ORIGINAL ARTICLE

Status Epilepticus in Children: Clinical Features, Management, and Outcomes in a Single-Center Experience

Hamit Acer, MD[1]; Mehmet Canpolat, MD[2]; Hüseyin Per, MD[2]; Hakan Gumus, MD[2]; Basak Nur Akyildiz, MD[3]; Sefer Kumandas, MD[2]

[1]Pediatric Neurology, Department of Pediatrics, Denizli State Hospital, Denizli, TURKEY
[2]Department of Pediatrics, Division of Pediatric Neurology, Erciyes University, Faculty of Medicine, Kayseri, TURKEY
[3]Department of Pediatrics, Division of Pediatric Intensive Care, Erciyes University, Faculty of Medicine, Kayseri, TURKEY

Received: 21 Aug 2025
Accepted: 20 May 2026
Published: 1 Sep 2026

Keywords:

Status epilepticus
Seizure management
Antiepileptic drugs

ABSTRACT

Objectives: The classification of status epilepticus (SE) has undergone several revisions, with the most recent update published by the International League Against Epilepsy (ILAE) in 2015. However, in seizures lasting longer than 10 minutes, there is still no consensus on optimal drug selection.
Materials and Methods: This retrospective study reviewed the medical records of patients diagnosed with SE and followed in the Pediatric Intensive Care Unit, Pediatric Neurology, and Pediatric Emergency Unit at Erciyes University Faculty of Medicine.
Results: A total of 64 patients were included. Sequential therapy with levetiracetam following phenytoin proved to be an effective treatment strategy prior to escalation to third-line agents. In our clinic, rapid MRI protocols, including axial T1, axial T2, sagittal T2, and diffusion-weighted sequences, were preferred for emergency neuroimaging. These rapid protocols were both practical and effective in the acute management of SE.
Conclusions: Rapid brain MRI techniques are easy to apply and provide valuable diagnostic insights into the etiology of SE. Sequential administration of second-line drugs may reduce the side-effect profile and optimize seizure control in children with SE.

How to cite this article: Acer H, Canpolat M, Per H, Gumus H, Akyildiz BN, Kumandas S. Status Epilepticus in Children: Clinical Features, Management, and Outcomes in a Single-Center Experience. Iran J Child Neurol. 2026;20(4):e4. https://doi.org/10.22037/ijcn.v20i4.50012

Introduction

Prolonged or recurrent seizures characterize status epilepticus (SE) without a return to baseline. Gastaut et al. initially proposed a definition consistent with the meaning of the original term status in Latin: "Status epilepticus is a term whenever a seizure that persists for a sufficient length of time or is repeated frequently enough to produce a fixed and enduring condition" (1).

Traditionally, SE has been defined as “a seizure lasting longer than 30 minutes or the recurrence of seizures without recovery of consciousness during this period” (2). The estimated incidence of SE is up to 61 episodes per 100,000 persons per year, with an overall mortality rate of approximately 20% (3). Most seizures terminate within 5 minutes; seizures lasting longer than 5 minutes are more likely to progress to SE (4).

The International League Against Epilepsy (ILAE) published a special report on the definition and classification of SE in 2015. According to this report, SE arises either from failure of normal seizure-termination mechanisms or from the initiation of

Corresponding Author:
Hamit Acer
Email: dr_hamitacer@hotmail.com

This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International (CC BY-NC 4.0) which allows users to read, copy, distribute and make derivative works for non-commercial purposes from the material, as long as the author of the original work is cited properly.

processes leading to abnormally prolonged seizures beyond time point t1. Past time point t2, persistent seizure activity can produce long-term sequelae such as neuronal death, neuronal injury, and altered neural networks, varying by seizure subtype and duration (5). Operationally, t1 indicates the duration threshold requiring prompt intervention to prevent continuous seizures, whereas t2 marks the time point beyond which long-term neurological consequences may occur.

According to the ILAE definition, the operational time points vary according to the type of SE. For generalized tonic-clonic convulsive SE, t1 is 5 minutes and t2 is 30 minutes. For focal SE with impaired consciousness, t1 is 10 minutes, whereas t2 is considered to be longer than 60 minutes. For absence SE, t1 is 10-15 minutes, while available evidence is insufficient to define t2 (6).

