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ORIGINAL ARTICLE

Electrophysiological and clinical markers of SUDEP risk in pediatric epilepsy

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Abstract

Objective: Sudden unexpected death in epilepsy (SUDEP) is a major cause of epilepsy-related mortality, especially in patients with drug-resistant epilepsy. However, pediatric data remain limited, and specific risk assessment tools for children are lacking. This study evaluates the association between peri-ictal prone positioning, postictal generalized EEG suppression (PGES), and SUDEP-7 Inventory scores in pediatric patients with epilepsy.

Methods: A retrospective review was conducted on 273 generalized convulsive seizures (GCS) from 117 pediatric patients who underwent video-EEG monitoring from 2002 to 2022. We assessed body position during seizures, PGES presence and duration, and SUDEP-7 Inventory scores. Statistical comparisons were made across PGES-positive and -negative, and prone vs. non-prone subgroups.

Results: Prone positioning occurred in 5.86% of seizures, with 91.6% of prone-onset seizures remaining in the prone position throughout. PGES was present in 50.4% of patients (mean duration: 120.5 s). SUDEP-7 scores were significantly higher in PGES-positive patients (mean: 7.04) compared to PGES-negative (mean: 6.36; $p = 0.031$). Daytime GCS occurred significantly earlier after sleep onset than night time GCS (53.3 vs. 131.4 minutes; $p = 0.001$).

Significance: PGES appears to be a reliable marker associated with higher SUDEP-7 scores and may indicate increased SUDEP risk in children. While SUDEP-7 has limitations in pediatric use, it remains the most accessible clinical tool. Integrating EEG and body position metrics could enhance individualized monitoring for pediatric patients at risk.

KEYWORDS

pediatric epilepsy, PGES, prone position, risk assessment, SUDEP, SUDEP-7 inventory

1 | INTRODUCTION

Sudden unexpected death in epilepsy (SUDEP) is a leading cause of epilepsy-related mortality, accounting for up to 17% of deaths in all epilepsy patients and as much as 50% in those with drug-resistant epilepsy.[1,2] Despite growing awareness, SUDEP remains unpredictable due to the multifactorial and poorly understood mechanisms involved.[3–5]

Among proposed mechanisms, the MORTEMUS study highlighted the role of cardiorespiratory dysfunction as a consequence of cerebral disturbances following

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generalized convulsive seizures (GCS). Notably, terminal apnea was found to precede terminal asystole in observed SUDEP cases, emphasizing the importance of autonomic and respiratory collapse.[6–8] This underscores the relevance of identifying physiological biomarkers that may signal impending collapse, such as postictal EEG suppression.

Several clinical risk factors for SUDEP have been identified, including frequent GCS, nocturnal seizures, and prone positioning during the peri-ictal period.[3] However, evidence-based recommendations for preventive strategies, particularly for pediatric patients, are lacking. Prone positioning, also implicated in sudden infant death syndrome (SIDS), is recognized as a modifiable SUDEP risk factor. Despite its importance, no formal guidelines exist on sleep positioning in children with epilepsy, making this an area of concern. Despite its low prevalence in monitored seizures, the strong association between prone positioning and PGES, as well as SUDEP, warrants further scrutiny in pediatric cohorts.

Peri-ictal biomarkers such as prolonged postictal generalized EEG suppression (PGES) have been explored as physiological indicators of SUDEP risk. PGES is thought to reflect impaired seizure termination and may indicate prolonged postictal suppression of brainstem centers controlling respiration and circulation.[9–11] While prolonged PGES has shown associations with elevated SUDEP risk, optimal duration thresholds remain unclear. Although some studies suggest using thresholds of 20 or 80 s, a ≥10-s cutoff has been widely applied in pediatric cohorts and remains a sensitive measure for capturing early suppression dynamics. Additional physiological markers including peri-ictal cerebral oxygenation and heart rate variability (HRV) have also been investigated, although their roles remain exploratory.[12–15]

To quantify clinical SUDEP risk, the SUDEP-7 Inventory was introduced, incorporating seizure frequency, antiseizure medication burden, seizure-related injuries, and other factors.[4,5] While widely used, its practicality and predictive accuracy, particularly in pediatric populations, are debated. Our use of SUDEP-7 does not imply it as a gold standard but rather reflects the lack of validated, age-specific alternatives. In the absence of pediatric-specific tools and given the lack of observed SUDEP events in our cohort, we used the SUDEP-7 Inventory to evaluate risk stratification.

