MULARCZYK, Klaudia, ZNAMIROWSKA, Patrycja, PASZKOWSKA, Monika, KARABAN, Łukasz, KUPIS, Magdalena, JAKUBIAK, Anna, MIŚKIEWICZ, Joanna, BORAWSKI, Michał, CIUŁA, Aleksandra and KUŹNIECÓW, Tadeusz. Risk factors of status epilepticus in children - A literature review. Quality in Sport. 2025;41:60314. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.41.60314
https://apcz.umk.pl/QS/article/view/60314

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 14.04.2025. Revised: 30.04.2025. Accepted: 12.05.2025. Published: 12.05.2025.

Risk factors of status epilepticus in children - A literature review

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ABSTRACT

Introduction: Status epilepticus is one of the most common and severe neurological emergencies in children. It is defined by prolonged or recurrent seizures without full recovery in between and is associated with significant morbidity and mortality. This study aims to identify predictors of SE development and poor prognosis in children.

Material and Methods: A focused literature search was conducted in PubMed for studies published between 2015 and 2025, targeting human research related to pediatric status epilepticus. Search terms included “status epilepticus”, “children” and “risk factors”. Relevant meta-analyses, observational studies, and clinical trials were reviewed and evaluated for methodological quality and clinical significance.

State of Knowledge: SE is a neurological emergency, where ongoing seizures lead to drug resistance and brain injury. Classification by seizure duration informs treatment urgency. Benzodiazepines, especially intramuscular midazolam, remain first-line, with levetiracetam as a common second-line option. While mortality is declining, outcomes depend on seizure length, cause, and care setting, underscoring the need for rapid intervention**.**

Discussion: This study identifies key clinical and laboratory factors influencing the development and outcomes of SE in children. Younger age, perinatal complications, neurodevelopmental delays, and prolonged or biphasic seizures were significantly linked to higher risk and poorer outcomes. Laboratory markers were also associated with worse prognosis. Infectious, autoimmune, and genetic etiologies emerged as important underlying causes of SE, underscoring the need for early identification and targeted interventions.

Conclusion: Early age, early-life complications, seizure duration, and metabolic imbalances are major indicators of poor prognosis in pediatric status epilepticus. Recognizing infectious, autoimmune, and genetic triggers is essential for timely and effective intervention.
Key words: Status epilepticus, risk factors, children

1.INTRODUCTION AND PURPOSE

Status epilepticus (SE) is the most common life-threatening medical emergencies in children, with and annual incidence ranging from 10 to 58 per 100,000 [1,9,20]. This serious neurological emergency condition characterized by prolonged seizures or a series of seizures without recovery in between [17]. In children, the risk factors for developing status epilepticus can vary widely and may include underlying medical conditions, genetic predispositions, and environmental triggers. Understanding these risk factors is crucial for early identification and intervention, which can significantly improve outcomes for affected children. The following study aims to explore the various elements that contribute to the likelihood of status epilepticus in pediatric populations, highlighting the importance of awareness and proactive management in mitigating risks and ensuring the well-being of young patients.

The epidemiology of status epilepticus is complicated by inconsistent definitions, challenges in identifying all cases, and imprecise estimates of the denominator [17]. The phrase "time is brain" underscores the urgency of the situation, as prolonged seizures can lead to alterations in synaptic receptors, creating a more proconvulsant environment and heightening the risk of brain lesions and long-term complications such as neuronal injury or death [16,20]. Effective management of SE should focus on three key pillars: halting the seizures, stabilizing the patient to prevent secondary injuries, and addressing any underlying causes. Convulsive SE is classified as such after 5 minutes and is considered a medical emergency [15,16].

2.MATHERIALS AND METHODS

A comprehensive review of the literature available in the PubMed database was performed to evaluate the risk factors linked to Status Epilepticus in children. The search encompassed publications from 2015 to 2025, with a particular emphasis on studies involving human subjects, including meta-analyses of observational studies and randomized controlled trials. Key phrases such as "Status Epilepticus," "Children," and "risk factors" were utilized in the search strategy. The chosen articles were carefully assessed based on their findings and conclusions.

3.DESCRIPTION OF A STATE OF KNOWLADGE

3.1. Patopsychology

Status epilepticus (SE) is increasingly recognized as a dynamic process reflecting the brain's failure to transition from the ictal to post-ictal state. Instead of a discrete event, SE represents a pathological cycle in which seizure activity becomes self-perpetuating, exhausting endogenous inhibitory processes and reinforcing excitatory mechanisms. This cycle results in reduced seizure termination capacity and contributes to treatment resistance as SE progresses.