Early diagnosis and appropriate treatment of SE are essential to minimize morbidity and mortality rates associated with SE. In this study, clinical and demographic data and the prognosis of the cases followed up in the Pediatric Neurology Clinic with a diagnosis of SE will be discussed in light of the literature.

Materials & Methods

This study was conducted between 2000 and 2021, and the medical records and hospital information system data of patients diagnosed with SE who were followed up in the Pediatric Intensive Care Unit, Pediatric Neurology Clinic, and Pediatric Emergency Unit of Erciyes University, Faculty of Medicine, were retrospectively reviewed.

The data were recorded on a predesigned standardized form. Patients were classified and grouped according to age, sex, seizure type, primary diagnosis, biochemical markers, complete blood count, neuroimaging findings, electroencephalogram (EEG), medications used, effective drugs, drug side-effect profile, and prognosis. Patients with insufficient data in hospital records or files were excluded.

The study protocol was approved by the Ethics Committee of Erciyes University. Cases diagnosed with convulsive SE were included. Seizure durations were categorized as 10-30 min, 30-60 min, and >60 min (6). For generalized tonic-clonic seizures, SE was defined as a seizure duration longer than 5 minutes; for focal or focal-onset seizures, the threshold was defined as longer than 10 minutes.

Seizure types were grouped according to the ILAE 2017 classification as focal onset, generalized onset, and unknown onset. Primary etiologies were categorized into distinct groups: febrile, febrile infection-related epilepsy syndrome (FIRES), limbic encephalitis, subacute sclerosing panencephalitis (SSPE), and demyelinating disorders, the latter defined by characteristic neuroimaging findings in the absence of a known cause. Remaining etiologies were classified as neurometabolic, neurocutaneous, asphyxial, infarction-related, or post-hypoglycemic sequelae, with additional cases grouped under genetic epileptic syndromes or miscellaneous causes.

Neuroimaging findings were classified into 10 groups: normal, cortical malformation, infarct, cerebral atrophy, findings secondary to asphyxia, demyelinating lesion, cerebral edema, hemorrhage, hydrocephalus, and sequelae of hypoglycemia. EEG findings were grouped as normal, minimal background rhythm abnormalities, focal epileptiform discharges, generalized epileptiform discharges, severe diffuse background rhythm abnormalities, and burst-suppression pattern.

Data were analyzed using IBM SPSS Statistics 22.0 software. Descriptive statistics were presented as frequency (n), percentage (%), mean ± standard deviation (SD), median, and 25th-75th percentile [M (Q1-Q3)]. The normal distribution of numerical variables was assessed using the Shapiro-Wilk test. The Student-Newman-Keuls test was used for multiple comparisons. A p-value < 0.05 was considered statistically significant.

Results

Eighty patients diagnosed with SE were evaluated, of whom 64 were included in the study. Sixteen patients were excluded due to insufficient retrospective data. Among the patients, 36 (56.3%) were male and 28 (43.8%) were female. The mean age was 58.2 months, with a median of 36 months (range: 1.2-180 months). When classified according to seizure onset, 16 (25.0%) had focal-onset seizures, 35 (54.7%) generalized-onset seizures, and 13 (20.3%) seizures of unknown onset.

All included patients had seizures meeting the predefined duration criteria for SE. Seizure duration was 10-30 minutes in 30 patients (46.9%), 30-60 minutes in 21 (32.8%), and >60 minutes in 13 (20.3%).

A family history of febrile seizures was present in 7 patients (10.9%), most frequently in the febrile SE group. Fifteen patients had a history suggestive of asphyxia (incubator stay, mechanical ventilation, or need for advanced life support in the intensive care unit). Serum glucose, potassium, calcium, phosphorus, and magnesium levels were within normal limits, while two patients had sodium levels below 130 mEq/L.

All patients underwent neuroimaging: CT in 3 cases and MRI in the remaining patients. Imaging was

performed between 1 hour and 7 days after SE onset. Nineteen patients had normal imaging findings. In the febrile SE group, 9 patients (81.8%) had normal imaging, whereas 2 showed cerebral edema. Cerebral atrophy was detected in 13 patients (20.3%), while asphyxia-related findings were present in 9 (14.1%). One patient had intracranial hemorrhage.