This study aims to assess key clinical and electrophysiological variables associated with SUDEP risk in children with epilepsy. We focused on GCS timing, peri-ictal body position, PGES presence and duration, epilepsy duration, and SUDEP-7 scores in a video-EEG monitored cohort. By integrating these data, we hope to improve pediatric SUDEP risk stratification and inform targeted prevention strategies.

Key points

- PGES was observed in 50.4% of pediatric epilepsy patients undergoing video-EEG monitoring.
- PGES-positive patients had significantly higher SUDEP-7 scores than PGES-negative patients.
- Prone positioning was rare but strongly associated with PGES during generalized seizures.
- Generalized seizures occurred earlier during daytime sleep than nighttime sleep.
- Epilepsy duration >36 months was linked to significantly higher SUDEP-7 scores.

2 | METHODS

2.1 | Study design and setting

We performed a retrospective review of medical and EEG records from pediatric patients who underwent video-EEG (v-EEG) monitoring at a quaternary epilepsy center in Ankara, Türkiye, between January 1, 2002, and January 1, 2022. All patients were evaluated by pediatric epileptologists, with classification of seizures and epilepsy syndromes based on the 2017 ILAE criteria.[16–18] The study followed STROBE guidelines and received ethics approval from the local review board.[19] Inclusion criteria were a diagnosis of drug-resistant epilepsy and at least one documented GCS during v-EEG monitoring.

2.2 | Data collection

Standardized forms were used to extract clinical data, including epilepsy classification, seizure semiology, and seizure frequency. We recorded peri-ictal body positions, antiseizure medication (ASM) history, and time to medical staff intervention and oxygen administration. SUDEP-7 Inventory scores were calculated for each patient. Additional variables included number and timing of GCS during sleep and wakefulness, and body positions at seizure onset and termination.

Seizure generalization onset was defined as the brief transition from focal activity to GCS, typically marked by versive movements or vocalization, in accordance with Theodore et al.[20] Staff were instructed not to interfere with body position during the seizure but to place the patient in a lateral position during recovery. Peri-ictal positions (within 2 min before and after seizure onset) were categorized as supine, prone, lateral, sitting, or standing following Wang et al.[21]

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2.3 | Video-EEG monitoring and PGES evaluation

All patients underwent 32-channel long-term v-EEG using the international 10–20 system. Seizure semiology and EEG patterns, including ictal onset and postictal findings, were independently reviewed by two board-certified epileptologists (E.A., T.H.).

PGES was defined as generalized EEG suppression (<10 μV) occurring within 30 s of seizure offset. Interobserver reliability for PGES detection was high (Krippendorff's $\alpha = .95$), with no significant differences in PGES duration estimates ($p = .085$).

2.4 | SUDEP-7 inventory

Each patient was assigned a SUDEP-7 Inventory score based on seven established risk factors, excluding epilepsy duration >30 years due to age limitations.[22–24] The components included frequency of generalized tonic–clonic seizures, nocturnal seizure occurrence, seizure-related injuries, polytherapy, history of injury, and vagus nerve stimulator use.

Despite its use in risk assessment, the SUDEP-7 Inventory may not fully capture pediatric-specific risk factors such as autonomic instability, genetic vulnerability, or supervision during sleep.[4,5,8,10,11] In this study, we applied the tool to enable standardized risk comparison across subgroups, including prone vs. non-prone seizures, PGES-positive vs. PGES-negative patients, and sex differences.

2.5 | Statistical analysis

Statistical analyses were conducted using SPSS version 26 (IBM Corp., Armonk, NY). Continuous variables were compared using the Student *t*-test or Wilcoxon Mann–Whitney test as appropriate. Categorical variables were assessed using chi-squared or Fisher's exact tests.

Visualization of seizure-related body position changes was performed using Sankey plots generated in R (R Foundation for Statistical Computing, Vienna, Austria) with the ggalluvial extension for ggplot2. A *p*-value <.05 was considered statistically significant.

