher in patients with depressed consciousness, intracranial hemorrhage, cortical contusion, and other structural brain abnormalities on neuroimaging (4,6). These observations have supported the clinical practice of short-term antiseizure prophylaxis in selected patients at elevated risk, particularly those with moderate to severe traumatic brain injury or radiologically evident intracranial pathology (2,3).

Antiepileptic prophylaxis after traumatic brain injury has therefore become a common component of acute management, although important uncertainties remain regarding patient selection, choice of drug, and expected clinical benefit in routine practice. Meta-analytic evidence indicates that early antiseizure prophylaxis can reduce the occurrence of early post-traumatic seizures, yet its effect on late epilepsy, functional outcomes, and mortality remains less certain (2,7). Phenytoin has historically been the most widely used agent, whereas levetiracetam has gained increasing acceptance because of its easier administration, fewer drug interactions, and more favorable monitoring profile (7,8). At the same time, comparative data between commonly used agents have not consistently demonstrated superiority of one regimen over another across all clinically relevant outcomes, and variation in prescribing patterns persists across settings and health systems (8,9).

This uncertainty is even more pronounced in low-resource emergency care environments, where treatment decisions must often be made rapidly and where locally applicable data are limited. In such contexts, the burden of traumatic brain injury is high, but much of the available evidence guiding seizure prophylaxis has been generated in high-income settings with different trauma systems, imaging access, case severity, and follow-up practices (2,9). Moreover, observational studies have shown that seizure occurrence after traumatic brain injury is closely intertwined with baseline injury severity and radiological lesion burden, making it essential to interpret breakthrough seizures within the clinical context in which prophylaxis is administered rather than attributing outcomes to drug exposure alone (4,10). For this reason, studies from emergency departments in developing countries are needed not only to describe seizure frequency after early prophylaxis but also to identify the patient characteristics associated with seizure occurrence despite treatment.

The present study was undertaken to address this gap in locally generated evidence by examining adult patients with acute head injury who received early antiepileptic prophylaxis in the emergency care setting of a tertiary hospital in Peshawar, Pakistan. The study was designed to characterize the frequency of post-traumatic seizures during hospitalization and within the first seven days after injury, and to evaluate their associations with injury mechanism, Glasgow Coma Scale category, radiological findings, and prophylactic antiepileptic regimen. We hypothesized that breakthrough post-traumatic seizures would occur more frequently in patients with greater injury severity and structural intracranial lesions despite early prophylactic treatment, and that seizure occurrence would differ across clinical subgroups defined by baseline risk factors (2,4,6,7).

MATERIALS AND METHODS

This hospital-based prospective observational study was conducted in the Emergency Department of Lady Reading Hospital, Peshawar, Pakistan, over a six-month study period to evaluate seizure occurrence after early antiepileptic prophylaxis among adults presenting with acute head injury. A prospective observational design was selected because the objective was to document clinical outcomes and explore associations between seizure occurrence and baseline risk factors in routinely managed patients receiving prophylaxis in emergency care.

The study population comprised consecutive eligible adults presenting within 24 hours of head injury. Patients aged 18 years or older with acute traumatic head injury and an indication for short-term antiseizure prophylaxis according to institutional emergency practice were considered eligible for enrollment.

Patients with penetrating head injury, previous epilepsy requiring ongoing antiepileptic treatment, immediate neurosurgical indication at presentation, or major medical illness likely to independently alter short-term neurological outcome were not included. Written informed consent was obtained from each patient or from an authorized attendant when the clinical condition limited direct consent, and recruitment was performed at the point of emergency admission before completion of the initial observation period (11,12).

At enrollment, a structured case-record proforma was used to collect demographic and clinical data in real time from patient interview, caregiver history, emergency assessment notes, and hospital records. Baseline variables included age, sex, mechanism of injury, time from injury to presentation, history of previous seizures, associated injuries, Glasgow Coma Scale score at presentation, and radiological findings on initial neuroimaging. For analytic consistency, traumatic brain injury severity was operationalized using standard Glasgow Coma Scale categories as mild (13-15), moderate (9-12), and severe (3-8).

