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Parent-Reported Sleep Disturbance Profiles in Children with Epilepsy: A Cross-Sectional Study Using the Sleep Disturbance Scale for Children (SDSC)

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Highlights

What are the main findings?

- Exploratory analyses suggested higher scores for difficulties initiating and maintaining sleep in structural epilepsy and for night sweating in epileptic encephalopathies. These associations remained evident after adjustment for age and sex but were at or near the threshold of significance after correction for multiple testing across sleep domains.
- Lower sleep–wake transition scores in idiopathic generalized epilepsy were attenuated after adjustment for age.

What are the implications of the main findings?

- The observed domain patterns identify potential priorities for further study but require confirmation before being used to guide epilepsy-specific sleep assessment.
- Age should be taken into account when comparing SDSC subscale scores between groups of children.

Abstract

Background/Objectives: It is unclear whether sleep disturbances differ between epilepsy phenotypes defined by current International League Against Epilepsy (ILAE) criteria. We explored parent-reported sleep disturbances in children with idiopathic generalized epilepsy (IGE) and other epilepsies. **Methods:** Parents of 127 children with epilepsy completed the Polish Sleep Disturbance Scale for Children (SDSC). We compared IGE (n = 31) with other epilepsies by epilepsy type, etiology, and category, using non-parametric tests with false discovery rate (FDR) correction and linear regression adjusted for age and sex. **Results:** Children with IGE had lower total SDSC scores than children with other epilepsies (median 39 vs. 44), but this difference was small and not robust in sensitivity analyses. Compared with IGE, structural epilepsy (n = 18) was associated with higher scores for difficulties initiating and maintaining sleep (DIMS; β = 4.3, 95% CI 1.5 to 7.0) and epileptic encephalopathies (n = 15) with higher sleep hyperhidrosis (SHY) scores (β = 1.5, 95% CI 0.33 to 2.7); after FDR correction, both were at or near the threshold of significance (q = 0.053 and q = 0.047). Lower sleep–wake transition scores in IGE were explained by older age. **Conclusions:** Children with IGE may have milder parent-reported sleep disturbances. The DIMS and SHY findings are exploratory, and age should be considered when interpreting SDSC subscales.

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1. Introduction

Sleep and epilepsy influence each other in both directions [1–3]. Sleep, and especially non-rapid eye movement (NREM) sleep, facilitates interictal epileptiform discharges and certain seizure types, while sleep deprivation lowers the seizure threshold in susceptible patients [1,2]. Conversely, nocturnal seizures, interictal activity, antiseizure medications (ASMs), and comorbid neurological and psychiatric conditions can fragment sleep and change its macro- and microstructure [1,3,4]. Increased wake after sleep onset is the most consistent polysomnographic finding in people with epilepsy [1]. Sleep is also needed for brain development, synaptic plasticity, and memory consolidation, so in children with epilepsy, poor sleep may affect more than the night itself [5].

Sleep problems are more common in children with epilepsy than in the general pediatric population. In questionnaire studies, 57–84% of children with epilepsy have at least one abnormal sleep domain [6,7], and parents of children treated in a tertiary epilepsy center report major sleep complaints many times more often than parents of healthy children [1]. Sleep disturbances in this population are associated with behavioral and emotional problems [7,8] and, in adults, with seizure frequency, fatigue, and depressive symptoms [9]. Sleep problems are also easily missed in routine care: in one study, 75.7% of children whose parents said they "slept well" had abnormalities on a structured questionnaire [6].

The Sleep Disturbance Scale for Children (SDSC) is a parent-report questionnaire that gives a total score and scores for six domains corresponding to the main groups of childhood sleep disorders [10]. It has been validated in many languages, including Polish [11], and is widely used in children with epilepsy [6,7,12]. Previous SDSC studies in pediatric epilepsy have mainly reported how common sleep disturbances are, or compared children with epilepsy to healthy controls or to children with other neurological conditions [7,12]. Results on differences within epilepsy have been inconsistent. Zambrelli et al. found no differences in SDSC scores across epilepsy types or etiologies [7]. Cohen et al. concluded that the SDSC does not differentiate between neurological disorders, apart from more frequent sleep–wake transition disorders in epilepsy [12].

In contrast, Furones García et al. found that the type of sleep problem depended on the characteristics of epilepsy. Children with generalized epilepsy more often had sleep–wake transition disorders and excessive somnolence than children with focal epilepsy. Nocturnal seizures, drug resistance, and epileptic encephalopathy were associated with more frequent sleep–wake transition disorders, and epileptic encephalopathy and intellectual disability with more frequent difficulties initiating and maintaining sleep [6].

One likely reason is that broad categories such as "generalized epilepsy" combine clinically very different patients. In 2022, the International League Against Epilepsy (ILAE) defined idiopathic generalized epilepsies (IGEs) as a distinct subgroup of genetic generalized epilepsies, comprising four syndromes: childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE), juvenile myoclonic epilepsy (JME), and epilepsy with generalized tonic–clonic seizures alone (GTCA) [13]. IGEs have a presumed polygenic basis, typically normal development, and a generally good prognosis for seizure control. Additionally, they do not evolve into an epileptic encephalopathy [13]. In contrast, structural epilepsies and epileptic encephalopathies often co-occur with intellectual disability, drug resistance, and polytherapy, all of which are known to affect sleep [4,6]. IGEs also have a characteristic

relationship with the sleep–wake cycle: seizures typically occur after awakening, and sleep deprivation is a major trigger [1,13].

Thus, previous SDSC studies in pediatric epilepsy either compared children with epilepsy with other groups or used broad categories such as generalized versus focal epilepsy. To our knowledge, no study has separated IGE, as defined by the ILAE in 2022, from other generalized epilepsies or compared SDSC domain profiles across complementary levels of the ILAE classification. This study aimed to explore whether sleep disturbances in children with IGE differ in severity and profile from those in children with other epilepsies. Because the ILAE framework classifies epilepsy at several complementary levels [14], we compared total and domain SDSC scores at three levels: epilepsy type, contrasting IGE with all other epilepsies (stage 1, the main comparison); etiology, to examine whether differences reflect the underlying cause (stage 2); and epilepsy category, to separate IGE from other generalized epilepsies and to examine epileptic encephalopathies as a distinct group (stage 3).

