Brief Communication

Sleep-Related Breathing Disorders in Children with Epilepsy: Prevalence and Risk Factors

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Abstract

Sleep-related breathing disorders (SRBDs) in children with epilepsy (CWE) are often under-recognized despite their potential impact on seizure control and quality of life. This study aimed to determine the prevalence of SRBDs in CWE using the Pediatric Sleep Questionnaire (PSQ) and to identify clinical factors associated with SRBDs. This observational study included 238 CWE attending the pediatric epilepsy clinic of a tertiary care hospital in Mumbai, India, between June 2020 and May 2021. The PSQ was administered to all participants, with a score >8/22 suggesting SRBDs. Categorical variables were compared using the Chi-square test. SRBDs were present in 44.5% of CWE according to the PSQ. Age, gender, age at epilepsy onset, family history of seizures, seizure type, body mass index, and neck-circumference-to-height ratio did not differ significantly between children with and without SRBDs. SRBDs were more frequent in children on multiple anti-seizure medications (ASMs), those with higher seizure burden, and those with drug-resistant epilepsy. It was present in 36.7% of children on <2 ASMs versus 50.7% on ≥2 ASMs ($P = 0.004$). SRBDs affected 31.1% of drug-responsive children compared with 58.0% of those with drug-resistant epilepsy ($P = 0.0001$). SRBDs are a common comorbidity, affecting nearly half of CWE. Routine screening for SRBDs in CWE is recommended, especially if drug refractory, followed by confirmation with polysomnography. Early detection of SRBDs offers a modifiable target that can enhance seizure control and neurobehavioral outcomes in CWE.

Keywords: Epilepsy, pediatric sleep questionnaire, sleep-related breathing disorder

Introduction

Epilepsy is a chronic brain disorder marked by a persistent tendency to generate seizures, accompanied by neurobiological, cognitive, psychological, and social consequences. Drug-refractory epilepsy (DRE) refers to the failure to achieve seizure control despite the appropriate use of at least two well-chosen and adequately dosed anti-seizure medications (ASM). The relationship between epilepsy and sleep is bidirectional: Seizures can disrupt sleep architecture, and sleep disturbances can lower the seizure threshold.[1] Children with epilepsy (CWE) frequently exhibit alterations in total sleep time, sleep architecture, sleep latency, arousal patterns, and daytime alertness.[2] Sleep disturbances are increasingly recognized as a modifiable risk factor for sudden unexpected death in epilepsy, highlighting the importance of optimizing sleep in CWE.

Sleep-related breathing disorders (SRBDs) are common in CWE but often go under-recognized. These include obstructive sleep apnea (OSA) syndrome, upper airway resistance syndrome, and obstructive hypopnea syndrome.[3] The etiology is commonly due to upper airway obstruction, most often caused by adenotonsillar hypertrophy, along with contributing factors such as nasal congestion, allergic rhinitis, obesity, and craniofacial abnormalities.[4] Neuromuscular and genetic conditions that reduce airway tone, as well as prematurity and airway malacia, further increase vulnerability. Environmental factors like tobacco smoke and sleep-related reductions in airway tone can worsen obstruction, collectively leading to SRBDs.[4] The reported prevalence of SRBDs in CWE varies widely due to differences in diagnostic tools, clinical settings, and population characteristics.

Sleep disturbances can significantly impair neuropsychological development, memory, and cognition, particularly in CWE. Disrupted sleep architecture interferes with synaptic homeostasis and neuroplasticity, while frequent nocturnal seizures interrupt critical non-rapid eye movement and rapid eye movement (REM) stages required for memory consolidation and learning. Sleep deprivation also worsens attention, executive function, information processing speed, and emotional regulation, further contributing to academic and behavioral difficulties. Excessive daytime sleepiness reduces alertness and participation in school and therapy, compounding

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these challenges. Collectively, these mechanisms highlight how disturbed sleep can markedly worsen neurocognitive outcomes in CWE.[5] Sleep deprivation and disturbed sleep architecture can lower the seizure threshold, contributing to seizure exacerbation.[6] This study aimed to determine the prevalence of SRBDs in CWE using the Pediatric Sleep Questionnaire (PSQ) and to identify associated clinical factors.