3 | RESULTS

3.1 | Demographic characteristics

Of the 362 pediatric epilepsy patients evaluated in the video-EEG monitoring (VEM) unit, 117 (32.3%) had at least one documented generalized convulsive seizure (GCS), yielding 273 analyzable seizures. The average number of GCS per patient was 2.31 (range: 1–21). Nearly half (47.8%) experienced a single GCS, while 18.8% had four or more. The cohort was predominantly male (58.9%), with a mean age of 162 months (range: 7–228 months) and an average epilepsy duration of 80.9 ± 55.8 months. Patients were on an average of 2.3 antiseizure medications (ASMs), having failed a mean of 1.87 prior ASMs. Most patients (90.5%) had focal epilepsy, with 9.5% having primary generalized epilepsy (Table 1).

3.2 | Body position data

Among the 273 GCS events, initial seizure positions were as follows: supine (42.1%), right lateral (27.5%), left lateral (20.5%), sitting (6.9%), and prone (2.9%). Peri-ictal prone positioning occurred in 16 seizures (5.86%). In this subgroup, 11 of 12 prone-onset seizures remained prone throughout the event. Notably, only 1.14% of non-prone-onset seizures ended in the prone position, whereas 91.66% of prone-onset seizures concluded in the same position (Figure 1).

The prone-positive group ($n = 14$ seizures) consisted of 3 females and 11 males, with a mean age of 90.85 months and a SUDEP-7 score of 6.68. The prone-negative group had a mean age of 76.36 months and a SUDEP-7 score of 5.85 (Table 2), though this difference was not statistically significant ($p = .254$). There was no difference in initial sleep posture between seizure and seizure-free nights ($p = .243$) (Table 3). Body position changed during 45.4% of GCS events. At seizure termination, most patients were supine (57.1%), followed by right lateral (20.8%), left lateral (16.1%), and prone (5.1%).

No significant differences were identified between prone and non-prone groups regarding seizure duration, PGES presence or duration, or epilepsy exposure (Table 4, Figure 2).

3.3 | Electrophysiological data

PGES was observed in 69 of 117 patients (50.4%). Of these, 6 had primary generalized epilepsy, while 60 experienced secondarily generalized seizures. PGES durations ranged from 30 s to 35 min (mean: 122.2 ± 50.7 s). Among prone-positive patients, 93.75% of seizures were associated with PGES, with a mean duration of 108.5 s. However, PGES durations did not significantly differ between prone and non-prone groups ($p = .372$).

PGES-positive patients had significantly higher SUDEP-7 scores than PGES-negative patients (mean: 7.04

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TABLE 1 Summary of the patients' demographic data.

Patients	$n = 117$ (%)
Male gender	69 (59%)
Intellectual disability presence	62 (52.9%)
Age (months)[a]	162 ± 43 (range 7–228 months)
Age at epilepsy onset (months)[a]	74.1 ± 56.4 (range 1 day–192 months)
Epilepsy duration (months)[a]	80.9 ± 55.8 (range 1 month–210 months)
Epilepsy syndrome	**% (*n*)**
Focal epilepsy	106 (90.5%)
Idiopathic (genetic) generalized epilepsy	11 (9.5%)
Drug-refractory epilepsy	89 (76%)
V-EEG duration (day)[a]	4.47 ± 1.22 (range 1–10 day)
Seizure frequency	7.07 ± 8.64 (range 1–45)
GCS frequency	2.34 ± 2.4 (1–21)
Overall seizure frequency prior to admission	
At least one seizure per day	33 (28.2%)
At least once a week	42 (35.8%)
At least once a month	34 (29%)
Less than once a month or unknown	8 (6.8%)
Antiseizure drugs (mean)	2.37
Failed antiseizure drugs in the past (mean)	1.87
Current antiseizure drugs	***n***
Clobazam	26
Levetiracetam	59
Oxcarbazepine	29
Lacosamide	2
Valproate	53
Phenobarbital	18
Carbamazepine	38
Topiramate	41

[a]Mean ± standard deviation.