From an electrophysiological perspective, SE is marked by fluctuating EEG patterns and changes in autocorrelation and mean power, indicating unstable neural dynamics. In biological terms, this instability may stem from a failure of mechanisms that normally terminate seizures—particularly GABAergic inhibition. Pharmacologic agents that enhance these inhibitory pathways, such as benzodiazepines or neurosteroids, can help push the brain out of the ictal state. However, as seizures persist, pharmacoresistance emerges.

Experimental models show that prolonged stimulation (>30 minutes) can induce self-sustaining SE, even after the stimulus ends. This state may be temporarily suppressed with anesthetics but can recur, indicating the development of refractory or super-refractory SE. In humans, the longer a seizure persists, the lower the chance of spontaneous cessation, possibly due to alterations in neuronal networks, receptor expression, and neurotransmitter release.

Key to the resistance in prolonged SE is the internalization of synaptic GABA(A) receptors, especially those containing the γ-subunit, driven by NMDA receptor activation and calcineurin signaling. This process not only diminishes the effectiveness of benzodiazepines but also underscores the potential of NMDA antagonists, calcineurin inhibitors, and drugs targeting extrasynaptic GABA(A) receptors in treatment-refractory cases. Concurrently, the impairment of the KCC2 transporter disrupts chloride homeostasis, further reducing GABAergic efficacy.

Prolonged seizures also trigger a cascade of metabolic and inflammatory responses. Initially, the body compensates through increased blood pressure, heart rate, and cerebral blood flow. But as the seizure continues, these mechanisms fail, leading to systemic and cerebral hypoxia. Sustained glutamatergic activation promotes calcium influx, which activates nitric oxide synthase, calpains, and NADPH oxidase—key mediators of oxidative stress. Mitochondrial calcium overload leads to dysfunction and energy failure, while blood-brain barrier disruption and neuroinflammation further exacerbate neuronal injury.

These events culminate in excitotoxicity, apoptosis, and potentially long-term alterations in neural circuits. EEG patterns such as periodic lateralized epileptiform discharges and after-SE ictal discharges (ASIDs) following SE resolution are associated with poor prognosis and high mortality, reflecting persistent network instability [2,18].

3.2. Classification of Status Epilepticus Based on Seizure Duration and Risk of Long-Term Consequences

Status epilepticus (SE) is defined not only by the presence of continuous or rapidly recurring seizures, but also by specific operational time points that reflect the urgency of intervention and the potential for long-term neurological damage. These time points have been established to guide clinical decision-making. The first time point (t_1) indicates the duration beyond which a seizure is unlikely to terminate spontaneously and therefore requires prompt medical treatment to halt seizure activity. The second time point (t_2) marks the threshold beyond which ongoing seizure activity is believed to cause long-term consequences, such as neuronal injury, neuronal death, synaptic network reorganization, and persistent functional deficits. These time thresholds vary depending on seizure type. For generalized tonic–clonic status epilepticus, t_1 is defined at 5 minutes and t_2 at 30 minutes. In focal status epilepticus with impaired consciousness, intervention is recommended after 10 minutes (t_1), with potential for long-term consequences beyond 60 minutes (t_2). In absence status epilepticus, the time to initiate treatment typically ranges between 10 and 15 minutes (t_1); however, the duration at which long-term effects occur (t_2) remains uncertain due to limited data. These distinctions underscore the critical importance of rapid identification and timely treatment initiation in SE to minimize neurological injury and improve patient outcomes [8,13].

3.3. Treatment:

3.3.1. First-Line Treatment

Initial management of pediatric status epilepticus (SE) involves rapid administration of benzodiazepines, typically midazolam, lorazepam, or diazepam, with up to two doses [4,6]. In the absence of IV access, rectal diazepam remains a common option in younger children. A meta-analysis of 16 randomized trials (including six double-blinded) found that intramuscular (IM) midazolam and IV lorazepam are equally effective and superior to IV or IM diazepam in terminating seizures, with comparable risks of respiratory depression [3,7,10].

Non-IV routes have shown practical advantages. Systematic reviews and recent trials demonstrate that IM midazolam achieves faster administration and seizure control compared

to IV or rectal diazepam, while maintaining similar safety profiles. Given that treatment delays may prolong SE, non-IV and rectal formulations remain valuable in prehospital and emergency settings [7,10].

Neonatal seizures are traditionally treated with phenobarbital, followed by fosphenytoin. However, randomized data suggest that neither agent is definitively superior or consistently effective.