Methods

This observational study was conducted between June 2020 and May 2021 at the pediatric epilepsy clinic of a tertiary care hospital in Mumbai, India. Approval was obtained from the Institutional Ethics Committee (Letter No: IRB/1342/AL/20/39). Written informed consent was obtained from parents, and assent from children aged ≥7 years, wherever applicable. Children aged 2–12 years with epilepsy, as defined by the International League Against Epilepsy, were included.[7]

Children were excluded if they had neuromuscular disorders, genetic syndromes, or cerebral palsy; were receiving hypnotics or sedatives; had recently discontinued ASMs; or had comorbid conditions such as narcolepsy, asthma, chronic respiratory disease, or other primary sleep disorders requiring medical intervention.

The sample size was calculated using the formula: $n = (Z\alpha)^2 \times p\ (1 - p)/d^2$, where $Z\alpha = 1.96$ (5% type I error), P = expected prevalence, and d = absolute precision.[8] Based on a study by Blumer et al.,[9] which reported a 10.6% prevalence of SRBDs, the calculated sample size was: $n = (1.96)^2 \times 10.6 \times 89.4/5^2 = 145$. A total of 238 children were enrolled to enhance reliability and account for potential dropouts. After enrollment, detailed clinical and seizure history and relevant investigations were recorded. The PSQ was administered, and the neck circumference-to-height ratio (NHR) and body mass index (BMI) were measured.

The PSQ is a validated screening tool developed by Chervin et al.[10] for detecting SRBDs. The PSQ-SRBD subscale consists of 22 parent-reported yes/no items across three domains:

1. Snoring and breathing abnormalities
2. Daytime sleepiness
3. Behavioral symptoms

A score >8 out of 22 is considered significant based on the original validation by Chervin et al.[10] The tool demonstrates high sensitivity (about 81–85%) and specificity (about 87%) for detecting SRBDs.

Results

The demographic and clinical profiles of the study participants are presented in Table 1. The children were evenly distributed across the age groups: 2 < 5, 5 < 8, 8 < 11, and 11–12 years. The male-to-female distribution was nearly equal. Half of the children were responsive to ASMs. The majority had an onset of epilepsy at ≥2 years of age. The BMI analysis showed a mean BMI ± SD of 19.0 ± 2.4 with none of the children in the overweight or obese category. Overall, 44.5% of the children had SRBDs [Table 1]. No statistically significant differences were found between children with and without SRBDs in terms of age, gender, age at epilepsy onset, family history of seizures, seizure types, NHR, and BMI. However, children with SRBDs showed significantly higher values of PSQ (≥8/22) for the number of ASMs used (50.7% vs. 36.7%, P value = 0.004), seizure frequency (P value = 0.002), and drug resistance (P value = 0.001) [Table 2].

Table 1: Demographic and clinical profile of the study population

Variables	n=238
Age group in years, n (%)	
2 < 5	60 (25.2)
5 < 8	60 (25.2)
8 < 11	58 (24.4)
11–12	60 (25.2)
Gender, n (%)	
Female	114 (52.1)
Male	124 (47.9)
Epilepsy type, n (%)	
Responsive	119 (50.0)
Resistant	119 (50.0)
Age of onset of epilepsy in years, n (%)	
<1	18 (7.6)
1 < 2	85 (35.7)
≥2	135 (56.7)
BMI in kg/m^2, mean±SD	19±2.4
SRBD, n (%)	
Yes	106 (44.5)
No	132 (55.5)

BMI: Body mass index, SD: Standard deviation, SRBD: Sleep-related breathing disorder

Discussion

Sleep disorders are increasingly recognized as a significant comorbidity in CWE. Mechanistically, sleep fragmentation and nocturnal hypoxia may lower seizure thresholds, and ASMs may influence sleep architecture—factors that could explain the association observed between SRBDs and seizure severity.

In the present study, SRBDs were identified in 44.5% of children using the PSQ. Their prevalence was significantly higher among those receiving more than two ASMs, those with DRE, and those experiencing seizures on a daily or weekly basis. Prior studies have also used the PSQ in CWE, supporting its use as a feasible screening tool in this group.[11] Similar to our results, Jain et al.[11] also reported higher scores in PSQ in children with DRE (43.8% vs. 30.7%) and those on polytherapy (45.8% vs. 30.6%).