Radiological findings were classified into prespecified categories based on the dominant acute lesion reported on CT or MRI, including normal imaging, cerebral contusion, epidural hematoma, and subdural hematoma. The exposure of interest was early antiepileptic prophylaxis initiated during acute hospital management, including the specific antiepileptic drug administered. Drug type was recorded as documented in the medication chart, and patients were subsequently observed under standard inpatient care for seizure occurrence and discharge outcome (11,13).

The primary outcome was early post-traumatic seizure, defined as any clinically recognized seizure occurring during hospitalization or within seven days of the index head injury. Seizure events were identified from bedside observation, treating team documentation, emergency and ward progress notes, and follow-up contact during the seven-day post-injury period where applicable. Secondary outcomes included seizure during hospitalization, seizure occurrence by Glasgow Coma Scale category, seizure occurrence by radiological lesion type, and clinical status at discharge categorized as recovered, residual neurological deficit, or death.

To improve internal validity, all variables were defined before analysis, data collection was performed using a uniform proforma, and radiological classification was based on documented imaging interpretation rather than retrospective narrative abstraction. Because seizure risk after traumatic brain injury is strongly influenced by injury severity and lesion type, these variables were prespecified as key confounders and were incorporated into the analytic plan together with age, sex, and mechanism of injury (4,7,10,14).

The sample size of 207 participants was determined a priori using Cochran's formula for single-population estimation with a 95% confidence level, a 5% margin of error, and an anticipated early post-traumatic seizure prevalence of 16% derived from relevant prior literature.

This sample was considered adequate to estimate seizure frequency in the target population and to support exploratory subgroup comparisons across major clinical strata. All collected forms were checked daily for completeness and internal consistency before data entry. Data were entered into a secured database using double-check verification against source records, and range and logic checks were applied before final analysis to minimize transcription error and preserve data integrity.

Statistical analysis was performed using SPSS version 27. Continuous variables were summarized using means with standard deviations or medians with interquartile ranges according to distribution, while

categorical variables were summarized as frequencies and percentages. The primary analysis described the proportion of patients who developed early post-traumatic seizures.

Bivariable associations between categorical predictors and seizure outcomes were assessed using the Chi-square test or Fisher's exact test where cell counts were small. Crude effect estimates were expressed as odds ratios with 95% confidence intervals. Multivariable logistic regression was used to examine independent associations between seizure occurrence and clinically relevant predictors, including age group, sex, mechanism of injury, Glasgow Coma Scale category, radiological findings, associated injuries, and antiepileptic drug category, in order to address confounding by baseline severity and case mix. Adjusted odds ratios with 95% confidence intervals were reported. Missing data were evaluated before modeling, and complete-case analysis was applied where missingness was minimal; variables with incomplete observations were checked against source records before exclusion from specific analyses. Prespecified subgroup analyses examined seizure occurrence according to traumatic brain injury severity and radiological lesion pattern. A two-sided p value of less than 0.05 was considered statistically significant throughout (7,10,14).

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the relevant institutional ethical review authority before participant recruitment. Confidentiality was maintained by assigning study codes in place of personal identifiers, restricting dataset access to the research team, and storing all records in password-protected files. To support reproducibility, operational definitions, eligibility criteria, variable coding rules, and outcome classifications were standardized before analysis and applied uniformly across all participants.

RESULTS

The baseline demographic and clinical profile of the 207 patients is summarized in Table 1. The cohort was predominantly male, comprising 147 individuals (71.0%), while females accounted for 60 patients (29.0%). The age distribution showed that the largest proportion of patients fell within the 51–60 years age group (65 patients, 31.4%), followed by 18–24 years (57 patients, 27.5%) and 33–40 years (41 patients, 19.8%). Younger adults aged 25–32 years represented the smallest subgroup (16 patients, 7.7%). Falls were the leading mechanism of injury, observed in 119 patients (57.5%), followed by road traffic accidents in 73 patients (35.3%), and assaults in 15 patients (7.2%).