EEG was performed in all patients between 1 hour and 7 days after SE onset. Continuous EEG monitoring during seizures or for assessing infusion therapy effectiveness could not be performed due to technical limitations. Recordings lasted 30 minutes. The most common abnormality was focal epileptiform discharges in 16 patients (25.0%).

Regarding etiology, febrile SE in 11 patients (17.2%) and asphyxia-related SE in 12 (18.7%) were among the most common causes. The etiology could not be clarified in six patients despite metabolic testing, biochemical markers, and neuroimaging.

For first-line treatment, rectal diazepam, buccal midazolam, or both were administered sequentially. Twenty-five patients (39.0%) received buccal midazolam only, 28 (43.7%) rectal diazepam only, and 11 (17.2%) both sequentially. Before hospital admission, 39 patients (60.9%) had received rectal diazepam administered by family or healthcare staff, while 12 (18.7%) had received buccal midazolam administered by healthcare staff. Differences in pre-hospital treatment were mainly related to drug availability, usage habits, and the administering individual.

Table 1. Status Epilepticus Treatment Stages and Drug Efficacy

Stage	Drugs	Use in cohort	Seizure termination among recipients
Stage 1	Rectal diazepam	28/64 (43.7%) only	-
	Buccal midazolam	25/64 (39.0%) only	
		11/64 (17.2%) sequential	
Stage 2	Phenytoin	27 recipients	13/27 (48.1%)
	Levetiracetam	23 recipients	11/23 (47.8%)
	Sodium valproate	10 recipients	4/10 (40.0%)
Stage 3	Midazolam infusion	37/64 (57.8%)	17/37 (45.9%)
	Thiopental sodium	10/64 (15.6%)	6/10 (60.0%)
	Propofol	1/64 (1.6%)	1/1 (100%)
Refractory	IVIG	6/64 (9.4%)	3/6 (50.0%)
	PMP	2/64 (3.1%)	2/2 (100%)
	Topiramate	4/64 (6.3%)	1/4 (25.0%)
	Ketamine	5/64 (7.8%)	1/5 (20.0%)
	Lidocaine	2/64 (3.1%)	1/2 (50.0%)

Second-line treatment consisted of phenytoin, levetiracetam, and sodium valproate. Two patients required sequential intravenous loading with all three antiseizure medications (ASMs). Phenytoin followed by sodium valproate was administered sequentially in 7 patients (10.9%), whereas phenytoin followed by levetiracetam was administered in 20 (31.3%). No patient received sodium valproate followed by levetiracetam sequentially. Seizure termination was achieved with phenytoin in 13 patients (20.3%), levetiracetam in 11 (17.2%), and sodium valproate in 4 (6.3%). In patients whose seizures persisted after the initial second-line ASM, another second-line ASM was administered sequentially before escalation to continuous infusion therapy.

Third-line therapy included continuous infusion of midazolam, thiopental sodium, or propofol. Thirty-seven patients (57.8%) received midazolam infusion, 10 (15.6%) thiopental sodium, and 1 (1.6%) propofol infusion. Midazolam was the first-choice continuous infusion therapy, with alternative therapies used in refractory cases. Seizure termination was achieved in 17 of 37 patients (45.9%) receiving midazolam infusion, 6 of 10 patients (60.0%) receiving thiopental sodium infusion, and the single patient receiving propofol infusion.

In refractory cases, additional therapies such as pulse methylprednisolone (PMP), intravenous immunoglobulin (IVIG), topiramate, lidocaine, and ketamine were used. Six patients received IVIG, two PMP, four topiramate, five ketamine, and two lidocaine infusions. The seizure-terminating treatment was IVIG in 3 patients (4.7%), PMP in 2 (3.1%), topiramate in 1 (1.6%), ketamine infusion in 1 (1.6%), and lidocaine infusion in 1 (1.6%). Seizure control could not be achieved in one patient (1.6%). Patients responding to immunotherapy included two with FIRES, two with limbic encephalitis, and one with asphyxia-related West syndrome. Topiramate was effective in one patient with non-ketotic hyperglycinemia.

