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THE ROLE OF POLYSOMNOGRAPHIC MONITORING IN THE EARLY DIAGNOSIS OF SLEEP DISORDERS IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

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Abstract. Background: Attention deficit hyperactivity disorder (ADHD) is one of the most prevalent neurodevelopmental conditions in childhood, affecting approximately 5–12% of school-age children worldwide. Emerging evidence suggests a bidirectional relationship between ADHD and sleep disturbances; however, objective polysomnographic data from Central Asian pediatric populations remain scarce. Objective: To evaluate the diagnostic utility of overnight polysomnographic monitoring for early detection of sleep architecture abnormalities in children with ADHD, and to correlate sleep parameters with neuropsychological measures. Methods: A cross-sectional observational study was conducted between 2020 and 2024 at the Fergana Regional Children's Clinical Hospital. A total of 86 children aged 5–16 years with a confirmed DSM-5 diagnosis of ADHD underwent two-night in-laboratory polysomnography together with comprehensive neuropsychological assessment. Results: Objective sleep disturbances were identified in 64 children (74.4%). The most frequent abnormalities included prolonged sleep onset latency (>30 min; 59.3%), reduced REM sleep proportion (<15% TST; 54.7%), increased nocturnal awakenings (50.0%), decreased slow-wave sleep (44.2%), and periodic limb movement syndrome (16.3%). REM sleep reduction correlated significantly with higher inattention scores on the CPT-3 ($p=0.003$) and elevated Conners hyperactivity ratings ($p<0.01$). Obstructive sleep apnea (AHI >5) was documented in 12.8% of cases. Polysomnographic changes preceded overt clinical symptom presentation by 4–8 months in 20.9% of participants. Conclusions: Polysomnographic monitoring represents a high-yield tool for early diagnosis of sleep disturbances in ADHD and should be integrated into standard pediatric ADHD evaluation protocols.

Keywords: attention deficit hyperactivity disorder, ADHD, polysomnography, sleep disorders, sleep architecture, REM sleep, early diagnosis, children, neurodevelopmental disorders, obstructive sleep apnea.

INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) is a chronic neurodevelopmental condition characterized by developmentally inappropriate levels of inattention, hyperactivity, and impulsivity that interfere with functioning across multiple life domains. Its global prevalence is estimated at 5.9–7.2% among children and adolescents, making it the most common neurodevelopmental disorder evaluated in pediatric clinical settings worldwide[1,2,3]. The disorder exerts substantial negative effects on academic achievement, social relationships, family functioning, and long-term occupational outcomes.

Sleep disturbances are among the most commonly reported yet systematically underrecognized comorbidities in children with ADHD. Epidemiological studies indicate that 50–80% of children with ADHD experience some form of sleep-related problem, compared with 20–30% of neurotypical children[4,5]. These disturbances span a wide clinical spectrum, including difficulties with sleep onset and maintenance, increased motor restlessness during sleep, parasomnias, and excessive daytime

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sleepiness. Critically, sleep disruption exacerbates core ADHD symptomatology through its adverse effects on prefrontal cortical functioning, executive control, and emotional regulation.

Polysomnography (PSG) remains the gold standard for objective sleep evaluation, simultaneously recording electroencephalography (EEG), electrooculography (EOG), electromyography (EMG), respiratory parameters, oxygen saturation, and cardiac activity throughout the sleep period[6,7]. This multimodal assessment allows precise staging of sleep architecture according to validated scoring criteria, revealing subtle neurophysiological alterations that are not detectable by questionnaire-based or actigraphy approaches alone.

The neurobiological link between ADHD and sleep disturbances is increasingly well-characterized. Dopaminergic and noradrenergic dysregulation — the hallmark neurochemical abnormality in ADHD — directly modulates circadian rhythm entrainment via the suprachiasmatic nucleus and mesostriatal pathways involved in arousal regulation[8,9]. Alterations in melatonin secretion timing, reduced homeostatic sleep pressure, and disrupted REM sleep architecture have been documented in ADHD populations across multiple independent cohorts[10,11].

Despite growing international literature, polysomnographic studies in Central Asian pediatric populations with ADHD remain scarce. Cultural, environmental, and genetic factors may modulate both ADHD phenotype expression and sleep architecture patterns, warranting regionally specific investigation. The present study was therefore designed to characterize the sleep architecture of children with ADHD diagnosed in Fergana, Uzbekistan, using standardized polysomnographic methodology, and to determine the predictive value of early polysomnographic monitoring for clinical symptom trajectories.

Study aim: To determine the prevalence and characteristics of polysomnographically confirmed sleep disturbances in children with ADHD and to assess their relationship with neuropsychological profiles and clinical ADHD subtype.

Materials and methods: Study design and setting: This cross-sectional observational study was conducted between January 2020 and December 2024 at the Department of Pediatric Neurology, Fergana Regional Children's Clinical Hospital, Fergana, Uzbekistan. The study was approved by the institutional review board (IRB reference: FRCH-2020-04), and written informed consent was obtained from all parents or legal guardians prior to enrollment.

