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Seizure syndrome in stroke and traumatic brain injury: Incidence and treatment outcomes in the intensive care unit

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Abstract. Seizure syndrome is one of the most common and complex neurological complications in patients with acute brain injuries, particularly following stroke and traumatic brain injury. This complication significantly worsens clinical outcomes, increases the risk of mortality, and prolongs patients' length of stay in the intensive care unit. This study aimed to determine the frequency of seizure syndrome in patients with stroke and traumatic brain injury, to analyse the main causes of its development, and to evaluate the efficacy and safety of antiepileptic therapy in order to improve treatment approaches and clinical outcomes. A comprehensive literature review was conducted using 50 relevant scientific publications from PubMed, Scopus, Web of Science and Google Scholar databases. The selection was carried out in accordance with PRISMA guidelines and included studies with clearly defined patient populations with stroke or traumatic brain injury, data on seizure frequency, antiepileptic drug use, and statistically analysed results published in peer reviewed journals. Published studies indicated that seizure syndrome occurs in approximately 15%-30% of patients after stroke and in 20%-40% of individuals with traumatic brain injury. Early seizures (within the first seven days) are consistently reported in approximately 7% of patients after stroke and are associated with a poorer prognosis. The literature review identified multifactorial pathophysiological mechanisms, including primary structural brain tissue damage, secondary metabolic disorders, neuroinflammation, and an imbalance of neurotransmitter systems. Data from numerous studies demonstrated a high efficacy of modern antiepileptic therapy – particularly levetiracetam and lorazepam – estimated at 70%-85% with a favourable safety profile. The binding of levetiracetam to synaptic vesicle protein 2A is widely regarded as a key mechanism of seizure control, while lorazepam remains a first-line drug for the acute management of seizures due to its enhancement of GABAergic transmission

Keywords: antiepileptic therapy; levetiracetam; lorazepam; neurological complications; neuroinflammation

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Introduction

Seizure syndrome is one of the most common and clinically challenging neurological complications in patients with acute brain injuries, particularly following stroke and traumatic brain injury (TBI). Its occurrence significantly worsens the clinical course, increases the risk of mortality and long-term disability, and prolongs stays in the intensive care unit. The relevance of this issue stems from the heterogeneity of pathophysiological mechanisms underlying seizures in different types of brain injury and the persistent difficulty in selecting optimal anticonvulsant therapy, which remains limited by insufficient evidence and variability in clinical practice.

The problem of seizure syndrome in acute brain injuries, particularly following stroke and TBI, has been widely examined in the scientific literature. In the review by M. Galovic *et al.* [1], the authors summarised current findings on the epidemiology, biomarkers, and management of seizures and poststroke epilepsy. Their research emphasised the high incidence of early and late seizures, the central role of cortical structural injury, and the importance of emerging prognostic markers for identifying patients at risk of developing epilepsy after stroke. In a multicentre matched study conducted by C. Ferreira-Atuesta *et al.* [2], the authors analysed the frequency of seizures following ischaemic stroke and the clinical factors associated with their occurrence. They demonstrated that seizures are more common in patients with severe neurological deficits, cortical involvement, and haemorrhagic transformation, underscoring the need for individualised monitoring and preventive measures in high-risk patient groups. A systematic review and meta-analysis by A. Nandan *et al.* [3] synthesised data on the incidence of post-stroke seizures and epilepsy, as well as the principal risk factors contributing to their development.

The researchers reported a significantly increased risk of seizures in patients with cortical lesions, haemorrhagic strokes, and younger age, providing important insights for clinical risk stratification. Complementing these findings, S. Misra *et al.* [4] conducted a further large systematic review and meta-analysis that evaluated the outcomes of patients experiencing post-stroke seizures. Their results demonstrated clear associations between seizures and higher mortality, poorer functional outcomes, and an increased need for long-term rehabilitation services. A separate research focus has addressed the development of tools for predicting post-stroke epilepsy. In the study by Z. Wang *et al.* [5], the authors developed an automated deep learning model for predicting poststroke epilepsy in patients with intracerebral haemorrhage using non-contrast computed tomography imaging. Their study demonstrated the potential of artificial intelligence to enable early identification of high-risk patients, allowing more targeted application of anticonvulsant therapy and more efficient resource allocation in intensive care settings. The rehabilitation phase has also attracted considerable attention. In the study by M. Scarpino *et al.* [6], researchers assessed stroke-related epilepsy during inpatient post-stroke rehabilitation. They found that post-stroke epilepsy significantly complicates the rehabilitation process, reduces functional independence, and prolongs the duration of hospitalisation. These observations highlighted that seizures are clinically relevant not only in the acute stage but throughout the recovery period.

The effectiveness and safety of anticonvulsant therapy in acute brain injury settings were investigated in the PEACH randomised, double-blind, placebo-controlled phase III trial by L. PeterDerex *et al.* [7]. This study evaluated prophylactic levetiracetam for seizure prevention during the acute phase of intracerebral haemorrhage. The trial provided important data regarding the potential benefits and limitations of routine prophylactic therapy, as well as the drug's safety profile in critically ill neurological patients. Ukrainian clinical practice is guided by medical and technological documents approved by the Order of the Ministry of Health of Ukraine No. 276 [8], which outline standardised approaches to the diagnosis and treatment of epilepsy. Although these guidelines are not specific to seizures in stroke or TBI, they provide a regulatory framework for the use of modern anticonvulsant medications – such as levetiracetam and lorazepam – and may be adapted for seizure management in intensive care units.

Collectively, the reviewed studies demonstrated substantial advances in understanding the epidemiology, risk factors, and outcomes of seizure syndrome following stroke, as well as ongoing efforts to develop predictive models and evaluate anticonvulsant therapy. However, the majority of studies have focused primarily on stroke populations. Research addressing seizure syndrome specifically in patients with TBI and within intensive care settings remains comparatively limited, highlighting the need for further investigation to optimise diagnostic strategies and therapeutic interventions for this patient group. Accordingly, the aim of the present study was to determine the frequency and principal risk factors for the development of seizure syndrome in stroke and TBI, as well as to evaluate the efficacy and safety of anticonvulsant therapy in intensive care settings, to identify patterns of seizure development and the most promising treatment approaches for improving patient outcomes.

Materials and Methods

To conduct the literature review, 50 relevant peer-reviewed publications were selected addressing the incidence of seizure syndrome, its aetiological factors, and the effectiveness of anticonvulsant therapy in patients with stroke and TBI. The review also included an analysis of current international and national clinical guidelines [8-11] on the management of patients with stroke and acute TBI. The literature search was performed using major international scientometric databases – PubMed, Scopus, Web of Science, and Google Scholar – and applied English-language keywords, including "seizure syndrome", "stroke", "traumatic brain injury", "antiepileptic therapy", "intensive care", "epileptic complications", "anticonvulsant efficacy", and "seizure incidence in stroke and TBI". No strict time limits were imposed; however, priority was given to studies published between 2015 and 2025 to ensure topical relevance.