The most common adverse effects were decreased respiratory effort and respiratory depression, followed by hypotension and bradycardia. Midazolam infusion was the most frequent cause of respiratory complications.

Sequelae developed in 3 of 30 patients (10.0%) with seizure duration of 10-30 minutes, 3 of 21 (14.3%) with duration of 30-60 minutes, and 5 of 13 (38.5%) with duration >60 minutes. Overall, 11 of 64 patients (17.2%) developed sequelae. The highest sequela rate was observed in the >60-minute group.

Sequelae observed in prolonged SE included hemiparesis, moderate-to-severe intellectual disability, and moderate-to-severe delay in motor milestones. In the shorter-duration group, sequelae were limited to milder neurological deficits such as mild learning difficulties and borderline intellectual functioning.

Overall, 8 of 64 patients (12.5%) died. No deaths occurred in the 10-30-minute group. Mortality occurred in 6 of 21 patients (28.6%) in the 30-60-minute group and in 2 of 13 (15.4%) in the >60-minute group. Diagnoses among patients who died included non-ketotic hyperglycinemia, phenylketonuria, adrenal insufficiency, autoimmune epilepsy (n=2), and asphyxia-related West syndrome (n=3).

Discussion

SE represents a major pediatric neurological emergency warranting prompt and effective intervention to mitigate associated morbidity and mortality (7). In the present study, the mean patient age was 58.2 months. Consistent with the literature indicating that most SE episodes occur in children under three years of age (8), 34 patients (53.1%) were younger than three years and 14 (21.8%) were younger than one year. Generalized-onset seizures were the most common clinical presentation, occurring in 35 patients (54.7%), followed by focal-onset seizures.

Patients were evaluated in three seizure-duration groups: 10-30 minutes, 30-60 minutes, and >60 minutes. Seizure duration was 10-30 minutes in 30 patients (46.9%), 30-60 minutes in 21 (32.8%), and >60 minutes in 13 (20.3%). Thus, 34 patients (53.1%) had seizures lasting longer than 30 minutes and 13 (20.3%) had seizures exceeding 60 minutes (9). Ongoing seizures despite intervention in the initial treatment phase were included in the study. Refractory SE develops in approximately %10-40 of children with SE (10). In another study, seizures last longer than 1 hour in %26-45 of SE cases (11). In a previous study involving 89 pediatric patients with status epilepticus, seizure duration was 30–60 minutes in 65.2% of patients and exceeded 60 minutes in 34.8% (12). Refractory and super refractory SE rates were higher than in other studies due to the study inclusion of ongoing seizures despite buccal midazolam and rectal diazepam treatment.

The American Academy of Neurology (AAN) recommends neuroimaging after SE stabilization and seizure control. The Neuro-Intensive Care Association recommends neuroimaging within the first 60 minutes of the onset of SE (17). Neuroimaging was performed with a first preference for MRI in all patients. After the stabilization of the patient, imaging was performed. Although early imaging has been attempted, imaging has been delayed in some patients due to technical facilities and a prolonged patient stabilization phase. ILAE has stated that abnormal imaging results were obtained in approximately %50 of new-onset localization-related epilepsy cases; imaging in %2–4 of patient contributes to the change in medical treatment (13). Neuroimaging is important in childhood SE for identifying the underlying etiology and guiding acute management. In brain MRI findings, 7 of 10 patients with localized results had focal or focal-onset seizures. In neuroimaging, infarcts in 6 patients and demyelinating lesions in 3 patients, including 9 cases (%14), contributed to the immediate change in treatment. Emergency neuroimaging protocols preferentially employ rapid MRI techniques, including axial T1 and T2, sagittal T2, and diffusion-weighted sequences. The use of rapid MRI techniques may have provided an advantage in facilitating early treatment modifications.

Continuous EEG monitoring could not be performed because of technical limitations during the study period; a 30-minute EEG recording was evaluated for each patient. Nonconvulsive SE could therefore not be evaluated. Although focal epileptiform discharges were the most frequent EEG abnormality, generalized-onset seizures were the most common clinical presentation. This apparent discrepancy may partly reflect caregivers' greater recognition of dramatic generalized convulsions. Only one of the 11 patients with febrile SE had epileptiform activity, characterized by severe diffuse background rhythm abnormality; this patient died. Other patients with epileptiform activity were in acute symptomatic or remote symptomatic etiologic groups.