2. Materials and Methods

2.1. Study Design and Participants

This cross-sectional, questionnaire-based study was conducted at the Department of Developmental Neurology, Poznan University of Medical Sciences, between November 2025 and August 2026. All consecutive children treated in the department during this period whose parents or legal guardians completed the SDSC were entered into the study database (n = 301). The SDSC was completed during the child's stay in the department as part of a research project, and clinical data corresponding to the time of questionnaire completion were obtained from medical records. For the present analysis, the inclusion criteria were (1) a diagnosis of epilepsy according to the ILAE criteria [14] and (2) an SDSC questionnaire completed by a parent or legal guardian. No additional exclusion criteria were applied; in particular, children were not excluded because of comorbidities or partially incomplete questionnaires (Section 2.2). Of the 301 children in the database, 127 met the inclusion criteria. The other children in the database had neurodevelopmental disorders (n = 69), headache (n = 31), neuromuscular disorders (n = 20), cerebral palsy (n = 7), or other neurological conditions (n = 47). The children with epilepsy were aged 1–17 years (median 11, Q1–Q3 8–14). Twenty-eight children (22%) were outside the age range for which the SDSC was validated (6–15 years) [11]: 7 were younger than 6 and 21 were older than 15. They were included in the main analysis because excluding them would have removed 13 of the 31 children with IGE (42%; 12 older than 15 years and 1 younger than 6 years), whose syndromes typically begin in adolescence [13], and a sensitivity analysis was conducted restricting the sample to children aged 6–15 years.

Demographic data (age, sex) and clinical data (epilepsy type, category, and etiology) were collected from medical records. Epilepsy was diagnosed and classified by pediatric neurologists according to the ILAE 2017 classification of the epilepsies, which defines seizure type, epilepsy type, epilepsy syndrome, and etiology [14], and according to the 2022 ILAE definitions of epilepsy syndromes [13,15–17].

The study was conducted in accordance with the Declaration of Helsinki. The Bioethics Committee of Poznan University of Medical Sciences issued a formal written statement (no. KB-683/26, 30 September 2026) determining that the study, as a questionnaire-based study without the characteristics of a medical experiment, was not subject to review by the Bioethics Committee under Polish law and Good Clinical Practice. Parents or legal guardians were informed about the purpose of the questionnaire, and their oral consent was obtained before it was completed; participation was voluntary. The SDSC is a parent-report instrument, and the children did not complete any questionnaire or undergo any additional

procedure; therefore, separate assent from the children was not sought. Clinical data were transferred from medical records to a pseudonymized database in which patients were identified only by codes. The database was stored on a secured hospital computer accessible only to the research team, and the results are reported only in aggregate form.

2.2. Sleep Assessment

Sleep disturbances were assessed with the Polish version of the SDSC [10,11], completed by a parent or caregiver. The SDSC has 26 items describing the child's sleep over the previous 6 months, rated on a 5-point Likert scale. The total score ranges from 26 to 130, with higher scores indicating more severe sleep disturbances. The items form six domains: disorders of initiating and maintaining sleep (DIMS; items 1–5, 10, 11), sleep breathing disorders (SBD; items 13–15), disorders of arousal (DA; items 17, 20, 21), sleep–wake transition disorders (SWTD; items 6–8, 12, 18, 19), disorders of excessive somnolence (DOES; items 22–26) and sleep hyperhidrosis (SHY; items 9, 16) [10,11]. The Polish version has excellent internal consistency (Cronbach's $\alpha = 0.90$) and a confirmed six-factor structure [11]. In this study, total and domain scores were analyzed as continuous variables. In 19 questionnaires (15%), 1–4 items were unanswered (1 item in 12, 2 items in 4, 3 or 4 items in 3); missing items were not imputed, and domain and total scores were calculated as the sum of the answered items. Because each item is scored from 1 to 5, this approach underestimates the scores of incomplete questionnaires, and it could bias group comparisons if incomplete questionnaires were unevenly distributed between groups. Incomplete questionnaires were similarly frequent in children with IGE (5/31, 16%) and in children with other epilepsies (14/96, 15%; Fisher's exact test $p = 0.78$), and in the other groups they ranged from 7% (encephalopathies) to 24% (genetic etiology). To assess the effect of this approach, we performed two sensitivity analyses (Section 2.4).

To describe the clinical relevance of group differences, we used the Polish normative data for children aged 6–15 years (mean ± SD: total score 40.8 ± 11.7; DIMS 13.1 ± 4.3; SWTD 8.9 ± 3.2; SHY 2.8 ± 1.4) [11]. These data were used only descriptively, to express group differences in normative SD units and to report the proportion of children with scores more than 1 SD above the normative mean (T-score > 60); they were not used for statistical comparisons with the general population.

2.3. Classification of Epilepsy

Epilepsy type (stage 1). Children were divided into those with IGE and those with all other epilepsy types. The IGE group included children with CAE, JAE, JME, or GTCA, in line with the ILAE 2022 definition [13]: JME (n = 17), JAE (n = 6), CAE (n = 4), and GTCA (n = 1); in 3 children, the IGE syndrome was not further specified in the records.

Etiology (stage 2). Etiology was classified according to the ILAE 2017 framework as genetic, structural, immune, or unknown [14]. The ILAE defines IGEs as genetic generalized epilepsies with a presumed polygenic basis [13]. Children with IGE were therefore analyzed as a separate etiological group (IGE, presumed polygenic etiology), and the genetic group included children with other genetic epilepsies with a confirmed or strongly presumed monogenic or chromosomal cause. Stage 2 included only the three groups with known etiology and adequate size: IGE (n = 31), genetic (n = 25), and structural (n = 18); N = 74. Children with unknown etiology (n = 44) or no etiological data in the records (n = 6) were excluded because this group was clinically heterogeneous, and the immune group (n = 3) was excluded because it was too small.