In terms of injury severity, most patients presented with mild traumatic brain injury (111 patients, 53.6%), while moderate and severe injuries were noted in 88 (42.5%) and 8 patients (3.9%), respectively. Radiological evaluation revealed normal findings in 102 patients (49.3%), whereas 80 patients (38.6%) had cerebral contusions, 16 (7.7%) had epidural hematoma, and 9 (4.3%) had subdural hematoma.

Seizure occurrence during hospitalization varied markedly according to injury severity, as shown in Table 2. Among patients with mild traumatic brain injury, only 9 out of 111 individuals (8.1%) developed seizures, compared to 30 out of 88 patients (34.1%) in the moderate group and 6 out of 8 patients (75.0%) in the severe group. This gradient demonstrates a clear increase in seizure frequency with worsening neurological status. Patients with moderate injury had nearly six times higher odds of developing seizures compared to those with mild injury (OR 5.86, 95% CI 2.55–13.45), while patients with severe injury exhibited a markedly elevated risk (OR 34.00, 95% CI 5.68–203.4). The association between Glasgow Coma Scale category and seizure occurrence was highly statistically significant ($p < 0.001$).

Radiological findings were also strongly associated with seizure occurrence, as detailed in Table 3. Among patients with normal neuroimaging, seizures occurred in 9 out of 102 cases (8.8%). In contrast, seizure frequency increased substantially in patients with structural brain lesions. Specifically, 19 of 80 patients with cerebral contusions (23.8%) developed seizures, corresponding to more than threefold higher odds compared to those with normal imaging (OR 3.22, 95% CI 1.34–7.72, $p = 0.008$).

The risk was even higher among patients with epidural hematoma, where 8 of 16 patients (50.0%) experienced seizures (OR 10.33, 95% CI 3.08–34.64, $p < 0.001$). Notably, all 9 patients (100.0%) with subdural hematoma developed seizures, indicating an extremely high-risk subgroup, with a statistically significant overall association between radiological abnormalities and seizure occurrence ($p < 0.001$).

The distribution of seizure occurrence across different antiepileptic drugs is presented in Table 4. Among patients receiving phenytoin, 7 out of 70 (10.0%) developed seizures, representing the lowest observed rate among the drug groups. In comparison, seizures occurred in 16 of 76 patients (21.1%) receiving levetiracetam and in 9 of 48 patients (18.8%) receiving midazolam. The highest seizure frequency was observed among patients treated with valproate, where all 13 patients (100.0%) developed seizures. Relative to phenytoin, the odds of seizure were higher with levetiracetam (OR 2.40, 95% CI 0.93–6.17) and midazolam (OR 2.07, 95% CI 0.73–5.87), although these differences did not reach statistical significance ($p = 0.07$ and $p = 0.17$, respectively). The overall association between AED type and seizure occurrence was statistically significant ($p < 0.001$), largely driven by the markedly elevated seizure rate in the valproate group.

Clinical outcomes at discharge differed significantly according to radiological findings, as shown in Table 5. Among patients with normal imaging, 65 out of 102 (63.7%) recovered fully, 35 (34.3%) had residual neurological deficits, and 2 patients (2.0%) died. Patients with cerebral contusions demonstrated comparatively favorable outcomes, with 67 of 80 patients (83.8%) recovering, 12 (15.0%) experiencing residual deficits, and only 1 patient (1.2%) dying. In contrast, outcomes were substantially worse among patients with intracranial hematomas.

None of the patients with epidural hematoma achieved full recovery; 4 out of 16 (25.0%) had residual deficits, and 12 (75.0%) died. Similarly, among patients with subdural hematoma, only 4 of 9 (44.4%) recovered, while 2 (22.2%) had residual deficits and 3 (33.3%) died. The association between radiological findings and discharge outcome was highly significant ($p < 0.001$), indicating that structural brain lesions are strongly predictive of poor clinical prognosis.