Approximately 25% of childhood SE cases have been reported to be febrile SE (14). Seizures may be generalized or focal, and in most cases seizure duration has been reported to be between 30 and 60 minutes (14). In the present study, febrile SE was identified in 11 of 64 patients (17.2%). Among the 11 patients with febrile SE, 7 (63.6%) had a seizure duration of 10-30 minutes. Seizures were tonic or tonic-clonic in all patients with febrile SE. Approximately 95% of febrile SE cases have been reported to occur in children younger than five years (15), whereas all 11 patients (100%) with febrile SE in our series were younger than five years.

SE secondary to autoimmune etiologies is less common but still well-described. In a recent prospective cohort study in adults, autoimmune and

paraneoplastic etiologies accounted for %2.5 of cases of SE (16). SE due to autoimmune causes was seen in 6 (%9.4) patients. Three cases were diagnosed as FIRES, 2 cases with limbic encephalitis, and one case with SSPE. The recent proportional increase in the diagnosis of resistant epilepsy is largely attributable to the facility's status as a tertiary referral center, combined with the specialized monitoring of such resistant cases within the unit.SE treatment consists of 4 stages. Providing and maintaining vital functions and preventing complications during this period; regulating drug therapy to stop seizures; detecting the cause of SE; preventing seizure recurrence. The drug used in the treatment of SE should be an intravenous (IV) preparation; the effect should start quickly, be easy to administer, have a broad spectrum of activity, have a short elimination time, and have a broad therapeutic safety margin.

Benzodiazepines remain the cornerstone of initial treatment. The Neurocritical Care Society (NCS) recommends benzodiazepines as immediate initial therapy (17). According to the convulsive SE directive of the American Epilepsy Society (AES), administration of IV lorazepam and diazepam is useful in stopping seizures lasting at least five minutes (level A). Rectal diazepam, intramuscular, buccal, and intranasal midazolam applications are also effective (level B). Insufficient data of intranasal, sublingual, rectal lorazepam in children (level U) are available (17,18), because lorazepam is not available in our country. For this reason, buccal midazolam and rectal diazepam were used as first-line treatment. SE patients who were resistant to first-line treatment were included in the study.

In patients with ongoing seizure activity despite first-line benzodiazepine therapy, escalation to a second-line ASM is recommended (18). If SE persists after a 10-minute seizure, only benzodiazepines can provide seizure control in only half of the pediatric age group (19). Therefore, AES recommends giving one of the second-line drugs (20). This ongoing seizure activity is called Stage 2 SE. In Stage 2 SE, phenytoin/fosphenytoin, phenobarbital, valproate, levetiracetam, lacosamide) are commonly used, and there is no class I evidence to choose between drugs (21). Phenytoin, levetiracetam, and valproate were used as second-line drugs in the present cases. The unavailability of intravenous formulations precluded the administration of fosphenytoin and phenobarbital. Lacosamide treatment was not preferred during the follow-up of patients due to the lack of evidence and case series. In a meta-analysis of convulsive SE, despite benzodiazepine phenytoin (%50), levetiracetam (%69), phenobarbital (%74), and valproate (%76) were found to be effective in ongoing seizures (21,22). Our cohort, when efficacy was calculated among recipients, seizure termination occurred in 13/27 (48.1%) with phenytoin, 11/23 (47.8%) with levetiracetam, and 4/10 (40.0%) with sodium valproate. Before escalation to continuous infusion therapy, sequential use of second-line ASMs was preferred when the first second-line agent failed. Phenytoin followed by sodium valproate was used in 7 patients (10.9%), and phenytoin followed by levetiracetam in 20 (31.3%); two patients required sequential loading with all three second-line ASMs. These data describe the treatment sequences and seizure-control rates observed in the cohort but do not establish the superiority of one treatment approach over another. Following phenytoin treatment, seizure control was achieved in 11 of 20 patients (55%) who received levetiracetam. Among 21 patients who received an intravenous midazolam bolus after phenytoin and before escalation to third-line therapy, seizure control was achieved in 13 (61.9%).