Epilepsy category (stage 3). IGE (n = 31) was compared with three groups defined by epilepsy category: other, non-idiopathic generalized epilepsies (n = 28), focal epilepsies (n = 47), and epileptic encephalopathies (n = 15); N = 121. The "other generalized

epilepsies" group included children with generalized epilepsy who did not meet the ILAE criteria for any of the four IGE syndromes [13,15]. The focal epilepsies group (n = 47) included focal epilepsies of unknown (n = 21), structural (n = 16), genetic (n = 8), and immune (n = 2) etiology. In line with ILAE terminology, the epileptic encephalopathies group included both developmental and epileptic encephalopathies (DEE), in which both the underlying cause and the epileptic activity contribute to developmental impairment, and epileptic encephalopathies (EE), in which the epileptic activity itself causes cognitive and behavioral impairment beyond that expected from the underlying pathology [14–17]. This group comprised Lennox–Gastaut syndrome (n = 5), Dravet syndrome (n = 1), infantile epileptic spasms syndrome (West syndrome; n = 1), epilepsy with myoclonic–atonic seizures (Doose syndrome; n = 1) and DEE/EE without a specified syndrome (n = 7); etiology was genetic in 10, unknown in 4 and structural in 1 child. Six children had no data on epilepsy category or etiology in the records and were not included in stage 3.

2.4. Statistical Analysis

Continuous variables are presented as medians and interquartile ranges (Q1–Q3), and categorical variables as numbers and percentages. Because SDSC scores were not normally distributed, we used non-parametric tests. In stage 1, age and SDSC scores were compared using the Mann–Whitney *U* test, and sex distribution was compared using Pearson's chi-square test. Effect size was expressed as the rank-biserial correlation (r); a positive value means lower scores in the IGE group. In stages 2 and 3, SDSC scores were compared with the Kruskal–Wallis test, with effect size expressed as epsilon-squared (ε^2). Significant results were followed by Dunn's post-hoc test with Holm correction within each outcome; ε^2 was calculated as $H/(n-1)$. The total SDSC score was the primary outcome, and the six domain scores were secondary, exploratory outcomes. To control the false discovery rate (FDR) across the six domains, the *p* values of the domain comparisons within each stage were adjusted using the Benjamini–Hochberg procedure [18] and are reported as q values. No correction was applied across the three stages, which represent complementary, overlapping groupings of the same children rather than independent hypotheses.

In each stage, we fitted a multiple linear regression model with the total SDSC score as the dependent variable and group (IGE vs. other epilepsies; etiology; or epilepsy category), age, and sex as independent variables. IGE and female sex were the reference categories. Linear regression was used to estimate group differences in the original score units while adjusting for age and sex, which the non-parametric tests do not allow; the two approaches were therefore complementary. We report regression coefficients (β) with 95% confidence intervals (CI), R^2, and adjusted R^2. Because the groups differed in age, we also fitted models with group, age, and sex for the domain scores that differed between groups in unadjusted analyses; adjusted models for all domains are shown in Table S1. Model assumptions were checked using the Shapiro–Wilk test of residuals, the Breusch–Pagan test for heteroscedasticity, variance inflation factors (VIF), and Cook's distance (Table S2). Because residuals deviated from normality in several models, we also calculated *p* values based on heteroscedasticity-consistent (HC3) standard errors and refitted the models with rank-transformed outcomes. To assess a possible non-linear effect of age, we refitted the models with age modeled as a restricted cubic spline with three knots (at the 10th, 50th, and 90th percentiles of age). We repeated the adjusted analyses in children aged 6–15 years, the age range for which the SDSC was validated [11]. To assess the effect of missing items, we repeated the main group comparisons (1) with prorated scores, in which each missing item was replaced by the mean of the child's answered items in the same domain, and (2) with complete data only, excluding for each domain the children with missing items in that domain and, for the total score, the children with any missing item. Given the small size of

some groups, we emphasize effect estimates and their 95% CIs rather than p values alone. All tests were two-sided with a significance level of 0.05. All analyses were performed in R (version 4.6.1; R Foundation for Statistical Computing, Vienna, Austria) in RStudio (version 2025.09.2 Build 418; Posit Software, PBC, Boston, MA, USA). Generative AI (Claude Opus 5.5; Anthropic PBC, San Francisco, CA, USA) was used to assist with generating the R code for the statistical analyses and with interpreting the results; the authors ran the analyses and verified the results.

3. Results

3.1. Stage 1: IGE vs. Other Epilepsies

The study included 127 children with epilepsy: 31 with IGE and 96 with other epilepsy types. Children with IGE were significantly older than children with other epilepsies (median 14 vs. 9 years; $p < 0.001$). Twelve of the 31 children with IGE (39%) were older than 15 years, and one was younger than 6 years, compared with 9 of the 96 children (9%) with other epilepsies. Sex distribution did not differ significantly ($p = 0.2$) (Table 1).

Table 1. Baseline characteristics of children with idiopathic generalized epilepsy (IGE) and other epilepsies.

Characteristic	IGE N = 31	Other Epilepsies N = 96	*p*
Age (years)	14 (12–16)	9 (7–13.5)	<0.001
Sex			0.2
Girls	19 (61%)	43 (45%)	
Boys	12 (39%)	53 (55%)	

Median (Q1–Q3); n (%). Mann–Whitney *U* test; Pearson's chi-square test.

Children with IGE had significantly lower total SDSC scores than children with other epilepsies (median 39, Q1–Q3 33–46, vs. 44, Q1–Q3 37–53; $p = 0.021$; r = 0.28). Among the domains, children with IGE scored lower for SWTD ($p = 0.004$; r = 0.34) and SHY ($p = 0.032$; r = 0.22) (Table 2). After FDR correction across the six domains, only the SWTD difference remained significant (q = 0.024; SHY, q = 0.097). For DIMS, SBD, and DA, there was a non-significant trend in the same direction ($p = 0.075$–0.090). DOES scores did not differ between groups ($p = 0.7$) (Table 2).

