Review article

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Post-traumatic seizures and post-traumatic epilepsy after traumatic brain injury: current approaches to prevention, diagnosis, and risk prediction

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Objective: To summarize current international and national evidence regarding the mechanisms underlying posttraumatic seizures and posttraumatic epilepsy following traumatic brain injury, to identify the main clinically relevant risk factors, and to outline approaches to seizure prophylaxis, neurophysiological monitoring, and prediction of long-term outcomes.

Materials and methods: Publications from the PubMed, Scopus, and Web of Science databases, published predominantly between 2020 and 2025, as well as relevant national sources, were analyzed. The review included clinical practice guidelines, systematic reviews, meta-analyses, and key original studies addressing posttraumatic seizures and epileptogenesis after traumatic brain injury.

Results: Early posttraumatic seizures were found to be primarily associated with acute metabolic and structural brain disturbances, whereas late seizures are usually considered a clinical manifestation of established posttraumatic epilepsy resulting from prolonged processes of epileptogenesis, neuroinflammation, microglial activation, and synaptic network remodeling. Prophylactic use of antiseizure medications is associated with a reduction in the incidence of early posttraumatic seizures but does not prevent the development of posttraumatic epilepsy. Electroencephalography and continuous EEG monitoring play an important role in risk stratification and in the detection of subclinical (nonconvulsive) seizures.

Conclusions: Management of patients with traumatic brain injury and posttraumatic seizures requires a differentiated multidisciplinary approach integrating clinical, neurophysiological, and neuroimaging risk factors. Further research should focus on improving prediction of posttraumatic epilepsy and identifying validated biomarkers of epileptogenesis.

Keywords: *traumatic brain injury; posttraumatic seizures; posttraumatic epilepsy; electroencephalography; seizure prophylaxis*

Introduction

Traumatic brain injury (TBI) remains one of the leading causes of persistent neurological impairment, disability, and symptomatic epileptic seizures in the adult population. According to contemporary epidemiological studies, the prevalence of TBI continues to increase, which is associated with road traffic injuries, urbanization, occupational trauma, and armed conflicts [1].

According to estimates from the Global Burden of Disease study, approximately 20.8 million new cases of TBI were registered worldwide in 2021, whereas the total number of individuals living with the consequences of traumatic head injury reached approximately 37.9 million. Mild TBI predominates (70–90%), whereas moderate and severe injuries are less common but are associated with the highest risks of mortality, disability, and long-term neurological complications [2–5].

Among the most clinically significant complications of TBI are post-traumatic seizures and post-traumatic epilepsy. Post-traumatic seizures not only complicate the acute phase of injury but also adversely affect recovery processes, are associated with an increased risk of secondary brain injury, prolonged hospitalization, and reduced long-term quality of life [6, 7].

According to various studies, early post-traumatic seizures occur in 2–10% of patients, depending on injury severity and the clinical population studied [8, 9]. The risk of developing delayed post-traumatic epilepsy after TBI is substantial and increases with injury severity. In some patients with severe TBI, the risk of epilepsy may exceed 12–15% during subsequent years of follow-up [10, 11]. Several reviews have summarized data regarding the incidence and risk factors of post-traumatic epilepsy across different age groups and clinical subpopulations [12, 13].

It has been established that seizure development after TBI is associated with poorer functional outcomes and an increased risk of long-term neurological complications, including cognitive impairment and dementia [14, 15]. Studies by leading researchers

emphasize the necessity of a differentiated approach to the prevention of post-traumatic seizures, timely implementation of EEG monitoring, and interdisciplinary management of patients with severe TBI [16, 17].

Objective: To summarize current international and national evidence regarding the mechanisms underlying post-traumatic seizures and post-traumatic epilepsy following traumatic brain injury, to identify the principal clinically significant risk factors, and to outline current approaches to seizure prophylaxis, neurophysiological monitoring, and prediction of long-term outcomes.

Materials and methods

A literature search was conducted using the PubMed and MEDLINE databases, as well as specialized Ukrainian scientific publications. The analysis included clinical practice guidelines issued by professional societies, systematic reviews, meta-analyses, cohort studies, and review articles published predominantly between 2020 and 2025, together with seminal studies of conceptual importance for understanding post-traumatic epileptogenesis.

The inclusion criteria comprised publications addressing post-traumatic seizures, post-traumatic epilepsy, seizure prophylaxis after TBI, the use of electroencephalography (EEG) and continuous EEG monitoring, as well as prediction of neurological outcomes following traumatic brain injury. In total, 31 sources were analyzed, including both contemporary publications and classical studies that established the principal approaches to this problem.