According to the definition of NCS, the appropriate dose of the benzodiazepine and the state where seizures persist despite an antiepileptic are defined as refractory SE (RSE) (20-21). RSE develops in approximately 10-40% of children with SE (22). The mortality rate in children with RSE varies between %15 and %65 (23). Patients treated with a continuous infusion or inhaled anesthetics require intensive monitoring due to the problems including below: [1] mechanical ventilation for the protection of the airway, and maintaining proper oxygenation and ventilation; [2] frequent laboratory tests and vasopressors or central venous and arterial access due to the possibility of developing hypotension requiring inotropic support; [3] High dose sedatives and body temperature management as it may disrupt the shaking response of anesthetics and endogenous thermoregulation; [4] evaluation for the development of lactic acidosis, anemia, thrombocytopenia and end organ dysfunction (for example, acute hepatic or renal injury); [5] the risk of secondary infection due to permanent catheters (eg, central catheters, endotracheal tubes, and foley catheters) and certain medications (eg, pentobarbital) (17). In SE cases, the first drug achieves seizure control in 55% of cases; in cases where seizure cannot be controlled despite the first drug, the second drug activity is %7, and the third drug activity is %2.3.18 At this stage, Intravenous anesthetic drugs, also called refractory SE (thiopental/pentobarbital, midazolam, or propofol), have been used extensively for the last 30 years. In a global audit of 488 RSE episodes, continuous midazolam was the most commonly used anesthetic (59%), followed by propofol (32%) and barbiturates

(8%) (24). In our study, 37 patients (57.8%) received midazolam, 10 (15.6%) thiopental sodium, and 1 (1.6%) propofol infusion. These results were similar to those of previous studies (24).

Among treatment recipients, seizure termination was achieved in 17/37 (45.9%) with midazolam infusion, 6/10 (60.0%) with thiopental sodium, and in the single patient receiving propofol. Direct comparisons of efficacy are inappropriate because the numbers receiving thiopental sodium and propofol were small. The most common adverse effect of midazolam infusion was respiratory depression or decreased respiratory effort (67.6%). Other adverse effects included hypotension (40.5%) and bradycardia (35.1%). These findings emphasize the need for close respiratory and hemodynamic monitoring during continuous midazolam infusion.

In the 4th step treatment protocol, only case reports and a limited number of samples are available to guide the treatment. In a study of 602 SE pediatric cases, it was noticed that SRSE had progressed in 7.14% of the patients (25). Nineteen treatment courses were used from the 4th group medicines. Ketamine infusion treatment was given to five patients, lidocaine infusion to two, and topiramate to four. As the most common immunomodulatory treatments, corticosteroids, immunoglobulins, and plasmapheresis were applied.In our cohort, 6 patients received IVIG, 2 PMP, 4 topiramate, 5 ketamine, and 2 lidocaine. Among recipients, seizure termination occurred in 3/6 (50.0%) with IVIG, 2/2 (100%) with PMP, 1/4 (25.0%) with topiramate, 1/5 (20.0%) with ketamine, and 1/2 (50.0%) with lidocaine. These percentages should be interpreted cautiously because of the very small numbers. In a published series of 13 patients, ketamine infusion was given over a period ranging from 22 hours to 17 days. Importantly, it was found to be effective in 4 patients (26). In a study evaluating 58 cases, it was reported that complete or partial response was obtained in 19(32%) patients (27). In a previous study on topiramate use in our clinic, in the studied 14-case series, complete response was obtained in 9 cases and partial response in three (28). In this study, topiramate was effective in 25% of cases and ketamine in 20%. Lidocaine infusion was effective in one patient; the sample size was too small to draw reliable conclusions because it was used in 2 cases.

Underlying etiology and seizure duration are important determinants of outcome in SE. In our study, sequelae developed in 3/30 (10.0%) patients with seizure duration of 10-30 minutes, compared with 8/34 (23.5%) among those with seizure duration >30 minutes. The highest sequela rate was observed in patients with seizure duration >60 minutes [5/13 (38.5%)]. Overall, 11/64 (17.2%) developed sequelae. In the >60-minute group, sequelae included hemiparesis, moderate-to-severe intellectual disability, and moderate-to-severe delay in motor milestones, whereas sequelae in the 10-30-minute group were generally milder.