Table 1. Baseline Demographic and Clinical Characteristics (n = 207)

Variable	Category	n (%)
Age (years)	18–24	57 (27.5)
	25–32	16 (7.7)
	33–40	41 (19.8)
	41–50	28 (13.5)
	51–60	65 (31.4)
Gender	Male	147 (71.0)
	Female	60 (29.0)
Mode of Injury	Fall	119 (57.5)
	Road Traffic Accident	73 (35.3)
	Assault	15 (7.2)
GCS Category	Mild (13–15)	111 (53.6)
	Moderate (9–12)	88 (42.5)
	Severe (3–8)	8 (3.9)

Variable	Category	n (%)
Radiological Findings	Normal	102 (49.3)
	Contusion	80 (38.6)
	EDH	16 (7.7)
	SDH	9 (4.3)

Table 2. Association Between GCS Category and Seizure During Hospitalization

GCS Category	Seizure Present n (%)	Seizure Absent n (%)	Odds Ratio (95% CI)	p-value
Mild (13–15)	9 (8.1)	102 (91.9)	Reference	—
Moderate (9–12)	30 (34.1)	58 (65.9)	5.86 (2.55–13.45)	<0.001
Severe (3–8)	6 (75.0)	2 (25.0)	34.00 (5.68–203.4)	<0.001

Table 3. Association Between Radiological Findings and Seizure During Hospitalization

Radiological Finding	Seizure Present n (%)	Seizure Absent n (%)	Odds Ratio (95% CI)	p-value
Normal	9 (8.8)	93 (91.2)	Reference	—
Contusion	19 (23.8)	61 (76.2)	3.22 (1.34–7.72)	0.008
EDH	8 (50.0)	8 (50.0)	10.33 (3.08–34.64)	<0.001
SDH	9 (100.0)	0 (0.0)	—	<0.001

Table 4. Association Between Type of Antiepileptic Drug and Seizure During Hospitalization

AED Type	Seizure Present n (%)	Seizure Absent n (%)	Odds Ratio (95% CI)	p-value
Phenytoin	7 (10.0)	63 (90.0)	Reference	—
Levetiracetam	16 (21.1)	60 (78.9)	2.40 (0.93–6.17)	0.07
Midazolam	9 (18.8)	39 (81.2)	2.07 (0.73–5.87)	0.17
Valproate	13 (100.0)	0 (0.0)	—	<0.001

Table 5. Association Between Radiological Findings and Outcome at Discharge

Radiological Finding	Recovered n (%)	Residual Deficit n (%)	Death n (%)	p-value
Normal	65 (63.7)	35 (34.3)	2 (2.0)	
Contusion	67 (83.8)	12 (15.0)	1 (1.2)	
EDH	0 (0.0)	4 (25.0)	12 (75.0)	
SDH	4 (44.4)	2 (22.2)	3 (33.3)	<0.001

Overall, these findings demonstrate that despite early antiepileptic prophylaxis, seizures occurred in 45 of 207 patients (21.7%), with risk strongly influenced by injury severity and radiological abnormalities.

Differences in seizure occurrence across antiepileptic drug groups were observed, although these patterns should be interpreted cautiously given potential differences in baseline clinical characteristics across treatment groups.