Table 2. SDSC scores in children with IGE and other epilepsies.

Variable	IGE N = 31	Other Epilepsies N = 96	*p*	q	r
SDSC total	39 (33–46)	44 (37–53)	0.021	–	0.28
DIMS	13 (9–16)	14 (11–16)	0.083	0.11	0.21
SBD	3 (3–4)	3.5 (3–5)	0.090	0.11	0.19
DA	3 (3–3)	3 (3–5)	0.075	0.11	0.18
SWTD	7 (6–11)	10 (8–12.5)	0.004	0.024	0.34
DOES	9 (7–11)	8 (6–12)	0.7	0.7	−0.04
SHY	2 (2–2)	2 (2–4)	0.032	0.097	0.22

Median (Q1–Q3). Mann–Whitney *U* test; q, Benjamini–Hochberg FDR-adjusted p value across the six domains; r = rank-biserial correlation. DIMS, disorders of initiating and maintaining sleep; SBD, sleep-breathing disorders; DA, disorders of arousal; SWTD, sleep–wake transition disorders; DOES, disorders of excessive somnolence; SHY, sleep hyperhidrosis.

In multiple linear regression adjusted for age and sex, having an epilepsy other than IGE remained significantly associated with a higher total SDSC score (β = 5.6; 95% CI 0.24–11; $p = 0.041$). Age and sex were not significant predictors. The model explained a small proportion of variance ($R^2 = 0.049$; adjusted $R^2 = 0.026$; model $p = 0.10$) (Table 3).

Table 3. Multiple linear regression for the total SDSC score (stage 1).

Predictor	β	95% CI	*p*
(Intercept)	42	32 to 51	<0.001
Group: other epilepsies (vs. IGE)	5.6	0.24 to 11	0.041
Age	−0.14	−0.73 to 0.46	0.7
Sex: boy (vs. girl)	−0.22	−4.4 to 4.0	>0.9

N = 127; R^2 = 0.049; adjusted R^2 = 0.026; model p = 0.10.

3.2. Stage 2: Etiology

Among the 74 children with known etiology (IGE, n = 31; genetic, n = 25; structural, n = 18), total SDSC scores differed significantly between groups (p = 0.041). Scores were lowest in the IGE group (median 39, Q1–Q3 33–46), intermediate in the genetic group (median 41, Q1–Q3 37–46), and highest in the structural group (median 46, Q1–Q3 40–54). DIMS scores also differed significantly (p = 0.009), with the highest scores in children with structural epilepsy (median 16, Q1–Q3 14–18) compared with IGE (median 13, Q1–Q3 9–16) and genetic epilepsy (median 14, Q1–Q3 10–15); within-domain Holm-adjusted pairwise comparisons indicated higher scores in structural epilepsy than in IGE (p = 0.007) and genetic epilepsy (p = 0.048). After FDR correction across the six domains, the DIMS difference was just above the threshold of significance (q = 0.053). The other domains did not differ significantly (Table 4).

Table 4. SDSC scores by epilepsy etiology (known etiology only).

Variable	IGE N = 31	Genetic N = 25	Structural N = 18	*p*	q	ε^2
SDSC total	39 (33–46)	41 (37–46)	46 (40–54)	0.041 [a]	–	0.088
DIMS	13 (9–16)	14 (10–15)	16 (14–18)	0.009 [b]	0.053	0.130
SBD	3 (3–4)	3 (3–5)	3 (3–5)	0.5	0.8	0.018
DA	3 (3–3)	3 (3–3)	3 (3–5)	0.8	0.8	0.005
SWTD	7 (6–11)	8 (7–12)	10 (8–12)	0.10	0.2	0.063
DOES	9 (7–11)	8 (7–10)	9.5 (6–12)	0.7	0.8	0.012
SHY	2 (2–2)	2 (2–4)	2 (2–4)	0.091	0.2	0.066

Median (Q1–Q3). Kruskal–Wallis test; q, Benjamini–Hochberg FDR-adjusted p value across the six domains; ε^2, epsilon-squared. Dunn's post-hoc test with Holm correction: [a] IGE vs. structural p = 0.035; [b] IGE vs. structural p = 0.007; genetic vs. structural p = 0.048.

After adjustment for age and sex, structural epilepsy was associated with a significantly higher total SDSC score than IGE (β = 6.8; 95% CI 0.63–13; p = 0.031). Genetic epilepsy did not differ significantly from IGE (β = 2.9; p = 0.3). Age and sex were not significant predictors (R^2 = 0.090; adjusted R^2 = 0.037; model p = 0.2) (Table 5).

Table 5. Multiple linear regression for the total SDSC score (stage 2).

Predictor	β	95% CI	*p*
(Intercept)	38	29 to 47	<0.001
Etiology: genetic (vs. IGE)	2.9	−2.6 to 8.3	0.3
Etiology: structural (vs. IGE)	6.8	0.63 to 13	0.031
Age	0.10	−0.55 to 0.75	0.8
Sex: boy (vs. girl)	1.4	−3.2 to 5.9	0.6

N = 74; R^2 = 0.090; adjusted R^2 = 0.037; model p = 0.2.

In a preliminary analysis that also included children with unknown etiology (n = 50), SWTD scores differed significantly between groups. This difference was driven mainly by the unknown-etiology group and was no longer significant once that group was excluded.

3.3. Stage 3: Epilepsy Category

Comparing IGE with other generalized epilepsies, focal epilepsies, and epileptic encephalopathies showed no significant difference in total SDSC score ($p = 0.083$). However, scores tended to be lower in IGE (median 39, Q1–Q3 33–46) than in the other groups (medians 40–45). SWTD scores differed between groups ($p = 0.016$; q = 0.047), with a significant post-hoc difference between IGE (n = 31) and focal epilepsies (n = 47; $p = 0.009$). SHY scores also differed ($p = 0.011$; q = 0.047), with a significant post-hoc difference between IGE and epileptic encephalopathies (n = 15; $p = 0.005$) (Table 6). The other domains did not differ significantly (Table 6).