The selection of publications followed the PRISMA methodology [12] (Preferred Reporting Items for Systematic Reviews and Meta-Analyses), including the removal of duplicates, initial screening of titles and abstracts, and subsequent full-text evaluation. A PRISMA flow diagram was not included, which represents a methodological limitation of the review. Inclusion criteria comprised defined patient populations with stroke or TBI; data on the incidence of seizure syndrome; information on anticonvulsant therapy and its effectiveness; statistically analysed outcomes; and publication in peer-reviewed journals. Exclusion criteria included review articles, case reports, studies with insufficient sample sizes or inadequate statistical reporting, and publications unrelated to seizure syndrome in stroke or TBI. The quality of sources was evaluated based on study design (RCTs, cohort studies, and retrospective analyses), sample size, availability of control groups, clarity of statistical methodology, and adherence to ethical standards. Studies with low levels of evidence or significant methodological shortcomings were excluded. The temporal distribution of publications was also considered to ensure the chronological relevance and representativeness of the included material.

Results and Discussion

Incidence and predictors of seizure syndrome following stroke and TBI. In a review by S. Neri *et al.* [13], it was reported that early seizures develop in approximately 3.3%-3.9% of patients following an acute cerebrovascular event, with notably lower rates observed in lacunar stroke (0.9%). Post-stroke epilepsy occurs in approximately 7% of patients after stroke, accounting for 30%-49% of all new-onset seizures in individuals aged over 65 years. The authors emphasised that stroke severity and extent, cortical involvement, and haemorrhagic transformation are the principal predictors of seizure occurrence, with haemorrhagic stroke almost doubling the risk of post-stroke epilepsy. In addition, younger age at stroke onset was identified as a significant predictor of post-stroke epilepsy in this population. Similar conclusions were drawn by S. Ma *et al.* [14], who demonstrated in their meta-analysis of 18 case-control studies involving 13,289 patients that cortical involvement [OR 5.00, 95% CI (2.85-8.74)], cerebral infarction with haemorrhagic transformation [OR 2.77, 95% CI (1.87-4.11)], and intracerebral haemorrhage [OR = 1.83, 95% CI (1.13-2.97)] were significantly associated with early-onset seizures after stroke. In a Ukrainian cohort study, O. Kauk *et al.* [15] reported that inflammatory biomarkers – particularly elevated C-reactive protein (CRP) (>10 mg/L, peaking at 24-48 hours) and IL-6 (>20 pg/mL, peaking at 2-6 hours) – correlate with larger infarct size, blood-brain barrier disruption, and early neurological deterioration in intensive care patients with ischaemic stroke. These findings indirectly support a mechanistic link between post-stroke neuroinflammation and an increased risk of seizures. Y. Bondarenko *et al.* [16] further noted that both ischaemic and haemorrhagic strokes with cortical involvement frequently lead to neurological complications that adversely affect cognitive recovery in intensive care settings.

Across these studies, a consistent pattern emerges: seizures following stroke occur more frequently in haemorrhagic subtypes, cortical infarctions, and cases characterised by a pronounced inflammatory response. Early seizures (<7 days) repeatedly appear to be a marker of more severe brain injury and a predictor of poorer functional outcomes. The reviewed data collectively support the understanding that the development of seizures reflects both the extent of structural brain damage and the degree of neuroinflammatory activation in acute cerebrovascular disease.

M. Pease *et al.* [17] examined patients with severe TBI and found that post-traumatic epilepsy develops in approximately 25%-32% of cases, with higher rates among individuals exhibiting cortical contusions, intracranial haematomas, and severe impairment on the GCS at admission. Their findings indicated that seizures serve as a marker of diffuse and penetrating neuronal injury. A complementary analysis by H.F. Sødal *et al.* [18] demonstrated that early post-traumatic seizures occur in 5.6% of hospitalised TBI patients, with risk factors including alcohol abuse (OR 3.6, 95% CI 2.3-5.7), moderate and severe brain injury (OR 2.2, 95% CI 1.3-3.8 and OR 2.1, 95% CI 1.2-3.6, respectively), brain contusion (OR 1.6, 95% CI 1.0-2.4), and subdural haematoma (OR 1.6, 95% CI 1.0-2.6). In an extensive review, S. Fordington & M. Manford [19] described a sequence of acute pathological processes following traumatic injury, including necrosis, microhaemorrhage, axonal injury, apoptosis, demyelination, microgliosis, inflammation, and oxidative stress. Later phases of neurodegeneration, regeneration, revascularisation, and neural remodelling were shown to contribute to circuit alterations that ultimately result in post-traumatic epilepsy. R.E. Teneralli *et al.* [20] demonstrated that among 205,183 individuals with newly diagnosed epilepsy, those who developed drug-resistant epilepsy within one year (4.1%) already exhibited a substantially higher burden of physical and psychiatric comorbidities – such as pain syndromes, headache, neuropathy, musculoskeletal disorders, traumatic brain injury, depression, anxiety, bipolar disorder, suicidal ideation, substance dependence, and sleep disorders – even prior to the initial epilepsy diagnosis. These findings suggest that broader systemic vulnerability may predispose certain patients to more severe seizure phenotypes.

Post-traumatic seizures represent a dynamic continuum in which the extent of structural brain disruption interacts with an individual's physiological resilience to shape long-term neurological trajectories. Across studies, a consistent pattern emerges: epileptogenesis following traumatic brain injury is a multifactorial process driven by acute tissue damage, inflammatory activation, and subsequent network remodelling. Within this framework, clinical outcomes range from isolated early seizures to persistent or drug-resistant epilepsy, highlighting not only the severity of the initial injury but also the systemic vulnerability that predisposes some patients to more severe seizure phenotypes.

Early seizures in acute neurological injury. A comparative analysis of the reviewed literature demonstrated that seizure syndrome occurs more frequently after TBI than after stroke, although both conditions share overlapping risk factors such as cortical involvement and intracerebral haemorrhage. Early seizures consistently emerge as a negative prognostic indicator across both groups. Moreover, the reviewed studies collectively identified brain oedema, inflammatory activation, and metabolic instability as common mechanisms contributing to early seizure development. While stroke-specific literature emphasised the roles of haemorrhagic transformation and neuroinflammation, TBI-focused studies highlighted mechanical tissue disruption and haematoma-related cortical irritation. Despite methodological differences, the overall evidence converges on the conclusion that seizure incidence in intensive care settings reflects the severity of structural brain damage and systemic physiological stress following acute neurological injury.