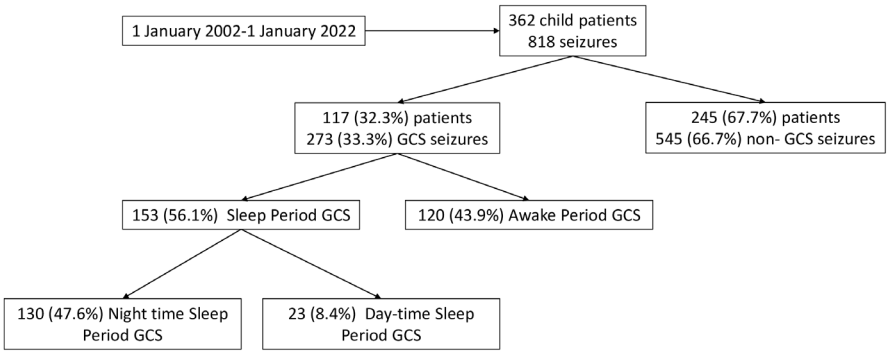


FIGURE 1 Sankey diagram of seizure body positions. Sankey diagram illustrating the transition of body positions during 273 generalized convulsive seizures. Initial positions included supine, lateral (right/left), sitting, and prone. The diagram highlights that prone-onset seizures ($n = 12$) predominantly remained in the prone position throughout the ictal phase, while other positions showed greater variability. These findings underscore the stability and clinical relevance of prone positioning as a potential SUDEP risk factor.

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TABLE 2 The comparison of patients, prone positive versus negative.

	Prone positive patients ($n=14$, %)	Prone negative patients ($n=113$)	p
Gender (Female)	3 (21.4%)	45 (39.8%)	**.012**
Age (months) (mean ± SDS)	90.85 (±4.83)	76.36 (±6.67)	.075
SUDEP 7 inventory score (mean ± SDS)	5.85 (±2.19)	6.68 (±1.80)	**.021**
Epilepsy exposure duration (min) (mean ± SDS)	103.3 (±59.2) m	86.45 (±56.1) m	**.003**
PGES	10 (14.4%)	59 (85.6%)	.142
PGES duration (s)	85 (±158)	126 (±428)	.372

Note: Bold values indicate significance p values.

vs. 6.36; $p=.031$) and a greater total number of seizures (mean: 7.26 vs. 6.68; $p=.02$), suggesting an association between PGES and elevated SUDEP risk (Table 4).

3.4 | Timing data

The mean latency to GCS onset after sleep initiation was 131.2 ± 120.6 min. GCS occurred significantly earlier during daytime sleep (mean: 53.3 ± 41.4 min) compared to nighttime sleep (mean: 131.4 ± 119.0 min; $p=.001$) (Table 5).

3.5 | SUDEP-7 inventory data

SUDEP-7 Inventory scores ranged from 4 to 9, with a mean of 6.64 ± 1.81. No significant differences were found between sexes ($p=.377$) or between prone and non-prone groups ($p=.609$). Patients with epilepsy durations exceeding 36 months had significantly higher SUDEP-7 scores than those with shorter durations (6.97 ± 1.80 vs. 5.70 ± 1.48; $p=.001$).

4 | DISCUSSION

SUDEP remains a major concern in pediatric epilepsy care, particularly among patients with drug-resistant seizures. While prone positioning has been implicated as a modifiable risk factor, its significance in pediatric peri-ictal dynamics has not been well established.[8,24] Our study adds to the limited literature by analyzing the associations between prone positioning, PGES, and SUDEP-7 Inventory scores in children.

Although the SUDEP-7 Inventory is frequently used in clinical settings, its applicability in pediatric populations is debatable.[8,24–26] Due to the lack of a validated, pediatric-specific alternative and the absence of SUDEP cases in our cohort, we employed this tool for standardized risk assessment. Our use of SUDEP-7 does not imply it as a gold standard but reflects the lack of validated age-specific alternatives. Notably, SUDEP-7 scores were significantly higher in PGES-positive patients (mean: 7.04 vs. 6.36, $p=.031$), suggesting that PGES may reflect increased SUDEP susceptibility.[8,11] However, this inventory does not account for critical pediatric-specific variables such as autonomic dysfunction, genetic predisposition, or nocturnal supervision, limiting its standalone predictive value.