Table 6. SDSC scores by epilepsy category.

Variable	IGE N = 31	Other Generalized N = 28	Focal N = 47	Encephalopathies N = 15	*p*	q	ε^2
SDSC total	39 (33–46)	40 (37–54)	45 (38–50)	45 (34–56)	0.083	–	0.056
DIMS	13 (9–16)	14 (11–16)	15 (10–17)	15 (10–17)	0.3	0.4	0.030
SBD	3 (3–4)	4 (3–5)	3 (3–5)	4 (3–5)	0.3	0.4	0.034
DA	3 (3–3)	3 (3–3.5)	3 (3–5)	3 (3–6)	0.080	0.2	0.056
SWTD	7 (6–11)	10 (7.5–12)	10 (8–13)	8 (7–11)	0.016 [a]	0.047	0.087
DOES	9 (7–11)	7.5 (7–13)	9 (6–12)	8 (7–13)	>0.9	>0.9	0.001
SHY	2 (2–2)	2 (2–3)	2 (2–4)	4 (2–6)	0.011 [b]	0.047	0.093

Median (Q1–Q3). Kruskal–Wallis test; q, Benjamini–Hochberg FDR-adjusted *p* value across the six domains; ε^2, epsilon-squared. Dunn's post-hoc test with Holm correction: [a] IGE vs. focal $p = 0.009$; [b] IGE vs. encephalopathies $p = 0.005$ (encephalopathies vs. focal $p = 0.077$ and vs. other generalized $p = 0.097$).

In regression analysis adjusted for age and sex, no group differed significantly from IGE, although the difference for focal epilepsies was close to significance (β = 5.9; 95% CI −0.02 to 12; $p = 0.051$). The model as a whole was not statistically significant ($R^2 = 0.058$; adjusted $R^2 = 0.017$; model $p = 0.2$) (Table 7).

Table 7. Multiple linear regression for the total SDSC score (stage 3).

Predictor	β	95% CI	*p*
(Intercept)	42	32 to 52	<0.001
Group: other generalized (vs. IGE)	5.6	−1.1 to 12	0.10
Group: focal (vs. IGE)	5.9	−0.02 to 12	0.051
Group: encephalopathies (vs. IGE)	6.7	−1.3 to 15	0.10
Age	−0.16	−0.80 to 0.47	0.6
Sex: boy (vs. girl)	−0.26	−4.6 to 4.1	>0.9

N = 121; $R^2 = 0.058$; adjusted $R^2 = 0.017$; model $p = 0.2$.

3.4. Age-Adjusted Analyses

In the whole cohort, SWTD scores decreased with age (Spearman's ρ = −0.35; $p < 0.001$). The other domains and the total score were not significantly correlated with age ($|\rho| \leq 0.14$; $p \geq 0.11$). Table 8 shows the adjusted group differences for the total score and for the domains that differed between groups in unadjusted analyses. After adjustment for age and sex, the SWTD differences between IGE and other epilepsies (β = 1.0; 95% CI −0.56 to 2.6) and between IGE and focal epilepsies (β = 1.3; 95% CI −0.33 to 3.0) were no longer significant, and neither was the SHY difference between IGE and other epilepsies

(β = 0.48; 95% CI −0.32 to 1.3). In contrast, DIMS scores remained higher in structural epilepsy (n = 18) than in IGE (n = 31; β = 4.3; 95% CI 1.5 to 7.0), and SHY scores remained higher in epileptic encephalopathies (n = 15) than in IGE (β = 1.5; 95% CI 0.33 to 2.7). The total score remained higher in other epilepsies (β = 5.6; 95% CI 0.24 to 11.0) and in structural epilepsy (β = 6.8; 95% CI 0.63 to 12.9) than in IGE.

Table 8. Age- and sex-adjusted group differences in SDSC scores and sensitivity analyses.

Comparison	β (95% CI)	*p*	*p* (HC3)	*p* (Rank)	Age Spline: β (*p*)	Age 6–15 Years: β (*p*)
Other epilepsies vs. IGE: total	5.6 (0.24 to 11.0)	0.041	0.028	0.068	5.5 (0.047)	6.3 (0.059)
Other epilepsies vs. IGE: SWTD	1.0 (−0.56 to 2.6)	0.20	0.16	0.17	0.94 (0.24)	0.92 (0.35)
Other epilepsies vs. IGE: SHY	0.48 (−0.32 to 1.3)	0.24	0.17	0.10	0.43 (0.29)	0.48 (0.35)
Structural vs. IGE: total	6.8 (0.63 to 12.9)	0.031	0.040	0.028	7.0 (0.024)	6.9 (0.070)
Structural vs. IGE: DIMS	4.3 (1.5 to 7.0)	0.003	0.002	0.002	4.3 (0.002)	4.9 (0.006)
Focal vs. IGE: SWTD	1.3 (−0.33 to 3.0)	0.12	0.11	0.065	1.3 (0.12)	1.5 (0.14)
Encephalopathies vs. IGE: SHY	1.5 (0.33 to 2.7)	0.012	0.030	0.003	1.5 (0.014)	1.6 (0.032)

Linear regression with group, age, and sex; β, difference vs. IGE in score points. HC3, heteroscedasticity-consistent standard errors; rank, model with rank-transformed outcome; age spline, age modeled as a restricted cubic spline with three knots. Sample sizes in children aged 6–15 years: stage 1, IGE 18 and other epilepsies 81; stage 2, IGE 18, genetic 21, and structural 15; stage 3, IGE 18, other generalized 26, focal 38, and encephalopathies 12.