The findings of this review underscore the multifactorial nature of seizure syndrome following acute brain injury, demonstrating that both structural and molecular factors contribute to seizure vulnerability. T. Okada *et al.* [21] highlighted that blood-brain barrier (BBB) disruption after stroke plays a central role in generating cortical hyperexcitability through inflammatory activation, ionic dysregulation, and impaired metabolic homeostasis. This concept aligns with evidence that early seizures often coincide with peak BBB permeability and neuroinflammatory cascades, a relationship further supported by clinical risk-stratification data in hypoxic-ischaemic brain injury described by D.S. Mankovskyi [22]. These mechanisms help explain the observed associations between haemorrhagic stroke, cortical involvement, and an increased incidence of seizures. Management strategies for acute seizures must also be informed by evidence-based clinical guidelines. F. Rosenow & J. Weber [23], in the S2k guideline for status epilepticus, underscore the necessity of prompt benzodiazepine administration, continuous electroencephalography (EEG) monitoring, and timely escalation to second-line antiseizure medications when required. Their recommendations emphasised that early recognition and intervention significantly reduce morbidity and mortality, particularly in neurocritical care settings. The importance of EEG monitoring is further supported by F. Misirocchi *et al.* [24], who demonstrated that ICU-based EEG services improve outcomes in patients with status epilepticus by enabling earlier detection of subclinical seizures. Pharmacological therapy remains central to seizure control in stroke and TBI populations. T. Hakami [25] reported that over the past three decades, approximately 20 newer-generation (second- and third-generation) antiseizure drugs (ASDs), characterised by diverse mechanisms of action and pharmacokinetic profiles, have been introduced into clinical practice. This development has expanded the therapeutic armamentarium of epilepsy and broadened the selection of ASDs to better match individual patient characteristics. J.A. French *et al.* [26] and A.M. Kanner *et al.* [27] observed that newer ASDs do not differ significantly in seizure control compared with older agents, but some demonstrate improved tolerability, particularly with fewer neurotoxic adverse effects. For focal-onset seizures, lamotrigine is considered the first-line drug of choice [25]. Other widely used first-line agents include levetiracetam and zonisamide; however, findings from the SANAD II trial reported by A. Marson *et al.* [28] suggested that these drugs are inferior to lamotrigine with respect to time to 12 month remission. Medication selection must therefore consider seizure type, patient characteristics, adverse-effect profile, potential drug-drug interactions, and cost. Despite the expansion of therapeutic options, drug-resistant epilepsy occurs in approximately 25%-30% of patients, and treatment with first-generation ASDs fails in 30%-40% of individuals due to intolerable adverse effects, as noted by T. Hakami [25]. A. Bayat *et al.* [29] found that genetic testing significantly influences treatment decisions in childhood-onset epilepsies, enabling optimised medication selection and avoidance of ineffective therapies. Similarly, B. Castellotti *et al.* [30] demonstrated that next-generation sequencing facilitates personalised therapeutic planning based on channelopathy-specific or metabolism-related gene mutations. These findings are reinforced by A. Balaji *et al.* [31], who highlighted the emerging role of genome-based therapeutics in drugresistant epilepsies. Non-pharmacological therapies also play an important role in the management of refractory seizures. J.H. Na *et al.* [32] reported high clinical efficacy and acceptable safety of the ketogenic diet in patients with genetically confirmed drug-resistant epilepsy, suggesting its potential application in selected adult and paediatric populations following acute brain injury. Advanced neurostimulation techniques offer additional therapeutic benefits: E. Sharma *et al.* [33] showed that responsive neurostimulation results in significant seizure reduction in patients with refractory mesial temporal lobe epilepsy, underscoring the potential applicability of neuromodulation strategies in carefully selected survivors of TBI or stroke.

Taken together, the reviewed findings indicate that seizure syndrome following acute brain injury arises from a dynamic interplay of structural lesions, BBB dysfunction, excitatory-inhibitory imbalance, and systemic metabolic disturbances. Early seizures consistently function as a marker of severe neurological damage and are associated with poorer functional outcomes. Evidence clearly demonstrates that prompt EEG-based diagnosis, appropriate selection of antiseizure medication, and individualised treatment strategies improve prognosis, reduce complications, and shorten the duration of ICU stays. Overall, the integrated data confirm that optimising seizure management requires a multimodal approach, combining rapid pharmacological intervention, continuous monitoring, and precision medicine tools, such as genetic testing.

Main causes of seizure syndrome. Seizure syndrome is a complex manifestation of neuronal hyperexcitability, characterised by sudden, paroxysmal disturbances in motor, sensory, or cognitive function. S. Wu & D.R. Nordli [34] described seizure semiology as the clinical manifestation of a seizure generated by activation of the symptomatogenic zone. Semiology represents a major component of epilepsy evaluation, providing important information for seizure classification and assisting in seizure localisation. The site of seizure onset and the network of seizure propagation contribute to semiology, together with age, underlying pathophysiology, and the rate of electrical discharge propagation, all of which influence the clinical presentation of seizures. Importantly, seizure syndrome is not a discrete disease entity but rather reflects impaired neuronal regulation caused by organic, metabolic, infectious, or toxic factors. R.E. Stirling *et al.* [35] noted that epilepsy is characterised by unpredictable, recurrent seizures that vary widely between individuals. Advances in seizure forecasting, including machine-learning approaches and the investigation of non-EEG biomarkers, have substantially improved the understanding of seizure patterns. Most individuals exhibit circadian rhythms of seizure occurrence, with many also experiencing longer multiday cycles. M.Y. Xu [36] highlighted that ischaemic and haemorrhagic strokes induce neuronal injury, excitotoxicity, and inflammatory activation, thereby creating epileptogenic zones, particularly within cortical regions. R. Brondani *et al.* [37] further demonstrated that large-territory strokes, especially malignant middle cerebral artery infarctions requiring decompressive hemicraniectomy, markedly increase seizure risk as a result of extensive cortical disruption. In their retrospective cohort of 36 patients with a mean follow-up of 1,086 days, seizures occurred in 22 patients (61.1%, 95% CI 45.17%-77.03%), with 13 patients (36.1%) developing seizures within the first week after stroke. Among the 34 patients who survived the acute phase, 19 (55.9%, 95% CI 39.21%-72.59%) developed post-stroke epilepsy. Notably, no specific risk factors – including age, sex, stroke laterality, vascular risk profile, haemorrhagic transformation, or timing of craniectomy – distinguished patients who developed seizures from those who remained seizure-free, suggesting that stroke volume and the extent of cortical ischaemia are the primary determinants of epileptogenesis in this population. The authors emphasised that seizures may occur particularly early in patients not receiving anticonvulsant prophylaxis. MRI-based investigations, such as the systematic review by F.P. Mariajoseph *et al.* [38], demonstrated seizure-associated cortical abnormalities, supporting the concept that structural and metabolic stressors exacerbate cortical instability after stroke. TBI constitutes another major aetiological category. According to R.E. Stirling *et al.* [35], recent advances in seizure forecasting have shown substantial progress, including improvements in predictive algorithms using machine learning and exploration of non-EEG measures of seizure susceptibility, such as physiological biomarkers, behavioural changes, environmental factors, and cyclical seizure patterns. Investigations into periodicities in individual seizure activity have demonstrated that over 90% of individuals exhibit circadian seizure rhythms, with many also experiencing multiday, weekly, or longer cycles. Potential indicators of seizure susceptibility include stress levels, heart rate, and sleep quality, all of which can be captured non-invasively over extended periods. Applications of seizure-forecasting technologies include improving quality of life, guiding treatment planning and medication titration, optimising presurgical monitoring, and directing future scientific research. A. Szűcs *et al.* [39] observed that reflex mechanisms may also contribute to seizure development in certain TBI patients, particularly when cortical circuits become hypersensitised after injury. These findings help explain why prolonged impaired consciousness and mass-effect lesions substantially increase seizure susceptibility. Metabolic disturbances constitute an additional major group of seizure triggers. Hyponatraemia, hypocalcaemia, and disturbances in glucose homeostasis alter neuronal membrane stability and intracellular signalling, thereby facilitating uncontrolled depolarisation. S. Wu & D.R. Nordli [34] described motor semiology as a major component of epilepsy evaluation, providing essential information for seizure classification and localisation. Typical motor seizures include tonic, clonic, tonic-clonic, myoclonic, atonic seizures, epileptic spasms, automatisms, and hyperkinetic seizures. Beyond "positive" motor signs, negative motor phenomena, such as atonic seizures and Todd's paralysis, are crucial for seizure analysis. Several motor signs – including version, unilateral dystonia, and asymmetric clonic termination – have significant clinical value in seizure lateralisation. The study reviewed the localisation value and underlying pathophysiology using updated evidence from intracranial electroencephalographic recordings, particularly stereoelectroencephalography. Overall, seizure syndrome arises from the convergence of primary structural damage, secondary metabolic instability, and inflammatory alterations within the neuronal microenvironment. Across the reviewed studies, a consistent theme is that cortical involvement – whether resulting from stroke, TBI, infection, or metabolic crisis – plays a central role in seizure generation. These findings underscore the importance of early diagnostic evaluation, including neuroimaging and electrophysiological monitoring, to guide timely therapeutic intervention and prevent progression to chronic epilepsy. Figure 1 illustrated a fundamental transition, whereby acute brain injury initiates a convergent pathophysiological process in which structural disruption, neuroinflammation, metabolic failure, and an imbalance between excitatory and inhibitory signalling collectively drive the brain towards network hyperexcitability.