In adults, SUDEP-7 Inventory has shown moderate predictive value in retrospective cohorts, particularly in those with frequent GTCS and medication non-adherence. However, its ability to predict individual SUDEP risk remains limited.[10,22] Our findings in children must therefore be interpreted within this contextual limitation.

Our findings support the role of PGES as a physiologic marker in SUDEP risk assessment. Patients exhibiting PGES had significantly higher SUDEP-7 scores, consistent with studies showing PGES as a surrogate for prolonged electroclinical disturbance during seizure termination.[25,26] Importantly, PGES was present in nearly all prone-positioned seizures, underscoring a potential compounding effect.[8,24]

While the pathophysiology of PGES in SUDEP is still debated, evidence suggests that postictal cerebral suppression may contribute to respiratory arrest or impaired arousal responses.[21,22] Lhatoo et al.[4] reported that PGES durations exceeding 80 s quadrupled SUDEP risk. Although our study used a ≥30-s threshold, consistent with prior pediatric research, recent work supports the need to consider longer thresholds (e.g., 20 or 80 s) in future risk models.[4,10,22]

Although peri-ictal prone positioning was rare (5.86%), nearly all prone-onset seizures remained in that position throughout the ictal phase. This finding is consistent with earlier reports suggesting that, without intervention, patients often remain prone at seizure onset, especially during sleep.[27] These dynamics, also observed in the MORTEMUS study, are concerning given the

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TABLE 3 In this table, the seizure onset, before/ after version, and end-of-seizure positions of all seizures in which the prone position was observed were monitored one by one.

Seizure	Patient	Gender	Age (months)	Seizure onset position	Before version position	After version position	End GCS	PGES	PGES duration (s)	EEG	SG	MR	SUDEP 7 Inventory scale
1	1	M	102	P	P	RL	RL	+	30	Left frontotemporal	+	Normal	9
2	2	M	172	LL	P	S	S	−	0	Right temporal	+	Right Temporal FCD	7
3	3	M	120	P	P	P	P	+	35	Right frontal	+	Normal	4
4	4	M	216	P	P	P	P	+	120	Right frontotemporal	+	Normal	4
5	5	F	180	P	P	P	P	+	40	Right frontal	+	Right Frontal DNET	5
6	6	M	192	RL	RL	P	P	+	360	Left temporal	+	Left Temporal Encephalomalasia	8
7	7	M	168	S	S	RL	P	+	30	Left temporal	+	Left Mesial Temporal Sclerosis	4
8		M	168	S	S	P	P	+	25	Right frontotemporal	+	Right Mesial Temporal Sclerosis	
9	8	M	188	P	P	P	P	+	90	Right temporal	+	Right Temporal Atrophy	6
10		M	188	P	P	P	P	+	60	Right temporal	+	Right Temporal Atrophy	
11	9	M	178	P	P	P	P	+	120	Left temporal	+	Left Hippocampal Sclerosis	8
12	10	M	108	P	P	RL	P	+	40	Right frontocentral	+	Right Frontal FCD	6
13	11	M	132	P	P	RL	RL	+	300	Left frontotemporal	+	Normal	4
14	12	F	156	LL	P	S	S	+	60	Left frontotemporal	+	Normal	4
15	13	F	36	P	P	P	P	+	45	Left frontocentral	+	Left Frontocentral FCD	9
16	14	M	192	P	P	P	P	+	30	Left posterior temporal	+	Normal	4

Abbreviations: EEG, electroencephalography; F, female; FCD, focal cortical dysplasia; GCS, generalized covulsive seizures; LL, left lateral; M, male; MDNET, dysembryoplastic neuroepithelial tumor; P, prone; PGES, postictal generalized electroencephalograpic suppression; RL, right lateral; S, supin; SG, seconder generalization.

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TABLE 4 The comparison of prone position with respect to GCS and PGES presence.