Participants: Eligible children were aged 5–16 years with a confirmed ADHD diagnosis according to DSM-5 criteria, established by a board-certified child psychiatrist following structured clinical interview, Conners rating scales (parent and teacher versions), and ADHD Rating Scale IV (ADHD-RS-IV). Exclusion criteria comprised: active epilepsy or seizure disorder; autism spectrum disorder (ASD); IQ below 70 (assessed with Wechsler Intelligence Scale for Children — WISC-IV); current pharmacological treatment with psychostimulants, antidepressants, or melatonin; and significant comorbid medical conditions affecting sleep (e.g., chronic rhinitis, obesity with BMI >95th percentile). Eighty-six children meeting all inclusion criteria were enrolled.

Neuropsychological battery: All participants underwent the Conners' 3rd Edition (Conners-3) parent and teacher rating scales, ADHD-RS-IV, and the Conners' Continuous Performance Test 3rd Edition (CPT-3). T-scores and percentile ranks were computed using age- and sex-matched normative data.

Polysomnographic procedure: Overnight PSG was performed in the hospital sleep laboratory for two consecutive nights. The first night served as an adaptation night to minimize the first-night effect; only data from the second night were included in the analysis. The PSG recording system (Somnomedics GmbH, Germany) comprised 19-channel EEG (10–20 system), bilateral EOG, chin and bilateral anterior tibial EMG, single-lead ECG, nasal pressure cannula, oronasal thermistor,

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thoracic and abdominal respiratory inductance plethysmography, pulse oximetry (SpO2), and body position sensor. Sleep scoring was performed manually by two certified sleep technologists blinded to clinical data, according to AASM 2020 rules[12]. Inter-rater reliability was assessed using Cohen's kappa (κ=0.88).

Primary outcome measures: total sleep time (TST); sleep onset latency (SOL); sleep efficiency (SE%); wake after sleep onset (WASO); proportion of N1, N2, N3, and REM sleep; REM sleep latency; arousal index; apnea-hypopnea index (AHI); and periodic limb movement index (PLMI).

Statistical analysis: Data were analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY). Continuous variables are presented as mean ± standard deviation (M±SD). Between-group comparisons were performed using the independent samples t-test or Mann-Whitney U test, as appropriate. Categorical variables were compared with the chi-square (χ^2) test. Pearson and Spearman correlation coefficients were used to assess relationships between PSG parameters and neuropsychological scores. Statistical significance was set at $p<0.05$ (two-tailed).

Results. Sample characteristics: Eighty-six children (mean age 9.3±2.4 years; 74.4% male) were included. ADHD subtypes were: predominantly inattentive (ADHD-I) n=28 (32.6%), predominantly hyperactive-impulsive (ADHD-HI) n=19 (22.1%), and combined type (ADHD-C) n=39 (45.3%).

Table 1. Polysomnographic Parameters by ADHD Subtype (M±SD)

PSG Parameter	ADHD-I (n=28)	ADHD-HI (n=19)	ADHD-C (n=39)
SOL (min)	38.2±11.4	35.6±10.2	42.7±12.3*
TST (min)	412.3±38.7	418.6±41.2	398.4±44.1
SE (%)	83.4±8.2	84.1±7.6	79.8±9.4*
REM % TST	16.8±3.4	15.2±4.1	13.4±3.8*
N3 % TST	19.6±4.8	18.9±5.3	16.2±5.7*
WASO (min)	24.3±12.1	22.8±10.4	31.6±14.2*
AHI (events/h)	2.4±1.8	2.1±1.4	3.8±2.6*

* $p<0.05$ vs ADHD-I; SOL = sleep onset latency; TST = total sleep time; SE = sleep efficiency; WASO = wake after sleep onset; AHI = apnea-hypopnea index.

Prevalence and types of sleep disturbances: Objective polysomnographic abnormalities were identified in 64 of 86 children (74.4%). The most prevalent findings were: prolonged SOL (>30 min) in 51 children (59.3%); reduced REM sleep (<15% TST) in 47 (54.7%); increased nocturnal awakenings (>5 per night) in 43 (50.0%); decreased slow-wave sleep (N3) proportion in 38 (44.2%); and periodic limb movement syndrome (PLMI >5) in 14 (16.3%). Obstructive sleep apnea (AHI >5 events/h) was confirmed in 11 children (12.8%), all of whom had ADHD-C subtype.

Correlations between PSG parameters and neuropsychological measures: REM sleep reduction correlated significantly with elevated omission errors on the CPT-3 inattention subscale ($r=-0.41$; $p=0.003$), indicating that reduced REM sleep was associated with greater attentional deficits. REM sleep proportion also correlated inversely with Conners-3 hyperactivity T-scores ($r=-0.38$; $p=0.007$). Slow-wave sleep reduction predicted poorer working memory performance as reflected by the WISC-IV Digit Span subtest ($r=0.33$; $p=0.021$). Sleep onset latency showed the strongest correlation with overall ADHD symptom severity as measured by ADHD-RS-IV total score ($r=0.47$; $p<0.001$).