3.3.2. Second-Line Treatment

Phenytoin has long been the standard second-line therapy after benzodiazepine failure. Recent trials (ConSEPT and EcLiPSE) have evaluated levetiracetam as an alternative, showing non-inferiority with a better side-effect profile and simpler administration. While not superior, levetiracetam is a reasonable alternative. Results from the ongoing ESETT trial may provide further guidance [3,7].

3.3.3. Third-Line and Refractory SE (RSE/SRSE)

For RSE or super-refractory SE (SRSE), treatment options expand significantly, though high-quality evidence remains limited. Reported therapies include continuous infusions of benzodiazepines, barbiturates, propofol, and ketamine; inhaled agents such as isoflurane; and alternative approaches like immunotherapy, corticosteroids, neurosteroids, lidocaine, hypothermia, ECT, vagus nerve stimulation, magnesium, and the ketogenic diet. Most data come from case reports or small studies, and treatment should be individualized based on clinical context [3,7].

3.4. Mortality

Mortality in pediatric status epilepticus (SE) remains a critical concern, though its incidence has declined in recent decades, particularly in high-resource settings. Short-term mortality—defined as death occurring within 30 days or by hospital discharge—varies significantly across studies. In cohorts from the post-2000 era, reported short-term mortality rates range from 2.1% to 6%, compared to 2.7% to 11.5% in studies conducted prior to 2000. Notably, studies from developing countries have reported higher short-term mortality, with rates ranging between 7.1% and 17.5% [5].

These trends likely reflect advances in neurocritical care, early recognition, and improvements in prehospital emergency services. A 2007 systematic review focusing on high-quality studies reported a pooled short-term mortality rate of 2.7% to 5.2% [4]. Long-term mortality, which

encompasses deaths occurring after hospital discharge, has been reported to range from 2.3% to 11%, depending on the duration and methodology of follow-up [5].

A variety of factors have been implicated in SE-related mortality, including the underlying etiology, patient age, duration of seizure activity, and the presence of neurological or systemic comorbidities. Methodological differences between studies—such as variations in SE definitions, age cohorts, follow-up periods, and whether data were drawn from hospital- or population-based samples—also contribute to the variability in reported outcomes. Additionally, study quality, including sample size, statistical power, and potential biases, must be considered when interpreting mortality data [5].

4.DICUSSION

This study provides a detailed evaluation of the clinical and laboratory risk factors associated with the development of status epilepticus (SE) in children following a first epileptic seizure, as well as predictors of poor outcomes in pediatric SE. The results highlight several statistically significant and clinically meaningful predictors that can guide both acute management and long-term prognosis.

4.1. Age as a Primary Risk Factor

Age emerged as one of the most powerful and consistent predictors of both the development of SE and its unfavorable outcomes. Children under 1 year of age demonstrated a significantly higher risk of progressing to SE (OR: 1.29, $p < 0.001$), and when analyzing outcomes, those under 24 months had markedly worse prognoses (OR: 29.76, $p = 0.001$). This finding is in line with previous studies suggesting that the immature brain is more susceptible to excitotoxic injury due to prolonged epileptic activity. The increased vulnerability in infants and toddlers may stem from both physiological factors—such as immature inhibitory neurotransmission and incomplete myelination—as well as diagnostic challenges that delay timely intervention in this age group [11,12,14,19].

4.2. Perinatal and Developmental Background

Perinatal complications were also found to be strongly linked to SE development. Children with a history of perinatal suffering had nearly double the odds of developing SE (OR: 1.96, $p < 0.001$). Neuromotor developmental delay, often a surrogate marker for underlying structural or metabolic brain abnormalities, had an even stronger association (OR: 5.0, $p < 0.001$). These findings highlight the role of pre-existing brain vulnerability in determining seizure severity and outcome. Motor or mental retardation was also associated with poor outcome following

SE, further emphasizing the importance of comprehensive developmental assessment in seizure evaluation [11,12,14,19].

4.3. Seizure History and Recurrence Patterns

Previous seizure history, including febrile and afebrile episodes, was associated with increased risk of SE (OR: 1.4, $p = 0.008$). However, when analyzing outcomes, past seizures did not differentiate significantly between good and poor prognosis groups. It is possible that seizure recurrence indicates a predisposition to a more unstable epileptic network, but does not independently determine functional outcome, especially when appropriate treatment is initiated.

The inaugural presentation of SE—where SE is the first recognized seizure—was significantly associated with future episodes of SE (OR: 1.3, $p < 0.001$). Seizure relapses within 24 hours were also associated with increased risk (OR: 1.4, $p = 0.01$), underscoring the need for close observation during the initial hours of recovery, particularly in patients with prolonged or complex seizures [11,12,14,19].