In contemporary clinical practice, post-traumatic seizures are generally classified as early (within 7 days after injury) and late (occurring more than 7 days after injury) [18]. Several clinical and neurosurgical publications describe seizures occurring within the first 24 h after TBI as manifestations of acute brain injury associated with primary structural and metabolic disturbances [7, 18, 19]. In the present review, such seizures are considered within the category of early post-traumatic seizures.

Early post-traumatic seizures are generally associated with acute structural and metabolic alterations, among which cerebral edema, intracranial hemorrhage, exposure of cortical tissue to blood products, disruption of ionic homeostasis, and ischemic injury play central roles [19]. These changes occur more frequently in patients with severe TBI, penetrating injuries, and intracranial hemorrhages and are associated with an increased risk of secondary brain injury and potentially poorer short-term treatment outcomes [8, 9].

Late post-traumatic seizures are regarded as a clinical manifestation of established or evolving post-traumatic epilepsy caused by prolonged processes of epileptogenesis, neuroinflammation, and neuronal network remodeling [20–22].

The principal risk factors for the development of post-traumatic seizures/post-traumatic epilepsy include severe TBI, cortical injuries (contusions), intracranial hemorrhages (particularly subdural hemorrhages), diffuse or multifocal traumatic brain injury, as well as neurosurgical interventions, which may serve as markers of injury severity and structural brain damage [10, 13, 19, 21, 23]. Particularly high risks of post-traumatic epilepsy are observed in patients with cortical contusions and other structural cortical injuries, especially in the presence of intracranial hemorrhage. This association is believed to result from prolonged epileptogenic processes and neuronal network remodeling within the area of traumatic injury [13, 20, 21, 24].

Electroencephalography performed at hospital admission after severe TBI may be useful for stratification of the risk of post-traumatic epilepsy [25].

According to current international clinical guidelines, prophylactic administration of antiseizure medications in patients with moderate-to-severe TBI who are at high risk of seizures significantly reduces the incidence of early post-traumatic seizures. However, convincing evidence supporting the effectiveness of such therapy in preventing the long-term development of post-traumatic epilepsy has not been demonstrated [25–27].

Meta-analyses conducted in recent years have demonstrated comparable efficacy of phenytoin and levetiracetam for the prevention of early post-traumatic seizures. Levetiracetam is characterized by a more favorable safety and tolerability profile, which is of particular importance in patients with polytrauma and concomitant somatic disorders [26–28].

The first post-traumatic seizure occurring during the acute phase of TBI is regarded as an acute symptomatic seizure and requires a therapeutic approach focused on correction of the acute underlying cause and seizure control rather than a purely prophylactic strategy [29–31].

Decisions regarding continuation or initiation of long-term antiseizure therapy after a seizure occurring during the acute phase of TBI should be made on an individual basis, taking into account clinical risk factors and neuroimaging findings associated with an increased risk of post-traumatic epilepsy [13, 21].

Conclusions

1. Post-traumatic seizures are a common and clinically significant complication of traumatic brain injury (TBI).

2. Prophylactic antiseizure therapy is effective only in preventing early post-traumatic seizures and does not reduce the risk of developing post-traumatic epilepsy.

3. Electroencephalography and continuous EEG monitoring play an important role in the detection of subclinical (nonconvulsive) seizures and in risk stratification for post-traumatic epilepsy.

4. Management of patients after TBI requires a differentiated multidisciplinary approach involving both neurologists and neurosurgeons.

Clinical recommendation

Prophylactic antiseizure therapy should be administered to patients with moderate-to-severe TBI who are at high risk of early post-traumatic seizures, including those with cortical injuries, intracranial hemorrhages, penetrating trauma, or a history of neurosurgical interventions.

Levetiracetam is considered the preferred agent at a dosage of 500–1000 mg twice daily, administered either intravenously or orally. Phenytoin may be used

as an alternative, with a loading dose of 15–20 mg/kg followed by maintenance therapy.

The duration of prophylactic therapy should not exceed 7 days in the absence of clinical or EEG evidence of seizure activity.

For patients with severe TBI admitted to intensive care units, the use of EEG and continuous EEG monitoring is recommended for early detection of subclinical (nonconvulsive) seizures and optimization of treatment strategies.

Disclosure information

Conflict of Interest

The authors declare no conflict of interest.

Ethical Standards

This article is a literature review; therefore, approval by Ethics Committee was not required.

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