GCS onset, $n=273$	GCS ending, Prone/non-prone, $n=14/n=259$	Patients (%)	p	PGES positivity
Prone $n=11$	Prone	11 (4.0%)	Kappa: .904 $p<.001$	11 (100%)
	Non-prone	1 (.4%)		
Non-Prone $n=262$	Prone	3 (1.1%)	Kappa: .486 $p<.001$	1 (33.3%)
	Non-prone	258 (98.85%)		

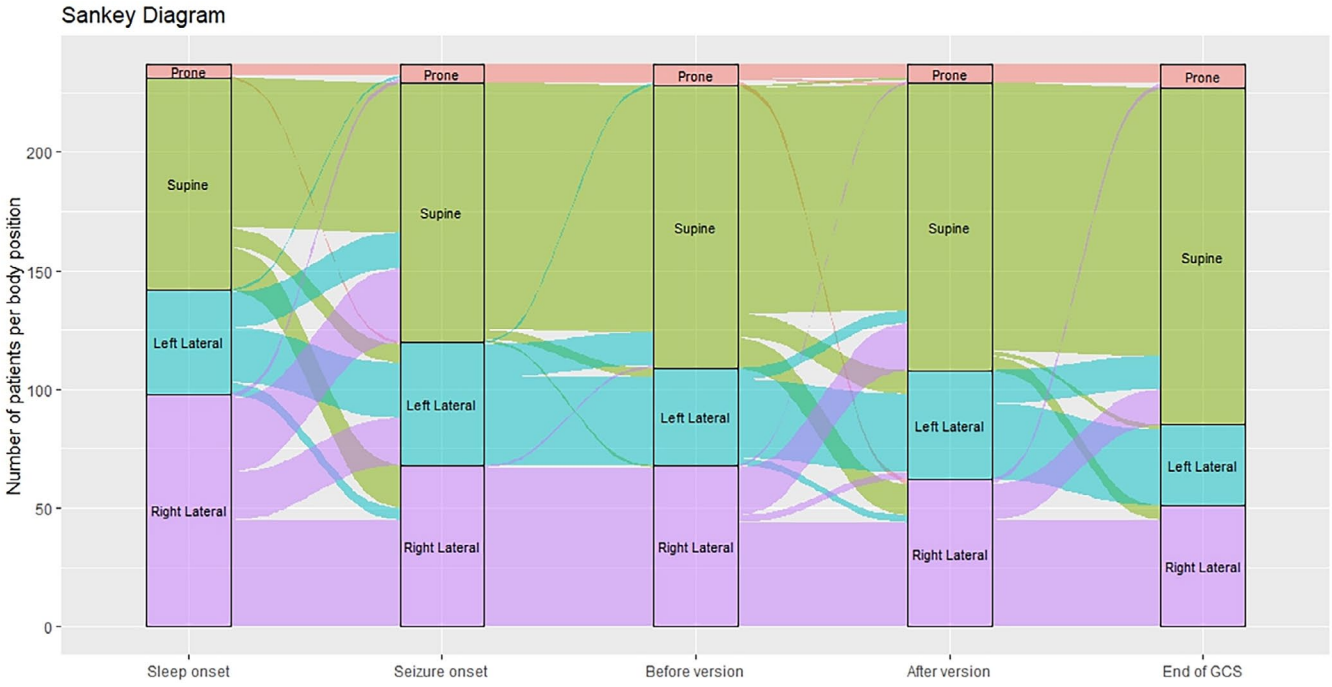


FIGURE 2 Positional change patterns in prone vs. non-prone seizures. Visualization of body position changes during GCS among patients who started in a prone position compared to those who did not. Notably, 91.6% of prone-onset seizures concluded in the same position, while only 1.14% of non-prone-onset seizures transitioned into prone. The diagram emphasizes the importance of initial sleep posture and its implications for SUDEP risk.

TABLE 5 GCS's sleep (day and night time sleep) and wakefulness timing data.

	$N=273$ (%)	GCS timing after sleep (min)	p
Sleep GCS	153 (56.1%)	131.15 (SDS: 120.65)	
Daytime sleep GCS	23 (8.4%)	53.27 (SDS: 41.37)	$p=.001$
Nighttime sleep GCS	130 (47.6%)	131.37 (SDS: 118.95)	
Wakefulness GCS	120 (43.9%)	—	

association between prone position and impaired postictal recovery.[8–10]

Our subgroup findings resonate with previous literature, suggesting that prone positioning is more frequent in younger individuals.[11] Although our sample did not include SUDEP cases, the implications of this finding in younger patients emphasize the need for age-adapted preventive strategies.[10,21] Additionally, our data revealed that GCS occurred earlier during daytime sleep than nighttime, suggesting time-specific windows of heightened vulnerability. These results may help inform optimal monitoring schedules or timing for supervision and device-based seizure detection.