4.4. Seizure Duration and Intractability

Prolonged seizure activity has long been associated with increased risk of morbidity, and this study reinforces that understanding. Seizures lasting more than 90 minutes were strongly predictive of poor outcomes (OR: 2.93, $p = 0.048$), which is consistent with prior evidence indicating that seizure-induced neuronal injury is both time- and intensity-dependent. Intractable seizures, defined here as those unresponsive to standard first- and second-line antiepileptic therapy, were also a significant predictor of poor prognosis (OR: 4.18, $p = 0.008$). These findings support the need for aggressive and early escalation of therapy when initial treatment fails, potentially including continuous infusion therapy or anesthetic agents [11,12,14,19].

4.5. Seizure Pattern: Biphasic and Generalized Seizures

Biphasic seizures, characterized by an initial seizure followed by a transient recovery and subsequent recurrence, were the strongest predictor of poor outcome in our cohort (OR: 10.06, $p < 0.001$). These seizures may reflect evolving underlying brain pathology or treatment resistance, and they complicate diagnosis and timely intervention. Generalized seizures were the most common seizure type observed but did not independently correlate with outcome, possibly because they include a broad spectrum of severity and underlying etiologies [11,12,14,19].

4.6. Glasgow Coma Scale and Neurological Status

An initial Glasgow Coma Scale (GCS) score of ≤14 was more commonly observed in children who developed SE (OR: 1.22, p = 0.018), suggesting that early signs of encephalopathy or postictal depression may be valuable predictors. Similarly, abnormalities in neurological examinations, though not an independent predictor of poor outcome, were more frequently seen in SE cases, reinforcing the need for detailed and repeated neurological assessments [11,12,14,19].

4.7. Laboratory and Biochemical Risk Factors

Laboratory abnormalities play a significant role in the pathogenesis and prognosis of status epilepticus (SE) in pediatric patients. Among the most notable findings is the impact of blood glucose dysregulation. Both hypoglycemia (<61 mg/dL) and hyperglycemia (>250 mg/dL) have been strongly associated with poor outcomes in children experiencing SE, with an odds ratio of 8.82 (p = 0.005). These glucose imbalances may indicate systemic metabolic stress or underlying illness and demand immediate correction to avoid further neuronal injury [9,11]

Electrolyte disturbances also emerged as critical contributors to acute symptomatic SE. The clinical data reveal cases of hyponatremia (4 patients) and hypernatremia (1 patient), both of which are known to alter neuronal excitability and may precipitate or exacerbate seizures. Furthermore, hypoglycemia was observed in 2 children, reinforcing the importance of glucose homeostasis in seizure control. Additional acute metabolic or biochemical triggers included drug-induced SE (3 patients) and hypoxic-ischemic encephalopathy (1 patient), the latter of which often results in severe metabolic derangement contributing to seizure persistence [9].

These findings underscore the importance of thorough laboratory evaluation in pediatric SE, particularly regarding glucose and electrolyte levels, to guide timely therapeutic interventions and reduce morbidity. Early identification and management of these derangements are essential for improving outcomes in this vulnerable population.

Elevated serum aspartate aminotransferase (AST) levels (≥56 U/L) were also strongly predictive of poor outcome (OR: 4.71, p < 0.001). Elevated AST can serve as a proxy for global tissue injury, including hepatic and muscular damage secondary to prolonged seizures, hypoxia, or systemic inflammation. Given its strong correlation with other enzymes such as ALT and LDH, AST was chosen as the representative biomarker for multivariate analysis.

C-reactive protein (CRP), a nonspecific inflammatory marker, was also predictive of poor outcome when levels exceeded 2.00 mg/dL (OR: 4.55, p = 0.005). This suggests a potential

role of systemic inflammation in modulating seizure threshold and recovery, possibly through cytokine-mediated neuroinflammation or blood-brain barrier disruption [11].

4.8. Antiepileptic Therapy and Treatment Adherence

A history of epilepsy (OR: 1.6, $p = 0.04$) and abrupt discontinuation of antiepileptic therapy (OR: 3.2, $p < 0.001$) were both associated with increased SE risk. Non-adherence or sudden withdrawal of medications has long been recognized as a precipitating factor for SE, particularly in patients with known epilepsy. These findings highlight the crucial importance of caregiver education and structured weaning protocols when modifying or withdrawing treatment [11].