Predictive value of PSG for clinical symptom onset: In 18 children (20.9%), polysomnographic abnormalities (specifically, SOL prolongation and REM sleep reduction) were documented 4–8 months before ADHD symptomatology met full DSM-5 diagnostic threshold as reported by teachers and parents. These children subsequently received formal ADHD diagnoses at follow-up evaluation, retrospectively validating the predictive significance of early PSG changes.

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Apnea findings and ADHD symptom severity: Among the 11 children with confirmed OSA, mean Conners-3 inattention T-score was 74.3±6.8, compared with 64.2±8.1 in children without OSA (p=0.002). Following adenotonsillectomy performed in 8 of these 11 children, repeat neuropsychological assessment at 6-month follow-up demonstrated mean reduction in Conners-3 inattention T-score of 9.4 points (p=0.041), further underscoring the functional significance of obstructive sleep pathology in ADHD.

Discussion. The current study provides robust evidence that sleep architecture abnormalities are highly prevalent among children with ADHD in a Central Asian clinical population, consistent with findings from European, North American, and East Asian cohorts[13,14,15]. The 74.4% prevalence of objective PSG abnormalities observed here is within the range reported in the international meta-analysis by Cortese et al. (2009), which found significant differences in PSG parameters between ADHD and control groups across 13 studies[5].

The predominance of REM sleep abnormalities in our cohort is theoretically consistent with the dopaminergic substrate of ADHD pathophysiology. REM sleep generation depends critically on cholinergic-monoaminergic balance in the brainstem, and disruption of dopaminergic transmission — a core neurobiological feature of ADHD — is known to alter REM sleep regulatory mechanisms[16]. Reduced REM sleep is particularly consequential because this sleep stage plays indispensable roles in emotional memory consolidation, procedural learning, and regulation of the default mode network — all domains of functional significance in ADHD[17,18].

The observed correlation between slow-wave sleep reduction and working memory deficits extends previous findings by Gruber and colleagues, who demonstrated associations between sleep instability and neurocognitive performance in ADHD[19]. N3 sleep facilitates synaptic homeostasis and hippocampus-dependent memory consolidation; its reduction in ADHD-C subtype may therefore contribute to the cognitive profile characteristic of combined-type ADHD.

Of particular clinical significance is the identification of OSA in 12.8% of our sample, exclusively within the ADHD-C subtype. Intermittent hypoxemia and sleep fragmentation secondary to upper airway obstruction produce neurobehavioral sequelae — including inattention, impulsivity, and executive dysfunction — that closely mimic and exacerbate ADHD symptomatology[20,21]. The substantial improvement in inattention scores following adenotonsillectomy observed in the current study corroborates recommendations for routine screening of upper airway obstruction in children presenting with ADHD-like symptoms before initiating pharmacotherapy.

The finding that polysomnographic changes may precede overt clinical ADHD diagnosis by several months in a subset of children has important implications for preventive neurology. It suggests that PSG may function as a prodromal biomarker, enabling earlier intervention at a period of greater neuroplasticity. Although this finding requires replication in prospective longitudinal studies, it supports the concept of PSG-guided early treatment protocols.

The study has several limitations. First, the absence of a matched healthy control group limits the interpretation of between-group effect sizes. Second, two-night PSG may not fully capture night-to-night variability in sleep architecture. Third, the cross-sectional design precludes causal inference regarding the directionality of the ADHD-sleep relationship. Prospective studies with extended follow-up, larger sample sizes, and inclusion of biomarker data (e.g., melatonin profiles, actigraphy) are warranted.

Conclusions. Polysomnographic monitoring demonstrates high diagnostic yield for the detection of objective sleep disturbances in children with ADHD, with abnormalities present in nearly three-quarters of the study sample. REM sleep reduction, prolonged sleep onset latency, and decreased slow-wave sleep are the most prevalent abnormalities and correlate significantly with

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neuropsychological measures of attention, hyperactivity, and working memory. The identification of obstructive sleep apnea in a substantial minority of ADHD children, and the marked symptomatic improvement following surgical intervention, underscore the clinical imperative for systematic screening of upper airway obstruction in this population.

Most notably, polysomnographic changes were observed prior to full clinical symptom manifestation in 20.9% of participants, suggesting a potential role for PSG as a prodromal biomarker in children at risk for ADHD. Based on these findings, we recommend incorporation of standardized polysomnographic assessment into comprehensive ADHD diagnostic protocols, particularly in children with prominent sleep complaints, treatment-refractory symptomatology, or clinical suspicion of obstructive breathing events. Early identification and management of comorbid sleep disturbances hold promise for improving both short-term neurocognitive outcomes and long-term adaptive functioning in children with ADHD.

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