Manokaran et al.[5] reported that sleep abnormalities assessed using the Children's Sleep Habits Questionnaire were significantly more prevalent in the DRE group (72.5%)

Table 2: Association of various factors with SRBDs

Variables	PSQ <8/22, SRBD (-)	PSQ ≥8/22, SRBD (+)	Total	*P*
Age groups in years, n (%)				
2 < 5	34 (56.7)	26 (44.3)	60 (100.0)	0.932*
5 < 8	31 (51.7)	29 (48.3)	60 (100.0)	
8 < 11	33 (56.9)	25 (43.1)	58 (100.0)	
11–12	34 (56.7)	26 (44.3)	60 (100.0)	
Gender, n (%)				
Female	60 (52.6)	54 (47.4)	114 (100.0)	0.435*
Male	72 (58.1)	52 (41.9)	124 (100.0)	
Age of onset of epilepsy in years, n (%)				
<1	10 (55.6)	8 (44.4)	18 (100.0)	0.412*
1 < 2	52 (61.2)	33 (38.8)	85 (100.0)	
≥2	70 (51.9)	65 (48.1)	135 (100.0)	
Family history of seizures, n (%)				
Not significant	50 (63.3)	29 (37.9)	79 (100.0)	0.098*
Significant	82 (51.6)	77 (62.1)	159 (100.0)	
Number of anti-epileptic drugs, n (%)				
≤2	57 (66.3)	29 (36.7)	86 (100.0)	0.004*
>2	75 (49.3)	77 (50.7)	152 (100.0)	
Seizure frequency, n (%)				
Controlled	49 (73.1)	18 (26.9)	67 (100.0)	0.002*
Yearly	26 (59.1)	18 (40.9)	44 (100.0)	
Monthly	16 (57.1)	12 (42.9)	28 (100.0)	
Weekly	15 (37.5)	25 (62.5)	38 (100.0)	
Daily	26 (44.1)	33 (55.9)	59 (100.0)	
Types of seizures, n (%)				
Focal seizures	60 (56.6)	46 (43.4)	106 (100.0)	0.273*
Focal to bilateral tonic clonic seizures	26 (65.0)	14 (35.0)	40 (100.0)	
Generalized seizures	46 (50.0)	46 (50.0)	92 (100.0)	
Neck circumference/height ratio, n (%)				
≤0.22	106 (53.3)	93 (46.7)	199 (100.0)	0.159*
>0.22	26 (66.7)	13 (33.3)	39 (100.0)	
BMI in kg/m^2, mean±SD	19.1±2.3	18.7±2.0		0.261**
Drug responsive, n (%)	82 (68.9)	37 (31.1)	119 (100.0)	0.0001*
Drug-resistant, n (%)	50 (42.0)	69 (58.0)	119 (100.0)	

*The Chi-square test was used. **An unpaired *t*-test was used. BMI: Body mass index, PSQ: Pediatric sleep questionnaire, SD: Standard deviation, SRBD: Sleep-related breathing disorder

compared to the well-controlled epilepsy (WCE) group (32.5%) and the typically developing children (TDC) group (15%) (*P* value = 0.01). Additionally, the modified Pediatric Epworth Daytime Sleepiness Scale revealed excessive daytime sleepiness in 52.5% of children with DRE, versus 12.5% in WCE and 5% in TDC groups (*P* value = 0.03). Kaleyias et al.[12] conducted a study to examine the spectrum of polysomnographic (PSG) abnormalities in CWE. The study reported that 17 (42.5%), 16 (40%), 5 (12.5), 8 (20.0%), 3 (7.5%), and 4 (10.0%) of the children exhibited snoring, sleep-disordered breathing, obstructive hypoventilation, OSA, upper-airway resistance syndrome, and periodic limb movements during sleep, respectively. Differences in diagnostic approaches (screening tools vs. PSG), cultural influences on symptom reporting, and disparities in healthcare access can all affect detection rates. Variability in ASM regimens, many of which alter sleep architecture, may also contribute to inconsistent prevalence across studies.