Modeling age as a restricted cubic spline did not materially change any of the estimates (Table 8). The DIMS and SHY differences were also consistent with HC3-based *p* values and with rank-transformed outcomes, whereas the total-score difference between IGE and other epilepsies was not significant in the rank-based model ($p = 0.068$). In children aged 6–15 years, the DIMS and SHY differences remained significant (structural, n = 15, vs. IGE, n = 18: $p = 0.006$; encephalopathies, n = 12, vs. IGE: $p = 0.032$), whereas the total-score differences did not (IGE, n = 18, vs. other epilepsies, n = 81: $p = 0.059$; of the 31 children with IGE, 12 were older than 15 years and 1 was younger than 6 years). In adjusted models for all domains (Table S1), the only additional nominal difference was higher DIMS scores in other epilepsies than in IGE (β = 2.3; 95% CI 0.07 to 4.5; $p = 0.044$). In the models shown in Table 8, residuals were approximately normal in the stage 2 models (Shapiro–Wilk $p \geq 0.29$) but not in the other models ($p < 0.001$), reflecting the skewed and bounded score distributions. There was no evidence of heteroscedasticity (Breusch–Pagan $p \geq 0.09$), multicollinearity (VIF ≤ 1.8), or highly influential observations (maximum Cook's distance 0.23) (Table S2).

These covariate-adjusted domain analyses followed selection based on the unadjusted comparisons and were not corrected for multiplicity. Their *p* values and confidence intervals should therefore be interpreted as exploratory.

3.5. Sensitivity Analyses for Missing Items

Each missing item affected only the total score and the domain to which it belonged. Incomplete questionnaires were similarly frequent in all groups (Section 2.2). Missing items occurred in the DIMS (n = 7 children), SWTD (n = 7), DOES (n = 6), DA (n = 4), and SHY (n = 1) domains, and not in the SBD domain. With prorated scores, the results did not change: total SDSC scores remained lower in IGE than in other epilepsies ($p = 0.016$), total and DIMS scores differed between etiological groups ($p = 0.029$ and $p = 0.007$, respectively), and SWTD and SHY scores differed between epilepsy categories ($p = 0.010$ and $p = 0.019$, respectively). Prorating increased the scores of incomplete questionnaires by a median of 2 points.

After excluding children with missing items in the relevant domain, the previously identified domain comparisons continued to meet the nominal significance threshold.

These analyses assessed sensitivity to missing responses but did not address multiplicity across domains.

SWTD scores were lower in IGE than in other epilepsies ($p = 0.003$) and differed between epilepsy categories ($p = 0.014$; IGE vs. focal, $p = 0.008$). SHY scores differed between epilepsy categories ($p = 0.013$; IGE vs. encephalopathies, $p = 0.006$; adjusted for age and sex, $p = 0.017$), and DIMS scores differed between etiological groups ($p = 0.048$; IGE vs. structural, $p = 0.048$; adjusted for age and sex, $p = 0.012$). Only the total score, which was affected by any missing item, no longer differed significantly between IGE and other epilepsies ($p = 0.19$) or between etiological groups ($p = 0.21$) when the 19 incomplete questionnaires were excluded. This was mainly due to the exclusion of five children with IGE who reported few symptoms (total scores 26–39). Thus, the domain findings were robust to the handling of missing items, whereas the total-score differences involving IGE were less robust.

3.6. Clinical Relevance of the Differences (Descriptive)

Expressed in units of the Polish normative SD [11], the adjusted differences corresponded to about 1.0 SD for DIMS (structural epilepsy vs. IGE; 4.3/4.3 points), 1.1 SD for SHY (encephalopathies vs. IGE; 1.5/1.4 points), and 0.5 SD for the total score (other epilepsies vs. IGE; 5.6/11.7 points). Scores more than 1 SD above the normative mean (T-score > 60) were observed for DIMS in 6 of 18 children with structural epilepsy (33%) and 3 of 31 children with IGE (10%), for SHY in 6 of 15 children with epileptic encephalopathies (40%) and 2 of 31 children with IGE (6%), and for the total score in 24 of 96 children with other epilepsies (25%) and 2 of 31 children with IGE (6%). Using the cut-offs for pathology proposed in the Polish validation (T-score > 70) [11], 4 of 15 children with encephalopathies (27%) and 2 of 31 children with IGE (6%) had a pathological SHY score, and 1 of 18 children with structural epilepsy (6%) and none with IGE had a pathological DIMS score. These figures are descriptive and were not tested statistically.

4. Discussion

This exploratory study suggested differences in selected parent-reported sleep domains across epilepsy groups. Children with IGE had lower total scores than children with other epilepsies, but this difference was small and sensitive to the handling of missing items and to age restriction. Higher DIMS scores in structural epilepsy and higher SHY scores in epileptic encephalopathies remained evident after adjustment for age and sex, regardless of whether age was modeled linearly or flexibly, and in children aged 6–15 years. However, after FDR correction across domains, both were at or near the threshold of significance ($q = 0.053$ and $q = 0.047$), and no correction was applied across stages or regression models. The findings therefore identify associations for further investigation rather than establish distinct sleep profiles for particular epilepsy groups. The SWTD differences involving IGE were explained by age.

4.1. IGE and the Role of Age

The median total SDSC score in children with IGE (39 points) was descriptively close to the mean reported for the Polish general school-age population (40.8 ± 11.7) [11], whereas children with other epilepsies had a median of 44 points. Because no control group was recruited, this comparison is descriptive only and was not tested statistically. This is consistent with the clinical picture of IGE, which includes normal development, no intellectual disability, and a generally good response to treatment [13]. In other studies, developmental delay, intellectual disability, and drug resistance were associated with sleep disturbances [4,6].