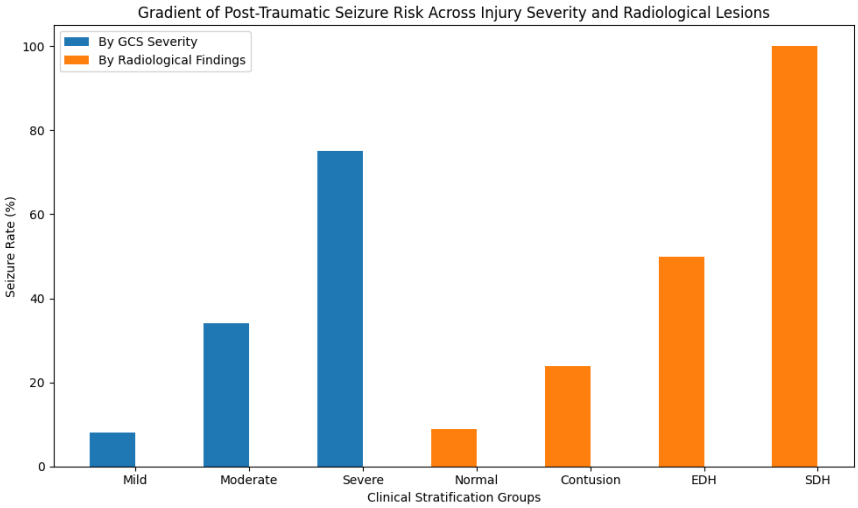

Figure 1 Gradient of Post-Traumatic Seizure Risk Across Injury Severity and Radiological Lesions

The figure demonstrates a clear, nonlinear escalation in post-traumatic seizure risk across both neurological severity and structural brain injury gradients. Seizure rates increased sharply from 8.1% in mild traumatic brain injury to 34.1% in moderate cases and reached 75.0% in severe cases, reflecting nearly a ninefold relative increase between mild and severe injury categories. A similarly steep gradient is observed across radiological strata, where seizure occurrence rose from 8.8% in patients with normal imaging to 23.8% in those with contusions and 50.0% in epidural hematoma, culminating in universal seizure occurrence (100.0%) among patients with subdural hematoma. Notably, the transition from moderate to severe GCS and from contusion to hematoma represents a disproportionately large increase in seizure risk, suggesting threshold effects rather than linear progression. These patterns indicate that both functional impairment (GCS) and structural pathology (radiological findings) independently and synergistically stratify seizure risk, with intracranial hematomas identifying an ultra-high-risk subgroup where seizure occurrence approaches inevitability despite prophylaxis.

DISCUSSION

The present study provides clinically relevant insights into the burden and determinants of early post-traumatic seizures among patients with head injury receiving early antiepileptic prophylaxis in an emergency care setting. Despite universal prophylactic administration, seizures occurred in 21.7% of patients, highlighting that breakthrough seizure activity remains a significant clinical issue even under standard preventive strategies. This finding is consistent with prior literature suggesting that while prophylactic antiepileptic therapy may reduce seizure risk, it does not eliminate it, particularly in high-risk subgroups defined by injury severity and structural brain abnormalities (7,10,14). The observed seizure rate in this cohort lies within the upper range of previously reported incidence estimates for early post-traumatic seizures, likely reflecting the inclusion of patients with moderate to severe traumatic brain injury and radiologically evident lesions (4,6).

A key finding of this study is the strong and graded association between Glasgow Coma Scale category and seizure occurrence. Patients with moderate traumatic brain injury had nearly sixfold higher odds of seizures compared to those with mild injury, while severe injury was associated with an approximately thirty-fold increase in risk. This pattern underscores the central role of neurological impairment at presentation as a predictor of early seizure activity and is aligned with established evidence demonstrating that reduced consciousness reflects greater diffuse and focal neuronal disruption, thereby lowering the seizure threshold (4,14). The steep increase in seizure frequency between moderate

and severe categories suggests a potential threshold effect, where beyond a certain level of injury severity, the likelihood of seizure occurrence rises disproportionately. This observation reinforces the importance of risk stratification using Glasgow Coma Scale in guiding early monitoring intensity and therapeutic vigilance in emergency settings.