Mortality in pediatric SE varies according to the study population and underlying etiology (29). Overall mortality in our cohort was 8/64 (12.5%). No deaths occurred among the 30 patients with seizure duration of 10-30 minutes, whereas 8 of 34 patients (23.5%) with seizure duration >30 minutes died. Thus, all deaths in this cohort occurred in patients whose seizures lasted longer than 30 minutes. Nevertheless, mortality cannot be attributed to seizure duration alone; underlying etiology and disease severity are also important determinants of outcome.

Taken together, our findings support the importance of early recognition and rapid treatment of pediatric SE. The greater burden of sequelae and mortality among patients with seizures lasting longer than 30 minutes underscores the clinical importance of terminating seizure activity as early as possible, while recognizing that outcome is also influenced by etiology, disease severity, and treatment response. Timely pre-hospital benzodiazepine administration and prompt progression through a staged treatment algorithm remain key components of pediatric SE management.

Conclusion

SE is a major pediatric neurological emergency requiring rapid and effective intervention. One of the key elements of successful treatment is early administration of appropriate therapy in the pre-hospital setting. In accordance with the ILAE operational time points, treatment should be initiated without delay when t1 is reached, with the aim of preventing prolonged seizure activity and potential long-term neurological consequences associated with t2. Education of healthcare providers and caregivers regarding the timely and correct use of first-line rescue treatments is therefore an important component of pediatric SE management.

Acknowledgment

The authors gratefully acknowledge the contribution of the Department of Pediatric Neurology, Erciyes University, Faculty of Medicine, to the execution of this study.

Ethics Statement

Written informed consent was obtained from all participating children and their parents/legal guardians. Ethical approval for the study was granted by the

Erciyes University Ethics Committee (Approval date: 22 December 2024, Reference number: 2017/605).

Authors' Contributions

Hamit Acer and Mehmet Canpolat performed the statistical analysis. Hamit Acer, Hüseyin Per, Hakan Gumus, Basak Nur Akyildiz, and Sefer Kumandas contributed to data collection. Mehmet Canpolat, Hüseyin Per, and Hakan Gumus performed the literature review. Hamit Acer, Mehmet Canpolat, Hüseyin Per, Hakan Gumus, Basak Nur Akyildiz, and Sefer Kumandas contributed to the conception and development of the manuscript. All authors critically reviewed the final version of the manuscript and approved it for publication

Conflict of Interest

The authors declared no competing financial or non-financial interests.