In contrast to our results, Furones García et al. found more sleep disturbances in generalized than in focal epilepsy [6]. However, 43.1% of their patients had an epileptic encephalopathy and 51.6% had intellectual disability. Therefore, their "generalized epilepsy" group probably included many children with severe genetic generalized epilepsies. In our cohort, other non-idiopathic generalized epilepsies had total and SWTD scores closer to those of focal epilepsies than to IGE. These observations suggest that distinguishing IGE from other generalized epilepsies may be informative when investigating sleep symptoms. Broad categories and a smaller sample may also explain why Zambrelli et al. found no differences by epilepsy type or etiology [7].

The lower total score in IGE should, however, be interpreted with caution. Unlike the SWTD difference, it was not explained by age: it persisted after adjustment for age and sex ($\beta = 5.6$; 95% CI 0.24 to 11.0), also when age was modeled flexibly. However, the effect was small (about 0.5 normative SD; $R^2 < 5\%$), and it was not significant in children aged 6–15 years ($p = 0.059$), in the rank-based model ($p = 0.068$), or when the 19 incomplete questionnaires were excluded ($p = 0.19$). This result therefore indicates a possible tendency rather than a definitive difference between epilepsy groups. Age was nevertheless an important potential confounder because the groups differed substantially in their age distributions.

Children with IGE were, on average, 5 years older. This reflects the composition of the IGE group: most children had JME (n = 17) or JAE (n = 6), syndromes that typically begin around or after the age of 10 [13], and the department treats patients up to 18 years of age. SWTD was the only domain strongly associated with age ($\rho = -0.35$). The SWTD subscale covers hypnic jerks, rhythmic movements, hypnagogic hallucinations, nocturnal hyperkinesia, sleep talking, and bruxism [10,11], and several of these phenomena are more common in younger children. After adjustment for age, SWTD scores no longer differed between IGE and other or focal epilepsies, and this did not depend on whether age was modeled linearly or flexibly. Therefore, the age-related explanation applies to the SWTD differences, but not to the total score, and our data do not show a specific association between epilepsy type and SWTD, although higher SWTD scores in children with epilepsy have been reported previously [7,12].

A clinical explanation remains possible and should be tested in age-matched groups. Sleep hypermotor epilepsy, self-limited epilepsy with centrotemporal spikes, and Panayiotopoulos syndrome are sleep-associated epilepsies. Furthermore, minor motor events and parasomnias share an arousal-related mechanism with sleep-related seizures [1,3]. In contrast, IGEs such as JME and GTCA are "awakening epilepsies" [1,13]. In children with focal epilepsy and high SWTD scores, video-EEG or video-polysomnography can help distinguish parasomnias from nocturnal seizures [1,3].

DOES scores did not differ between groups. Excessive daytime sleepiness is the most common sleep complaint in epilepsy (28–48%) [3]. It is associated with ASMs, nocturnal seizures, sleep apnea, and anxiety [3,4], which are present in all epilepsy groups. Additionally, in adolescents with IGE, the physiological increase in daytime sleepiness with age [11] may contribute. Because sleep deprivation is a major seizure trigger in JME and GTCA [1,13], children with IGE still require assessment of daytime sleepiness and sleep hygiene education.

4.2. Structural Epilepsy and Difficulties Initiating and Maintaining Sleep

The higher DIMS scores in structural epilepsy (n = 18) were the most consistent domain finding of the study, although they were just above the threshold of significance after FDR correction ($q = 0.053$). Structural epilepsy differed from both IGE and genetic epilepsy, and this difference remained significant after adjustment for age and sex, in children aged

6–15 years, and after excluding children with missing DIMS items. Increased wake after sleep onset is the most consistent polysomnographic feature of epilepsy and results from the awakening effect of generalized and focal seizures, including subtle ones [1]. Nocturnal seizures also shorten REM sleep and reduce sleep efficiency [3]. Furthermore, structural epilepsies are more often drug-resistant and treated with polytherapy. Drug resistance, nocturnal seizures, intellectual disability, and many ASMs are associated with insomnia or altered sleep architecture [3,4,6]. Poorer subjective sleep quality has also been described in specific lesions, such as hippocampal sclerosis [4]. Drug resistance and comorbidities may therefore mediate part of the association between structural etiology and sleep complaints: brain lesions predispose to drug-resistant epilepsy, which leads to more frequent nocturnal seizures and to polytherapy, and these factors, together with intellectual disability and psychiatric comorbidity, can disrupt sleep [3,4,6]. Because these factors were not recorded, we could not test this pathway. The genetic group was small and heterogeneous. Some genetic syndromes, such as Dravet syndrome and tuberous sclerosis complex, are associated with marked insomnia [4], whereas others may have little effect on sleep. The observed DIMS pattern supports further investigation of sleep initiation and maintenance complaints in structural epilepsy. Elevated questionnaire scores should prompt clinical assessment of the reported symptoms and their possible causes, rather than be interpreted as a diagnosis of insomnia.

4.3. Sleep Hyperhidrosis in Epileptic Encephalopathies

Children with epileptic encephalopathies (n = 15) had higher SHY scores (median 4 vs. 2 in IGE), although this difference was at the threshold of significance after FDR correction (q = 0.047). Notably, in contrast to the SWTD differences, this finding was independent of age and was not affected by missing items. Night sweating may reflect autonomic dysregulation. Sleep instability in epilepsy increases sympathetic tone during sleep [1], and nocturnal seizures are often accompanied by autonomic activation [3]. In the general population, SHY scores decrease with age, which has been attributed to maturation of the autonomic nervous system [10,11]. Delayed maturation in children with developmental encephalopathy could therefore contribute to night sweating. However, the mechanism underlying this observation is uncertain. No autonomic measurements were collected, and the findings provide no direct evidence of altered autonomic maturation. Other potential contributors include ASMs, sleep-related breathing problems, reduced mobility, environmental conditions, and seizure-related phenomena; their contributions were not evaluated in this study. The SHY result should therefore be interpreted as a difference in reported nocturnal sweating that requires clinical characterization and independent confirmation. Moreover, the group was small (n = 15) and combined DEE and EE of different causes, so this finding requires confirmation in larger studies.