4.9. Infectious causes

Infectious causes play a significant role in the etiology of refractory status epilepticus (RSE) and super-refractory status epilepticus (SRSE), with marked geographic variability. In particular, studies from South Asia, including India, have consistently reported a predominance of infectious etiologies. A notable study involving 148 adults with encephalitis identified 18 cases of SE, predominantly in patients with herpes simplex virus (HSV) and Japanese encephalitis, with children being particularly vulnerable to encephalitis-related SE. Crucially, early antiviral therapy, especially the administration of acyclovir within 24 hours of symptom onset in HSV encephalitis, has been associated with significantly improved outcomes.

Neurocysticercosis has also emerged as a relevant infectious cause of SE. In a recent cohort, 41 patients with neurocysticercosis developed convulsive SE; however, none progressed to RSE or SRSE. Interestingly, those with single calcified lesions experienced shorter SE duration than individuals with degenerating cysts. Another prospective study involving 141 children presenting with acute convulsive seizures reported neurocysticercosis in nearly half (49%) of the cases, although progression to RSE was not documented. Data on other common infectious causes, such as acute bacterial meningoencephalitis, cerebral malaria, and dengue, remain limited. Differentiating between acute symptomatic SE due to infection and febrile SE in children remains a diagnostic challenge with critical therapeutic implications [3].

4.10. Autoimmune Factors

Autoimmune encephalitis constitutes another increasingly recognized etiology of RSE/SRSE, particularly anti-NMDA receptor (anti-NMDAR) encephalitis. This condition may initially present with nonspecific prodromal symptoms such as fever and headache. As cortical

involvement progresses, children may exhibit prolonged seizures, behavioral disturbances (e.g., temper tantrums, aggression, speech regression), and eventually RSE/SRSE. Notably, unlike the temporal lobe onset often seen in adults, seizure foci in children may be extra-temporal or diffusely bilateral, warranting high clinical suspicion even in the absence of classic EEG findings. Other forms of limbic encephalitis, including non-paraneoplastic types, are increasingly diagnosed in pediatric populations. In contrast to adults, where symptoms include memory impairment and mood disturbances, pediatric patients may demonstrate broader executive dysfunction, including impaired attention, poor social regulation, and behavioral disinhibition, necessitating a developmental-context evaluation [3]

4.11. Genetic Etiology

Genetic epilepsies represent a crucial etiological category of pediatric RSE/SRSE. Prompt recognition of genetic syndromes such as Dravet syndrome, SCN1A-related epilepsies, ring chromosome 20 syndrome, and mitochondrial disorders involving POLG or aminoacyl-tRNA synthetase genes are essential. These syndromes not only predispose to prolonged seizures but often dictate specific therapeutic responses and prognoses. Angelman syndrome, characterized by severe developmental delay and epileptic encephalopathy, is another example where early genetic diagnosis can inform both acute and long-term management strategies [3].

5.CONCLUSIONS

This study contributes to the growing body of literature on pediatric status epilepticus (SE) by identifying and consolidating key clinical, biochemical, and etiological risk factors associated with both its development and adverse outcomes. Among these, age—particularly infancy—emerges as a powerful predictor, underscoring the unique vulnerability of the immature brain to excitotoxic injury. Similarly, perinatal insults, neurodevelopmental delay, and seizure duration are strongly linked with poor prognosis, highlighting the importance of early risk stratification and intervention. Biochemical disturbances, especially glucose and electrolyte imbalances, along with elevated inflammatory and hepatic markers, offer valuable prognostic insight and reinforce the need for prompt metabolic correction. Furthermore, the presence of identifiable infectious, autoimmune, or genetic etiologies should prompt targeted diagnostic and therapeutic approaches, as these subgroups may require etiology-specific interventions. Finally, the role of treatment adherence and seizure recurrence patterns supports the necessity of comprehensive follow-up and caregiver education. Taken together, these findings advocate for a multidimensional and personalized approach to managing pediatric SE—one that

integrates clinical vigilance, early etiological workup, and aggressive, protocol-driven treatment to improve both short- and long-term outcomes.

6. DISCLOSURE

Authors do not report any disclosures.

Author's contributions

Conceptualization: KM, PZ;

Methodology: KM, PZ;

Software: n/a; check: KM, MP, PZ;

Formal analysis: KM, AM;

Investigation: KM, PZ, MP, TK, MK, AM;

Resources: KM;

Data curation: KM,P,Z MP, ŁK, MK, AJ, JM, MB, AC, TK;

Writing - rough preparation: KM, PZ MP;

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Visualization: PZ, MP, JM;

Supervision: MK, AJ, TK;

Project administration: KM;

Receiving funding: n/a.

All authors have read and agreed with the published version of the manuscript.

Funding statement

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data availability statement

Not applicable.

Acknowledgments

The authors declare that there are no acknowledgments for this study.

Conflict of Interest Statement

The authors declare no conflict of interest.

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