References

1. Gastaut H. Clinical and electroencephalographical classification of epileptic seizures. Epilepsia 1970;11:102–13.
2. Guidelines for epidemiologic studies on epilepsy. Commissionon epidemiology and prognosis, international league against epilepsy. Epilepsia 1993;34(4):592-6. DOI: 10.1111/j.1528-1157.1993.tb00433.x
3. Betjemann JP, Lowenstein DH. Status epilepticus in adults. Lancet Neurol 2015;14(6):615–24.
4. Swaiman KF, Aswal S, Ferriero DM, Schor NF, Finkel RS, Gropman Al, Pearl Pl, Shevell MI, editors. Swaiman's Pediatric Neurology: Principles and Practice. 6th ed. UK/USA: Elsevier Saunders; 2017. p.543-51.
5. Trinka E, Cock H, Hesdorffer D, Rossetti AO, Scheffer IE, Shinnar S, et al. A definition and classification of status epilepticus: Report of the ILAE task force on classification of status epilepticus. Epilepsia 2015;56(10):1515–23.
6. Alford EL, Wheless JW, Phelps SJ. Treatment of generalized convulsive status epilepticus in pediatric patients. J Pediatr Pharmacol Ther 2015;20:260–89.
7. Haspolat S, Gokcay E, Sonmez M, Topaloğlu H, Tekgul H, Gurer Y. Turkish Child Neurology Association board of directors and editorial board. Child Neurology. Ankara: Anıl Group Printing; 2010. p.327-32.
8. Seinfeld S, Leszczyszyn DJ, Pellock JM. Status epilepticus and acute seizure. In: Pellock JM, Nordli DR, Sankar R, Wheless JW, editors. Pediatric Epilepsy. 4th ed. New York: Demos Medical Publishing; 2017. p.567-83.
9. Canpolat M, Kumandas S. Status epilepticus management in children. Turkiye Klinikleri J Pediatr Sci 2018;14(1):58-81.
10. Eriksson K, Metsaranta P, Mervaala M. Treatment delay and the risk of prolonged status epilepticus. Neurology 2005;65:1316–8.
11. Eriksson KJ, Koivikko MJ. Status epilepticus in children: Etiology, treatment, and outcome. Dev Med Child Neurol 1997;39:652–8.
12. Smith DM, McGinnis EL, Walleigh DJ, Abend NS. Management of status epilepticus in children. J Clin Med 2016;5(4):47.
13. Gaillard WD, Chiron C, Cross JH, Harvey AS, Kuzniecky R, Hertz-Pannier L, et al; ILAE Committee for Neuroimaging, Subcommittee for Pediatric. Guidelines for imaging infants and children with recent-onset epilepsy. Epilepsia 2009;50(9):2147-53.
14. Carman KB. Febrile status epilepticus. Turkiye Klinikleri J Neurol-Special Topics 2016;9(3):90-4.
15. Shinnar S, Seinfeld S. Febrile seizures. In: Swaiman KF, Aswal S, Ferriero DM, Schor NF, et al, editors. Swaiman's Pediatric Neurology: Principles and Practice. 6th ed. UK/USA: Elsevier Saunders; 2017.
16. Spatola M, Novy J, Du Pasquier R, Dalmau J, Rossetti AO. Status epilepticus of inflammatory etiology: A cohort study. Neurology 2015;85:464–70.
17. Smith DM, McGinnis EL, Abend NS, et al. Management of status epilepticus in children. J Clin Med 2016;5(4):47. doi:10.3390/jcm5040047.
18. 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;16:48–61.
19. Lewena S, Pennington V, Acworth J, Thornton S, Ngo P, McIntyre S, et al. Emergency management of pediatric convulsive status epilepticus: A multicenter study of 542 patients. Pediatr Emerg Care 2009;25:83–7.
20. Brophy GM, Bell R, Claassen J, Alldredge B, Bleck TP, Glauser T, et al. Guidelines for the evaluation and management of status epilepticus. Neurocrit Care 2012;17:3–23.
21. Yasiry Z, Shorvon SD. The relative effectiveness of five antiepileptic drugs in treatment of benzodiazepine-resistant convulsive status epilepticus: A meta-analysis of published studies. Seizure 2014;23:167–74.
22. Lewena S, Young S. When benzodiazepines fail: How effective is second line therapy for status epilepticus in children? Emerg Med Australas 2006;18:45–50.
23. Lowenstein DH. Status epilepticus: An overview of the clinical problem. Epilepsia 1999;40(Suppl 1):S3–8.
24. Ferlisi M, Hocker S, Grade M, Trinka E, Shorvon S; International Steering Committee of the StEp Audit. Preliminary results of the global audit of treatment of refractory status epilepticus. Epilepsy Behav 2015;49:318–24.
25. Kravljanac R, Djuric M, Jankovic B, Pekmezovic T. Etiology, clinical course and response to the treatment of status epilepticus in children: A 16-year single-center experience based on 602 episodes of status epilepticus. Eur J Paediatr Neurol 2015;19:584-90.
26. Ilvento L, Rosati A, Marini C, L'Erario M, Mirabile L, Guerrini R. Ketamine in refractory convulsive status epilepticus

in children avoids endotracheal intubation. Epilepsy Behav 2015;49:343–6.

27. Rosati A, L'Erario M, Ilvento L, Cecchi C, Pisano T, Mirabile L, Guerrini R. Efficacy and safety of ketamine in refractory status epilepticus in children. Neurology 2012;79:2355–8.

28. Akyıldız BN, Kumandas S. Treatment of pediatric refractory status epilepticus with topiramate. Childs Nerv Syst 2011;27:1425-30.

29. Ostrowsky K, Arzimanoglou A. Outcome and prognosis of status epilepticus in children. Semin Pediatr Neurol 2010;17:195-200.