4.4. Clinical Relevance

Statistical significance alone does not establish clinical relevance, and no minimal clinically important difference has been defined for the SDSC. Relative to the Polish normative data [11], the adjusted DIMS and SHY differences corresponded to about 1 SD, and scores more than 1 SD above the normative mean were three to six times more frequent in structural epilepsy (DIMS) and epileptic encephalopathies (SHY) than in IGE. However, the median DIMS score in structural epilepsy was only moderately above the normative mean, and pathological scores according to the Polish cut-offs were frequent only for SHY in epileptic encephalopathies (27%). The differences therefore concern a subgroup of children with elevated scores rather than the whole group, and these comparisons with normative data are descriptive. A single question about sleep quality misses most sleep disturbances

in children with epilepsy [6], so a structured tool such as the SDSC, interpreted at the domain level and with age in mind, can help identify children who need a more detailed sleep assessment. However, an elevated SDSC score should prompt a targeted sleep history and, when indicated, objective assessment such as video-polysomnography; it is not sufficient to diagnose a sleep disorder, because questionnaire reports may differ from objective findings [19].

4.5. Strengths and Limitations

The strengths of this study include the classification of epilepsy according to the current ILAE definition of IGE [13], the analysis of the data by epilepsy type, etiology, and category, the adjustment of the domain comparisons for age, and the assessment of the robustness of the findings in several sensitivity analyses.

Certain limitations should be acknowledged in this study. First, it was a single-center study, and some groups were small (structural, n = 18; encephalopathies, n = 15). Second, excluding children with unknown or missing etiology (n = 50) from stage 2 may have introduced selection bias, and the "other generalized epilepsies" and encephalopathy groups were clinically heterogeneous. Moreover, we could not account for important clinical covariates, including seizure frequency and timing, nocturnal seizures, drug resistance, the number and type of ASMs, intellectual disability, and psychiatric comorbidity [4,6,7]. These factors are more common in structural epilepsies and epileptic encephalopathies than in IGE and are themselves associated with sleep disturbances. They may therefore confound or mediate the observed differences, which could partly reflect disease severity and treatment burden rather than epilepsy type itself. The low R^2 values indicate that the models explained only a small proportion of the variability in SDSC scores. Residual confounding by age cannot be excluded. Multiple testing is an additional limitation: six SDSC domains were examined across three overlapping analytical stages, followed by pairwise comparisons and selectively fitted regression models. Benjamini–Hochberg correction controlled the FDR across domains within each stage, and Holm correction controlled multiplicity within the pairwise comparisons for each outcome; no correction was applied across stages or regression models. Across all 18 domain comparisons, no difference remained significant (lowest q = 0.066). Selecting domains for covariate adjustment on the basis of unadjusted significance also limits the confirmatory value of those models. Accordingly, the domain findings may include chance associations and require independent replication.

Additionally, the SDSC is a parent-report questionnaire and is subject to recall bias and to parents' limited awareness of night-time events. Parent reports may differ from objective measures; for example, parents overestimate total sleep time compared with polysomnography [19]. Some items were missing, and summing the answered items underestimates the scores of incomplete questionnaires. The results did not change with prorated scores, and all domain results remained significant when children with missing items in a given domain were excluded; however, the differences in total score involving IGE were no longer significant after excluding the incomplete questionnaires. Furthermore, the results were not confirmed by objective methods, such as polysomnography or actigraphy. Twenty-eight children (22%), including 13 of the 31 with IGE, were outside the 6–15-year age range for which the Polish SDSC was validated [11], although the key findings were similar when they were excluded. Finally, there was no healthy control group, so the comparison with Polish normative data [11] is only descriptive and was not used for statistical inference.

5. Conclusions

In this exploratory single-center study, the severity and profile of parent-reported sleep disturbances may differ between epilepsy phenotypes. Children with IGE tended

to have milder sleep disturbances overall, but this difference was small and not robust in all sensitivity analyses, and their lower sleep–wake transition scores were explained by their older age. Higher DIMS scores in structural epilepsy and higher SHY scores in epileptic encephalopathies persisted after adjustment for age and sex but were at or near the threshold of significance after correction for multiple comparisons and were based on small groups. These findings should be regarded as hypotheses to be confirmed in larger, prospective, multicenter studies with age-matched groups, objective sleep measures, and clinical data such as seizure frequency, treatment, and comorbidities. SDSC scores can help identify children who need a detailed sleep assessment, but they should be interpreted with age in mind and should not be used alone to diagnose sleep disorders.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/brainsci16111162/s1, Table S1: Age- and sex-adjusted group differences in all SDSC scores (β, 95% CI; p); Table S2: Regression model diagnostics.

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Informed Consent Statement: Oral informed consent was obtained from the parents or legal guardians of all participants before completion of the questionnaire. Separate written informed consent was not required because the study was a questionnaire-based study without the characteristics of a medical experiment, which, according to Polish law and the statement of the Bioethics Committee of Poznan University of Medical Sciences (no. KB-683/26), was not subject to review by the Bioethics Committee.

Data Availability Statement: The data presented in this study are available on request from the corresponding author due to privacy and ethical restrictions, as the dataset contains clinical information on pediatric patients.

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Abbreviations

The following abbreviations are used in this manuscript:

ASM	Antiseizure medication
CAE	Childhood absence epilepsy
DA	Disorders of arousal
DEE	Developmental and epileptic encephalopathy
DIMS	Disorders of initiating and maintaining sleep
DOES	Disorders of excessive somnolence

EE	Epileptic encephalopathy
FDR	False discovery rate
GTCA	Epilepsy with generalized tonic–clonic seizures alone
IGE	Idiopathic generalized epilepsy
HC3	Heteroscedasticity-consistent standard errors
ILAE	International League Against Epilepsy
JAE	Juvenile absence epilepsy
JME	Juvenile myoclonic epilepsy
SBD	Sleep breathing disorders
SDSC	Sleep Disturbance Scale for Children
SHY	Sleep hyperhidrosis
SWTD	Sleep–wake transition disorders

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