Although general pediatric studies have demonstrated a clear relationship between higher BMI and sleep-disordered breathing, evidence specific to CWE remains limited. Gogou et al.[6] compared CWE and healthy controls using PSG, but did not provide detailed BMI-SRBD analyses. More recent studies offer clearer insights: Urquhart et al.[13] reported significantly higher BMI z-scores in CWE compared to controls (0.7 vs. 0.1; *P* value = 0.03) using combined PSQ-SRBD screening and PSG. Hill et al.[14] also found a significant association, with CWE who screened positive on the PSQ-SRBD scale demonstrating higher mean BMI values (22.3 ± 6.0 vs. 19.1 ± 3.7 kg/m^2; *P* value = 0.03). In contrast, our study did not find a correlation between BMI and SRBDs, likely due to the absence of overweight or obese children in our study population.

ASMs can influence sleep architecture, but reported effects remain inconsistent. Older drugs such as phenytoin and

carbamazepine may reduce REM sleep or increase sleep fragmentation, whereas gabapentin may improve slow-wave sleep; other agents, including levetiracetam and lamotrigine, show variable findings across studies. Valproate may cause weight gain which can worsen OSA.[15] Because these effects are often confounded by seizure burden and polytherapy, results differ widely. As ASMs can independently alter sleep patterns, their potential confounding effect on SRBDs could not be fully determined in our cohort.

Messner and Pelayo[3] studied SRBDs in the general pediatric population and underscored the potential consequences of untreated SRBDs on cardiovascular health, neurocognitive development, and quality of life. They emphasized that SRBDs are treatable and management includes adenotonsillectomy as the first-line treatment, with positive airway pressure (continuous positive air pressure/bi-level positive air pressure) used when surgery is ineffective or not possible; conservative measures (weight loss and positional therapy) and medical therapy play limited roles, while complex craniofacial cases may require specialized maxillofacial surgery or tracheostomy.[9]

Given its treatable nature, pediatricians and pediatric neurologists should therefore routinely screen CWE, particularly those with poor seizure control, for signs and symptoms of SRBDs and refer high-risk cases to sleep centers, where PSG remains the gold standard for diagnosis. Early recognition enables timely management and may prevent unnecessary ASMs and unwarranted investigations. Incorporating routine screening can therefore improve diagnostic clarity and optimize care in CWE. Identifying SRBDs in this population is particularly important, as it can influence seizure control as well as broader neurodevelopmental outcomes.

SRBD diagnosis in this study relied on questionnaires rather than PSG, the diagnostic gold standard; however, pandemic-related constraints made questionnaire-based screening more feasible. We were unable to compare SRBD prevalence with TDC, limiting conclusions about whether SRBDs are truly higher in CWE, and a future multicentric case-control study would strengthen this evidence. Although BMI was analyzed, the absence of overweight or obese children in this study restricted its assessment as a risk factor. Several PSQ items reflect sleepiness and behavior that may be influenced by ASMs and comorbidities such as attention deficit hyperactivity disorder, potentially inflating scores in the absence of confirmatory PSG. Future studies evaluating outcomes after treating SRBDs in CWE would provide valuable insights, particularly regarding effects on seizure control and behavior.

Conclusions

SRBDs can adversely affect neuropsychological development, seizure control, and overall functioning in CWE. In the present study, 44.5% of CWE screened positive for SRBDs, with a higher prevalence observed in those on ≥2 ASMs, with DRE, or with frequent seizures. Despite uncertain causality, SRBDs are a treatable and clinically relevant comorbidity in epilepsy. Routine screening with the PSQ is practical and effective in epilepsy clinics, and children with positive scores should undergo PSG where available to confirm diagnosis and guide intervention. This stepwise approach is feasible even in resource-limited settings and may improve seizure control and quality of life in CWE.

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Authors' contribution

AA: research design, acquisition of data, analysis of data, interpretation of data, revising critically, approving the submitted version. ND: research design, acquisition of data, interpretation of data, revising critically, approving the submitted version. DP: research design, analysis of data, interpretation of data, drafting the paper, revising critically, approving the submitted version. VU: research design, acquisition of data, interpretation of data, revising critically, approving the submitted version.

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Conflicts of interest

There are no conflicts of interest.

Data availability statement

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

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