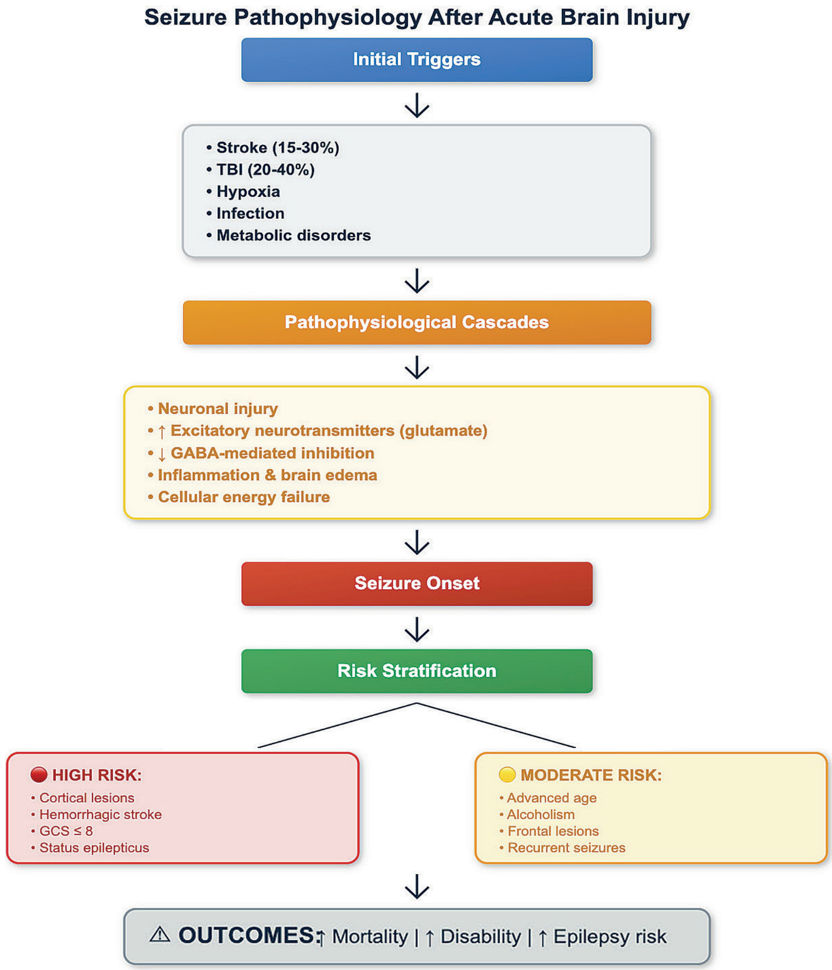

Figure 1. Post-acute brain injury seizure pathophysiology flowchart
Source: prepared by the authors during the course of the research

Although stroke and traumatic brain injury share several mechanistic determinants, their seizure profiles diverge according to the depth and spatial distribution of cortical involvement. Early seizures consistently signal profound circuit destabilisation and strongly predict adverse neurological and survival outcomes. Cortical lesions and intracranial haemorrhage remain the most robust predictors of seizure emergence across cohorts. These insights highlight the need for precise early detection, risk-based stratification, and timely targeted intervention to prevent progression towards chronic epileptogenesis and long-term disability.

Therapeutic approaches to seizure management. Anticonvulsant therapy is central to the management of seizure syndrome, as it controls abnormal cortical electrical activity, as noted by E. Perucca *et al.* [40] and G.J. Sills & M.A. Rogawski [41]. According to these studies, seizures arise from an imbalance between excitatory and inhibitory systems, whereby excessive glutamatergic activity, combined with insufficient inhibition mediated by γ-aminobutyric acid, leads to neuronal hyperexcitability. In addition, anticonvulsant drugs reduce neuronal excitability and stabilise synaptic transmission by acting on multiple mechanisms within the pathophysiological cascade of epileptogenesis.

Levetiracetam is a widely used modern anticonvulsant that acts by binding to the synaptic vesicle protein SV2A, thereby reducing glutamatergic neurotransmission while producing minimal sedative effects. I.J. Contreras-García *et al.* [42] and B.A. Lynch *et al.* [43] reported that levetiracetam's mechanism involves high-affinity binding to SV2A, with rapid penetration of the blood-brain barrier. For acute seizures and status epilepticus, E. Trinka *et al.* [44] and T. Glauser *et al.* [45] identified lorazepam as the first-line treatment, owing to its rapid and sustained enhancement of GABA-A-mediated inhibition. Intranasal midazolam provides a rapid and practical alternative when IV access is unavailable. R. Kienitz *et al.* [46] reported that in 20%-30% of drugresistant cases, neurosurgical approaches – such as vagus nerve stimulation, deep brain stimulation, and resective surgery – represent viable therapeutic options. T. Xue *et al.* [47] and F.V. Gouveia *et al.* [48] corroborated these findings, emphasising their relevance for carefully selected patients. G.W. Culler & B.C. Jobst [49] further underscored the clinical applicability of these interventions. A. Jiang *et al.* [50] additionally noted that ketogenic therapy may be considered as an alternative metabolic strategy. Pharmacogenetics enables treatment individualisation based on mutations in ion-channel and synaptic protein genes, and genetic targeting is increasingly recognised as an essential component of precision epileptology. K. Borowicz-Reutt *et al.* [51] and A.A. Shaimardanova *et al.* [52] highlighted the importance of pharmacogenetics in tailoring treatment strategies based on mutations in SCN1A, KCNQ2, PCDH19, and other ion-channel and synaptic protein genes. Their conclusions emphasised the role of gene-targeted diagnostics in modern precision epileptology. These molecular data may predict drug responsiveness, guide the avoidance of ineffective or potentially harmful medications, and help identify candidates for emerging genedirected therapies. Therapy should be individualised according to seizure type, age, aetiology, comorbidities, and EEG findings. Equally important is the role of systematic monitoring of treatment effectiveness and safety. Patient management includes regular clinical assessment of seizure frequency and characteristics, monitoring of laboratory parameters, and EEG surveillance, including video-EEG for the detection of subclinical seizures. Where necessary, dose titration or switching to alternative anticonvulsants is undertaken, taking into account pharmacokinetic and pharmacodynamic parameters, as summarised in Table 1.