Our findings provide important insights into SUDEP prevention and patient monitoring by identifying preferred or avoided sleeping postures for pediatric patients at risk of SUDEP and the times of day when seizures are more likely to occur. Tonic–clonic seizures, nocturnal seizures, past injuries, and a lack of evening supervision are significant criteria for recommending seizure-monitoring devices.[23] However, few studies have addressed how frequently or when such devices should be used.[23,27] Our

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findings suggest that early sleep periods, especially during daytime naps, may represent windows of increased vulnerability that could guide optimized supervision or alert system calibration.

Importantly, we observed that epilepsy duration >36 months was significantly associated with higher SUDEP-7 scores ($p = .001$). This clinical variable, often overlooked, may represent a simple and low-cost surrogate marker of cumulative risk. Unlike PGES or EEG-based indices, epilepsy duration is accessible and easily recorded in clinical settings. Given this robust association patients with epilepsy persisting beyond 36 months may benefit from closer longitudinal monitoring. Future pediatric studies should evaluate its longitudinal predictive power alongside physiological biomarkers.

Taken together, our findings support a multifactorial model of SUDEP risk involving physiological (PGES), clinical (epilepsy duration), and behavioral (prone positioning) elements. Monitoring strategies for high-risk pediatric patients should incorporate prone positioning, PGES presence, and seizure timing. Interventions such as nocturnal supervision, sleep posture guidance, or seizure detection devices may mitigate risk in susceptible individuals. We suggest that patients with epilepsy duration >36 months and PGES-positive seizures should be prioritized for device-based surveillance, especially during the first 2 hours after sleep onset.

Our study has limitations. First, the absence of SUDEP cases limits definitive conclusions about predictive validity. Second, as a single-center retrospective study, generalizability is constrained. Finally, the study did not systematically evaluate environmental or genetic risk factors. Larger, multicenter cohorts with longitudinal follow-up are needed to validate and expand upon our findings.

5 | CONCLUSION

Postictal generalized EEG suppression (PGES) was significantly associated with elevated SUDEP-7 scores, supporting its role as a physiological marker of SUDEP risk in pediatric epilepsy. Although infrequent, peri-ictal prone positioning strongly co-occurred with PGES and may contribute to cumulative risk. Epilepsy duration longer than 36 months also emerged as a simple, yet robust clinical marker linked to higher risk scores.

Our results suggest that children with prolonged epilepsy, PGES-positive seizures, or prone positioning may benefit from enhanced nocturnal monitoring, sleep posture guidance, and seizure detection technologies. The earlier occurrence of generalized convulsive seizures during daytime sleep highlights potential time windows for targeted supervision.

While the SUDEP-7 Inventory has limitations in pediatric populations, its integration with EEG, positional, and temporal markers may improve individualized risk stratification. We recommend prioritizing patients meeting multiple risk criteria for intensified follow-up. Future prospective studies are warranted to validate this multidimensional approach and develop age-appropriate SUDEP prevention strategies.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest related to this study.

DATA AVAILABILITY STATEMENT

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

INFORMED CONSENT

Written informed consent was obtained from the legal guardians of all participants.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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Test yourself

1. According to the study findings, which of the following was most significantly associated with higher SUDEP-7 Inventory scores in pediatric epilepsy patients?
   A. Seizures occurring during nighttime sleep
   B. Epilepsy duration shorter than 12 months
   C. Presence of postictal generalized EEG suppression (PGES)
   D. Seizures with focal onset without generalization
2. What did the study reveal about the relationship between seizure timing and sleep phase in pediatric patients?
   A. Seizures occurred later during daytime sleep than night time sleep
   B. Daytime seizures occurred significantly earlier after sleep onset than night time seizures
   C. No difference in seizure timing between day and night
   D. Seizures were exclusively nocturnal
3. Which clinical factor was identified as a simple, accessible marker associated with higher SUDEP-7 scores in children with epilepsy?
   A. Family history of epilepsy
   B. Use of vagus nerve stimulation
   C. Epilepsy duration longer than 36 months
   D. Number of antiseizure medications prescribed

Answers may be found in the supporting information.

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