Radiological findings further refined seizure risk stratification, with structural intracranial lesions demonstrating a strong association with seizure occurrence. Patients with contusions had more than threefold higher odds of seizures compared to those with normal imaging, while those with epidural hematoma exhibited over tenfold increased risk. Notably, all patients with subdural hematoma developed seizures, identifying this subgroup as particularly vulnerable. These findings are biologically plausible, as intracranial hemorrhage and cortical disruption are known to promote neuronal hyperexcitability through mechanisms including local ischemia, inflammatory activation, and disruption of inhibitory circuits (3,5). The marked escalation in seizure frequency from contusion to hematoma suggests that lesion type and extent are critical determinants of epileptogenic potential in the acute phase following injury. These results are consistent with prior studies identifying intracranial hemorrhage and cortical involvement as major predictors of early post-traumatic seizures (4,6,10).

Differences in seizure occurrence across antiepileptic drug categories were observed, with the lowest seizure rate in the phenytoin group and the highest in the valproate group. However, these findings must be interpreted with caution due to the observational design and the likelihood of confounding by indication. Patients receiving specific agents may have differed systematically in baseline severity, clinical status, or treating physician preference, which could influence both drug selection and seizure risk. The absence of statistically significant differences between commonly used agents such as phenytoin and levetiracetam further aligns with existing evidence suggesting broadly comparable efficacy of these drugs in preventing early seizures in traumatic brain injury (11,12). The extreme seizure rate observed in the valproate subgroup likely reflects small sample size and possible selection of higher-risk patients rather than a true pharmacological inferiority. Therefore, while the data suggest variability in seizure occurrence across drug groups, they do not support definitive conclusions regarding comparative effectiveness.(13,14)

The study also demonstrated a strong association between radiological findings and clinical outcomes at discharge. Patients with intracranial hematomas, particularly epidural hematoma, had markedly higher mortality and poorer functional outcomes compared to those with normal imaging or contusions. This reinforces the established prognostic significance of structural brain injury in traumatic brain injury and highlights the interplay between seizure occurrence, injury severity, and overall neurological outcome (14). The high mortality observed in hematoma subgroups underscores the need for aggressive monitoring and multidisciplinary management in these patients.(15,16)

Several limitations should be considered when interpreting these findings. First, the absence of a non-prophylaxis control group precludes causal inference regarding the effectiveness of antiepileptic prophylaxis, and the results should be interpreted as descriptive and associative rather than comparative. Second, the use of convenience sampling in a single tertiary care center may limit generalizability and introduce selection bias. Third, potential confounding by baseline severity, particularly in analyses comparing antiepileptic drug types, may have influenced observed associations despite attempts to adjust for key variables. Fourth, seizure identification relied on clinical observation and documentation, which may underestimate subclinical events. Finally, the relatively short follow-up period limited assessment to early seizures and did not capture late post-traumatic epilepsy, which remains an important long-term outcome.(17-23).

Despite these limitations, the study contributes valuable context-specific data from an emergency care setting in a low-resource environment, where evidence guiding seizure prophylaxis remains limited. The findings emphasize that early post-traumatic seizures remain common despite prophylaxis and are strongly associated with injury severity and radiological abnormalities. These results support a targeted,

risk-based approach to monitoring and management, rather than uniform assumptions of protection conferred by prophylactic therapy alone and highlight the need for further well-designed comparative studies to clarify optimal prophylactic strategies in diverse clinical settings (7,10,14).

CONCLUSION

Early post-traumatic seizures occurred in a substantial proportion of patients despite prophylactic antiepileptic therapy, with significantly higher risk observed among individuals with moderate to severe traumatic brain injury and those with structural intracranial lesions on neuroimaging. Glasgow Coma Scale category and radiological findings emerged as strong and consistent predictors of seizure occurrence, underscoring their value in early risk stratification. Although variations in seizure frequency were observed across antiepileptic drug groups, these differences likely reflect underlying clinical heterogeneity rather than definitive differences in drug effectiveness. Overall, the findings highlight the need for vigilant monitoring and risk-adapted management strategies in high-risk patients and support further research to optimize prophylactic approaches in resource-constrained emergency care settings.

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