Table 1. Therapeutic approach to seizure syndrome

Aspect	Medication/method	Mechanism/use	Notes
Core therapy	Anticonvulsants	Suppress cortical hyperexcitability; balance excitatory (glutamate) and inhibitory (GABA) signalling	Foundation of treatment
Primary drug	Levetiracetam	Binds SV2A protein; reduces glutamate release	Minimal adverse effects; rapid CNS penetration
Acute seizures	Lorazepam (IV)	Enhances GABA-A-mediated inhibition via Cl^- channel potentiation	Onset: 2-3 min; duration up to 12 h
Emergency (field use)	Midazolam (intranasal)	Rapid absorption via the nasal mucosa	Suitable for paediatric use and when IV access is unavailable
Refractory epilepsy	Vagus nerve stimulation, brain surgery, ketogenic diet	Modulates neuronal excitability or cerebral metabolism	Used in 20%-30% of drugresistant cases
Genetic targeting	Gene-based diagnostics	Analysis of SCN1A, KCNQ2, PCDH19 mutations	Enables personalised medicine
Drug selection factors	–	Seizure type, age, aetiology, EEG findings	Tailored therapeutic strategy
Monitoring	EEG, clinical assessment	Detection of subclinical seizures; monitoring of drug effects	Therapy adjusted according to response and safety

Source: prepared by the authors during the course of the research

Effective seizure management following acute brain injury requires an integrated, multimodal strategy capable of providing both rapid stabilisation and sustained modulation of neuronal network activity. Immediate control with lorazepam or intranasal midazolam forms the foundation of emergency management, while levetiracetam supports longer-term synaptic stability through targeted SV2A interaction. In pharmacoresistant cases, deeper modulation of neuronal and metabolic dysregulation may be achieved through vagus nerve stimulation, surgical resection of epileptogenic zones, or ketogenic metabolic therapy, with genetic approaches offering additional precision by accounting for individual ion-channel and synaptic abnormalities. Taken together, the combination of pharmacological and non-pharmacological interventions facilitates restoration of network stability, reduction of seizure recurrence, and mitigation of long-term neurological complications.

Recommendations for optimising the management of seizure syndromes. Optimisation strategies are based on current international clinical guidelines issued by the ILAE, AAN, and NICE [27, 53, 54] and are aimed at ensuring maximal therapeutic efficacy and patient safety. A key determinant of successful management is the early detection of seizure syndromes, achieved through comprehensive clinical monitoring, the use of standardised consciousness assessment scales (e.g. the Glasgow Coma Scale), neuroimaging modalities (CT or MRI), and EEG. EEG represents an essential diagnostic modality, enabling the identification of subclinical seizures, focal epileptiform discharges, and non-convulsive status epilepticus – conditions that frequently occur in the absence of overt motor manifestations – thereby rendering the timely initiation of targeted antiseizure therapy critically important.

Individualised treatment constitutes a core principle of modern antiseizure management. The selection of antiepileptic drugs should be guided by seizure aetiology, age-related patient characteristics, comorbid conditions, potential drug-drug interactions, the presence of hepatic or renal impairment, and – when available – the results of pharmacogenetic testing. For example, in seizures associated with structural brain pathology (including ischaemic stroke, traumatic brain injury, tumours, or congenital malformations), the use of agents with favourable blood-brain barrier penetration and central nervous system bioavailability, such as levetiracetam, lamotrigine, or topiramate, is considered appropriate In cases of acute symptomatic seizures (e.g. hypoglycaemia, hyponatraemia, or encephalitis), the prompt administration of benzodiazepines (lorazepam or midazolam), either intravenously or intranasally, is recommended to achieve rapid seizure termination. In pharmacoresistant epilepsy or refractory status epilepticus, alternative therapeutic approaches are warranted. These include continuous infusions of propofol or thiopental under strict monitoring of vital parameters, implementation of a ketogenic diet (particularly within paediatric practice), neurosurgical interventions (such as focal cortical resection), or the implantation of vagus nerve stimulation devices. The field of personalised medicine has undergone substantial development, with increasing attention to genetic polymorphisms (e.g. SCN1A, KCNQ2, GABRA1), which may assist in predicting drug responsiveness and reducing the risk of adverse drug reactions. Systematic monitoring of antiseizure therapy effectiveness is a critical component of high-quality patient care. This process encompasses not only clinical evaluation of seizure frequency, duration, and semiology, but also assessment of treatment tolerability, psycho-emotional status, and cognitive function. The use of video-EEG monitoring facilitates the detection of both clinically apparent and subclinical epileptic activity, enables objective evaluation of therapeutic response, and assists in identifying potential adverse effects, including behavioural changes, sedation, cognitive impairment, or hepatotoxicity. Dose adjustments should be informed by pharmacokinetic parameters – half-life, bioavailability, plasma protein binding, and individual metabolic characteristics. In patients with hepatic or renal dysfunction, therapeutic drug monitoring in serum is recommended to prevent drug accumulation and toxicity. Overall, effective seizure management following acute brain injury relies on an integrated, individualised strategy combining early diagnosis, continuous EEG surveillance, and aetiology-driven selection of antiseizure therapies (Table 2).

Table 2. Recommendations for optimising seizure management

Area	Key recommendation	Details/examples
Early detection and diagnosis	Ensure prompt seizure identification	Clinical observation, Glasgow Coma Scale assessment, CT/MRI, and EEG for detection of subclinical seizures and non-convulsive status epilepticus.
EEG monitoring	Central diagnostic modality	Identification of epileptiform discharges and subclinical activity; supports early initiation of targeted therapy.
Individualised therapy	Tailor treatment to patient-specific factors	Consider seizure aetiology, age, comorbidities, drug interactions, hepatic/renal function, and pharmacogenetic data.
Drug selection by aetiology	Align drug properties with the seizure cause	Structural lesions: lipophilic agents such as levetiracetam, lamotrigine or topiramate.
Acute symptomatic seizures	Use rapid-acting agents in emergency situations	In hypoglycaemia, hyponatraemia, or encephalitis: administer intravenous or intranasal benzodiazepines (lorazepam, midazolam).
Refractory epilepsy/ status epilepticus	Escalate to advanced therapeutic options	Use propofol or thiopental under intensive monitoring; consider a ketogenic diet, surgical resection, or vagus nerve stimulation.
Personalised medicine	Integrate genetic testing into clinical decision-making	Genes such as SCN1A, KCNQ2, and GABRA1 influence drug efficacy and the risk of adverse effects.
Therapeutic monitoring	Ensure continuous assessment of treatment effectiveness	Monitor seizure frequency and duration, treatment tolerability, cognitive function, and psychological status.
Video-EEG monitoring	Gold standard for longitudinal assessment	Captures both clinical and subclinical seizures and facilitates evaluation of drug effectiveness and adverse effects (e.g. sedation, behavioural changes).
Dose adjustment	Adjust dosing based on pharmacokinetic principles	Consider half-life, bioavailability, plasma protein binding, hepatic and renal function, and perform therapeutic drug monitoring when indicated.

Source: prepared by the authors during the course of the research

Current approaches focus on individualising therapeutic interventions, taking into account the patient's metabolic, pharmacokinetic, and genetic characteristics, with advanced strategies such as ketogenic metabolic modulation, targeted neurosurgical interventions, and vagus nerve neurostimulation playing a key role in the management of refractory cases. Continuous evaluation of network biomarkers, treatment response, and adaptive dose adjustment enhances both safety and therapeutic precision. Collectively, these elements constitute a next-generation, evidence-based framework aimed not merely at seizure suppression, but at modulating pathological network dynamics to improve long-term neurological outcomes and minimise chronic complications.

Conclusions

This comprehensive review of 54 scientific sources demonstrated that seizure syndrome represents a frequent and clinically significant complication of acute brain injury, occurring in 15%-30% of stroke patients and 20%-40% of individuals with traumatic brain injury. The findings consistently indicated that early seizures, particularly those occurring within the first seven days, serve as strong predictors of unfavourable clinical outcomes, including increased mortality, greater functional disability, and prolonged intensive care unit stays. Across the reviewed literature, cortical involvement, intracerebral haemorrhage, and severe neurological impairment (e.g. low Glasgow Coma Scale scores) repeatedly emerged as the most prominent risk factors. Neuroinflammation, excitatory-inhibitory neurotransmitter imbalance, cellular energy failure, and cerebral oedema constitute the core pathophysiological mechanisms underlying seizure development in both stroke and TBI populations. The analysis further confirmed that levetiracetam and lorazepam represent the most effective and safest antiseizure medications in acute care settings, owing to their favourable pharmacokinetic profiles and low systemic toxicity. Evidence also highlighted the critical importance of continuous EEG monitoring for detecting subclinical seizure activity, guiding therapeutic adjustments, and preventing secondary neurological deterioration.

Optimisation of treatment was shown to depend strongly on personalised management strategies that take into account comorbidities, drug-drug interactions, renal and hepatic function, and the aetiological subtype of brain injury. Moreover, non-pharmacological interventions, including neurostimulation, ketogenic dietary therapy, and surgical approaches, play an important role in the management of refractory seizure syndromes, although their application remains limited to carefully selected patient populations. A key conclusion of this review is that early identification and risk stratification of vulnerable patients significantly improve clinical outcomes by enabling the timely initiation of targeted therapy. The findings further underscore the need for unified clinical protocols in intensive care settings to standardise management, enhance diagnostic accuracy, and reduce variability in treatment outcomes across healthcare systems. Future research should prioritise the advancement of precision-medicine approaches and the development of reliable biomarkers for seizure prediction and therapeutic response assessment. In addition, long-term prospective studies are required to elucidate the cognitive and functional consequences of seizure syndrome following stroke and traumatic brain injury.

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Conflict of Interest

No financial or personal relationships are declared that could potentially influence the objectivity or integrity of this research.

References

[1] Galovic M, Ferreira-Atuesta C, Abraira L, Döhler N, Sinka L, Brigo F, et al. Seizures and epilepsy after stroke: Epidemiology, biomarkers and management. Drugs Aging. 2021;38(4):285–99. DOI: 10.1007/s40266-021-00837-7

[2] Ferreira-Atuesta C, Döhler N, Erdélyi-Canavese B, Felbecker A, Siebel P, Scherrer N, et al. Seizures after ischemic stroke: A matched multicenter study. Ann Neurol. 2021;90(5):808–20. DOI: 10.1002/ana.26212

[3] Nandan A, Zhou YM, Demoe L, Waheed A, Jain P, Widjaja E. Incidence and risk factors of post-stroke seizures and epilepsy: Systematic review and meta-analysis. J Int Med Res. 2023;51(11):3000605231213231. DOI: 10.1177/03000605231213231

[4] Misra S, Kasner SE, Dawson J, Tanaka T, Zhao Y, Zaveri HP, et al. Outcomes in patients with poststroke seizures: A systematic review and meta-analysis. JAMA Neurol. 2023;80(11):1155–65. DOI: 10.1001/jamaneurol.2023.3240

[5] Wang Z, Xu H, Liu J, Lin R, He D, Yang Y, et al. An automatic deep-learning approach for the prediction of post-stroke epilepsy after an initial intracerebral hemorrhage based on non-contrast computed tomography imaging. Quant Imaging Med Surg. 2025;15(2):1175–89. DOI: 10.21037/qims-24-1345

[6] Scarpino M, Grippo A, Campagnini S, Hakiki B, Maiorelli A, Sodero A, et al. Stroke-related epilepsy in the rehabilitation setting: Insights from the inpatient post-stroke rehabilitation study – RIPS. Epilepsy Behav Rep. 2024;28:100713.DOI: 10.1016/j.ebr.2024.100713

[7] Peter-Derex L, Philippeau F, Garnier P, André-Obadia N, Boulogne S, Catenoix H, et al. Safety and efficacy of prophylactic levetiracetam for prevention of epileptic seizures in the acute phase of intracerebral haemorrhage (PEACH): A randomised, double-blind, placebo-controlled, phase 3 trial. Lancet Neurol. 2022;21(9):781–91. DOI: 10.1016/S1474-4422(22)00235-6

[8] Oder of Ministry of Health of Ukraine No. 276. On the Approval and Implementation of Medical and Technological Documents for the Standardization of Medical Care for Epilepsy [Internet]. 2014 April 17 [cited 2025 April 17]. Available from: https://zakon.rada.gov.ua/rada/show/v0276282-14#Text

[9] Frontera JA, Gilmore EJ, Johnson EL, Olson D, Rayi A, Tesoro E, et al. Guidelines for seizure prophylaxis in adults hospitalized with moderate-severe traumatic brain injury: A clinical practice guideline for health care professionals from the Neurocritical Care Society. Neurocrit Care. 2024;40:819–44. DOI: 10.1007/s12028-023-01907-x

[10] Frontera JA, Rayi A, Tesoro E, Gilmore EJ, Johnson EL, Olson D, et al. Guidelines for seizure prophylaxis in patients hospitalized with nontraumatic intracerebral hemorrhage: A clinical practice guideline for health care professionals from the Neurocritical Care Society. Neurocrit Care. 2025;42:1–21. DOI: 10.1007/s12028-024-02183-z

[11] Kälviäinen R. Guideline 00766: Treatment of prolonged seizure episodes and status epilepticus. Kyiv: Ministry of Health of Ukraine; 2017. 5 P.

[12] Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. BMJ. 2021;372:71. DOI: 10.1136/bmj.n71

[13] Neri S, Gasparini S, Pascarella A, Santangelo D, Cianci V, Mammì A, et al. Epilepsy in cerebrovascular diseases: A narrative review. Curr Neuropharmacol. 2023;21(8):1634–45. DOI: 10.2174/1570159X20666220706113925

[14] Ma S, Fan X, Zhao X, Wang K, Wang H, Yang Y. Risk factors for early-onset seizures after stroke: A systematic review and meta-analysis of 18 observational studies. Brain Behav. 2021;11(6):e02142. DOI: 10.1002/brb3.2142

[15] Kauk O, Bondarenko Y, Kulyk D. Prognostic value of inflammatory biomarkers (CRP, IL-6, procalcitonin) in patients with ischemic stroke in intensive care. Psychiatry Neurol Med Psychol. 2025;13(29):344–56. DOI: 10.26565/2312-5675-2025-29-05

[16] Bondarenko Y, Kulyk D, Kauk O. Cognitive impairment after the hemorrhagic and ischemic stroke: Is it possible to minimize its development already in intensive care? Grail Sci. 2025;51:862–76. DOI: 10.36074/grail-of-science.18.04.2025.116

[17] Pease M, Gonzalez-Martinez J, Puccio A, Nwachuku E, Castellano JF, Okonkwo DO, et al. Risk factors and incidence of epilepsy after severe traumatic brain injury. Ann Neurol. 2022;92(4):663–9. DOI: 10.1002/ana.26443

[18] Sødal HF, Storvig G, Tverdal C, Robinson HS, Helseth E, Taubøll E. Early post-traumatic seizures in hospitalized patients with traumatic brain injury. Acta Neurol Scand. 2022;146(5):485–91. DOI: 10.1111/ane.13670

[19] Fordington S, Manford M. A review of seizures and epilepsy following traumatic brain injury. J Neurol. 2020;267(10):3105–11. DOI: 10.1007/s00415-020-09926-w

[20] Teneralli RE, Cepeda MS, Kern DM, Novak GP. Individuals who develop drug-resistant epilepsy within a year after initial diagnosis have higher burden of mental and physical diseases one-year prior to epilepsy diagnosis as compared to those whose seizures were controlled during the same interval. Epilepsy Behav. 2021;123:108243. DOI: 10.1016/j.yebeh.2021.108243

[21] Okada T, Suzuki H, Travis ZD, Zhang JH. The stroke-induced blood-brain barrier disruption: Current progress of inspection technique, mechanism, and therapeutic target. Curr Neuropharmacol. 2020;18(12):1187–212. DOI: 10.2174/1570159X18666200528143301

[22] Mankovskyi DS. Clinical and anamnestic predictors of postoperative hypoxic and ischemic brain lesions and algorithms for their assessment of neurological support of cardiac surgery patients. Bull Med Biol Res. 2021;3(3):42–8. DOI: 10.11603/bmbr.2706-6290.2021.3.12568

[23] Rosenow F, Weber J. S2k guidelines: Status epilepticus in adulthood: Guidelines of the German Society for Neurolog. Nervenarzt. 2021;92(10):1002–30. DOI: 10.1007/s00115-020-01036-2

[24] Misirocchi F, Quintard H, Kleinschmidt A, Schaller K, Pugin J, Seeck M, et al. ICU-electroencephalogram unit improves outcome in status epilepticus patients: A retrospective before-after study. Crit Care Med. 2024;52(11):545–56. DOI: 10.1097/CCM.0000000000006393

[25] Hakami T. Efficacy and tolerability of antiseizure drugs. Ther Adv Neurol Disord. 2021;14:17562864211037430. DOI: 10.1177/17562864211037430

[26] French JA, Kanner AM, Bautista J, Abou-Khalil B, Browne T, Harden CL, et al. Efficacy and tolerability of the new antiepileptic drugs I: Treatment of new onset epilepsy. Neurology. 2004;62(8):1252–60. DOI: 10.1212/01.wnl.0000123693.82339.fc

[27] Kanner AM, Ashman E, Gloss D, Harden C, Bourgeois B, Bautista JF, et al. Practice guideline update summary: Efficacy and tolerability of the new antiepileptic drugs I: Treatment of new-onset epilepsy. Neurology. 2018;91(2):74–81. DOI: 10.1212/WNL.0000000000005755

[28] Marson A, Burnside G, Appleton R, Smith D, Leach JP, Sills G, et al. The SANAD II study of the effectiveness and cost-effectiveness of levetiracetam, zonisamide, or lamotrigine for newly diagnosed focal epilepsy: An open-label, non-inferiority, multicentre, phase 4, randomised controlled trial. Lancet. 2021;397(10282):1363–74. DOI: 10.1016/S0140-6736(21)00247-6

[29] Bayat A, Fenger CD, Techlo TR, Højte AF, Nørgaard I, Hansen TF, et al. Impact of genetic testing on therapeutic decision-making in childhood-onset epilepsies – a study in a tertiary epilepsy center. Neurotherapeutics. 2022;19(4):1353–67. DOI: 10.1007/s13311-022-01264-1

[30] Castellotti B, Ragona F, Freri E, Messina G, Magri S, Previtali R, et al. Next-generation sequencing in pediatric-onset epilepsies: Analysis with target panels and personalized therapeutic approach. Epilepsia Open. 2024;9(5):1922–30. DOI: 10.1002/epi4.13039
[31] Balaji A, Mohanlal S, Pachat D, Babu SS, Kumar ES, Mamukoya N, et al. Genome-based therapeutics: Era of precision medicine in genetic epilepsies and epileptic encephalopathies. Ann Indian Acad Neurol. 2023;26(5):723–7. DOI: 10.4103/aian.aian_314_23
[32] Na JH, Lee H, Lee YM. Clinical efficacy and safety of the ketogenic diet in patients with genetic confirmation of drug-resistant epilepsy. Nutrients. 2025;17(6):979. DOI: 10.3390/nu17060979
[33] Sharma E, Pomianoski BW, Jabbar R, Ayesha A, Silva YP, Łajczak P, et al. Responsive neurostimulation for patients with refractory mesial temporal lobe epilepsy: A systematic review and meta-analysis. Epilepsy Behav Rep. 2025;30:100774. DOI: 10.1016/j.ebr.2025.100774
[34] Wu S, Nordli DR. Motor seizure semiology. Handb Clin Neurol. 2023;196:295–304. DOI: 10.1016/B978-0-323-98817-9.00014-4
[35] Stirling RE, Cook MJ, Grayden DB, Karoly PJ. Seizure forecasting and cyclic control of seizures. Epilepsia. 2020;62(1):2–14. DOI: 10.1111/epi.16541
[36] Xu MY. Poststroke seizure: Optimising its management. Stroke Vasc Neurol. 2019;4(1):48–56. DOI: 10.1136/svn-2018-000175
[37] Brondani R, Garcia de Almeida A, Abrahim Cherubini P, Mandelli Mota S, de Alencastro LC, Antunes ACM, et al. High risk of seizures and epilepsy after decompressive hemicraniectomy for malignant middle cerebral artery stroke. Cerebrovasc Dis Extra. 2017;7(1):51–61. DOI: 10.1159/000458730
[38] Mariajoseph FP, Muthusamy S, Amukotuwa S, Seneviratne U. Seizure-induced reversible MRI abnormalities in patients with single seizures: A systematic review. Epileptic Disord. 2023;23(4):552–62. DOI: 10.1684/epd.2021.1300
[39] Szűcs A, Rosdy B, Kelemen A, Horváth A, Halász P. Reflex seizure triggering: Learning about seizure producing systems. Seizure. 2019;69:25–30. DOI: 10.1016/j.seizure.2019.03.019
[40] Perucca E, Bialer M, White HS. New GABA-targeting therapies for the treatment of seizures and epilepsy: I. Role of GABA as a modulator of seizure activity and recently approved medications acting on the GABA system. CNS Drugs. 2023;37(9):755–79. DOI: 10.1007/s40263-023-01027-2
[41] Sills GJ, Rogawski MA. Mechanisms of action of currently used antiseizure drugs. Neuropharmacology. 2020;168:107966. DOI: 10.1016/j.neuropharm.2020.107966
[42] Contreras-García IJ, Cárdenas-Rodríguez N, Romo-Mancillas A, Bandala C, Zamudio SR, Gómez-Manzo S, et al. Levetiracetam mechanisms of action: From molecules to systems. Pharmaceuticals. 2022;15(4):475. DOI: 10.3390/ph15040475
[43] Lynch BA, Lambeng N, Nocka K, Kensel-Hammes P, Bajjalieh SM, Matagne A, et al. The synaptic vesicle protein SV2A is the binding site for the antiepileptic drug levetiracetam. Proc Natl Acad Sci USA. 2004;101(26):9861–6. DOI: 10.1073/pnas.0308208101
[44] Trinka E, Höfler J, Leitinger M, Brigo F. Pharmacotherapy for status epilepticus. Drugs. 2015;75:1499–521. DOI: 10.1007/s40265-015-0454-2
[45] Glauser T, Shinnar S, Gloss D, Alldredge B, Arya R, Bainbridge J, et al. Evidence-based guideline: Treatment of convulsive status epilepticus in children and adults: Report of the guideline committee of the American Epilepsy Society. Epilepsy Curr. 2016;16(1):48–61. DOI: 10.5698/1535-7597-16.1.48
[46] Kienitz R, Kay L, Beuchat I, Gelhard S, von Brauchitsch S, Mann C, et al. Benzodiazepines in the management of seizures and status epilepticus: A review of routes of delivery, pharmacokinetics, efficacy, and tolerability. CNS Drugs. 2022;36(9):951–75. DOI: 10.1007/s40263-022-00940-2
[47] Xue T, Chen S, Bai Y, Han C, Yang A, Zhang J. Neuromodulation in drug-resistant epilepsy: A review of current knowledge. Acta Neurol Scand. 2022;146(6):786–97. DOI: 10.1111/ane.13696
[48] Gouveia FV, Warsi NM, Suresh H, Matin R, Ibrahim GM. Neurostimulation treatments for epilepsy: Deep brain stimulation, responsive neurostimulation and vagus nerve stimulation. Neurotherapeutics. 2024;21(3):e00308. DOI: 10.1016/j.neurot.2023.e00308
[49] Culler GW, Jobst BC. Surgical treatments for epilepsy. Continuum. 2022;28(2):536–58. DOI: 10.1212/CON.0000000000001106
[50] Jiang A, Liu W, Liu Y, Zhang J. Efficacy of ketogenic diet therapy for pediatric drug-resistant epilepsy with monogenic etiology: A single-arm meta-analysis. Nutr Rev. 2025;83(11):2104–22. DOI: 10.1093/nutrit/nuaf140
[51] Borowicz-Reutt K, Czernia J, Krawczyk M. Genetic background of epilepsy and antiepileptic treatments. Int J Mol Sci. 2023;24(22):16280. DOI: 10.3390/ijms242216280
[52] Shaimardanova AA, Chulpanova DS, Mullagulova AI, Afawi Z, Gamirova RG, Solovyeva VV, et al. Gene and cell therapy for epilepsy: A mini review. Front Mol Neurosci. 2022;15:868531. DOI: 10.3389/fnmol.2022.868531

[53] International League Against Epilepsy. ILAE clinical practice guidelines [Internet]. [cited 2025 April 17]. Available from: https://www.ilae.org/guidelines
[54] NICE guideline. Epilepsies in children, young people and adults [Internet]. 2022 April 27 [cited 2025 April 17]. Available from: https://www.nice.org.uk/guidance/ng217

Судомний синдром при інсульті та черепно-мозковій травмі: частота і результати лікування у відділенні інтенсивної терапії

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Анотація. Судомний синдром є однією з найпоширеніших і найскладніших неврологічних ускладнень у пацієнтів з гострими ушкодженнями головного мозку, зокрема після інсульту та черепно-мозкової травми. Це ускладнення суттєво погіршує клінічні результати, підвищує ризик смертності та подовжує перебування пацієнтів у відділенні інтенсивної терапії. Метою дослідження було визначити частоту судомного синдрому у пацієнтів з інсультом і черепно-мозковою травмою, проаналізувати основні причини його розвитку та оцінити ефективність і безпечність протисудомної терапії для вдосконалення підходів до лікування й поліпшення клінічних результатів. Проведено комплексний огляд літератури з використанням 50 релевантних наукових джерел із баз даних PubMed, Scopus, Web of Science та Google Scholar. Відбір здійснювався згідно з принципами PRISMA і включав дослідження з чітко визначеними популяціями пацієнтів з інсультом або черепно-мозковою травмою, даними щодо частоти судом, використанням протисудомних препаратів та статистично опрацьованими результатами, опублікованими в рецензованих виданнях. Опубліковані дослідження свідчили, що судомний синдром виникає приблизно у 15-30 % пацієнтів після інсульту та у 20-40 % осіб із черепно-мозковою травмою. Ранні судоми (протягом перших 7 днів) стабільно реєструються приблизно у 7 % пацієнтів після інсульту і асоціюються з гіршим прогнозом. Огляд літератури описав мультифакторні патофізіологічні механізми, що включають первинне структурне ушкодження мозкової тканини, вторинні метаболічні порушення, нейрозапалення та дисбаланс нейромедіаторних систем. Дані численних досліджень демонстрували високу ефективність сучасної протисудомної терапії – зокрема леветирацетаму та лоразепаму – на рівні 70-85 % із сприятливим профілем безпечності. Зв'язування леветирацетаму з білком синаптичних везикул 2A часто називають ключовим механізмом контролю судом, тоді як лоразепам залишається препаратом першої лінії для невідкладного купірування судом завдяки посиленню ГАМК-ергічної нейротрансмісії

Ключові слова: протисудомна терапія; леветирацетам; лоразепам; неврологічні ускладнення; запалення