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CLINICAL AND ANATOMICAL FACTORS ASSOCIATED WITH OBSTRUCTIVE SLEEP APNEA SEVERITY IN CHILDREN: A PROSPECTIVE PRELIMINARY STUDY

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Abstract

Introduction: Pediatric obstructive sleep apnea (OSA) is a multifactorial disorder characterized by recurrent upper airway obstruction during sleep, leading to intermittent hypoxia and sleep fragmentation. Although adenotonsillar hypertrophy is the primary anatomical risk factor, disease severity may be influenced by additional variables including age, obesity, and allergic status.

Objective: To evaluate the association between adenotonsillar hypertrophy, age, body mass index (BMI), allergic status, and OSA severity measured by the apnea–hypopnea index (AHI).

Methods: A prospective analytical study was conducted in a preliminary cohort of 26 children (3–14 years) with suspected OSA. Participants underwent overnight cardiorespiratory polygraphy, otorhinolaryngological assessment (Brodsky tonsillar grading and endoscopic adenoid grading), anthropometric evaluation, allergy history assessment, and completion of the Pediatric Sleep Questionnaire (PSQ). Associations between clinical variables and AHI were analyzed using nonparametric statistical methods.

Results: Mild OSA predominated (88.5%). No significant associations were observed between AHI and adenotonsillar hypertrophy grade, age, BMI, or allergic status. A significant positive correlation was identified between PSQ total score and AHI ($\rho = 0.443$; $p = 0.023$).

Conclusion: In this preliminary cohort, OSA severity was not significantly associated with anatomical hypertrophy, age, obesity, or allergic status. Symptom burden assessed by PSQ correlated with objective disease severity, supporting its value as a screening tool. Larger studies are required to clarify the multifactorial determinants of pediatric OSA severity.

Keywords: pediatric OSA, adenotonsillar hypertrophy, BMI, allergy, AHI.

Introduction

Sleep-disordered breathing (SDB) in children is a term that refers to a spectrum of breathing changes ranging from simple snoring and mouth breathing to a serious condition such as Obstructive Sleep Apnea (OSA). OSA in children is characterized by recurrent episodes of partial or complete upper airway obstruction, disrupting breathing (intermittent hypoxia and intermittent hypercapnia) as well as normal sleep patterns (sleep fragmentation). These physiological disturbances can lead to serious consequences for the child's cognitive development, behavior, growth, and cardiometabolic health [1–4].

The pathophysiology of OSA in children is complex, involving the interaction of airway collapsibility and neuromuscular compensation. The most common cause in children is adenotonsillar hypertrophy, which causes mechanical obstruction in the nasopharynx and oropharynx. Additional contributing factors include obesity, allergic rhinitis, chronic nasal congestion, and inflammatory changes leading to lymphoid tissue hyperplasia [5–8].

The prevalence of OSA in the general pediatric population is estimated to be 1–5%, with a peak between 3 and 8 years of age—a period that coincides with the increase in size of the adenoids and tonsils. Although adenotonsillar hypertrophy is the primary anatomic factor, the severity of OSA does not always correlate directly with its degree. Current studies suggest that the combination of multiple factors—age,

body mass index (BMI), and the presence of allergic conditions—has a synergistic effect on the severity of the disorder [9–12].

In clinical practice, the diagnosis of OSA is made on the basis of clinical symptoms and instrumental confirmation with polysomnography, which is the gold standard. However, in many resource-limited settings, cardiorespiratory polygraphy is used as a practical alternative, providing valid parameters for assessing the apnea-hypopnea index (AHI) [13–17].

Despite extensive research on adenotonsillar hypertrophy, the relative contribution of anatomical, anthropometric, and allergic factors to objective OSA severity remains incompletely defined, particularly in smaller prospective pediatric cohorts.

Given that adenotonsillar hypertrophy remains the leading cause of obstructive sleep apnea in childhood, it is of particular importance to investigate factors that modify its severity—particularly age, body weight, and allergic status. Such data are essential for accurate clinical guidance, risk assessment, and timely therapeutic management.

We hypothesized that adenotonsillar hypertrophy alone would not independently predict OSA severity, and that additional factors such as BMI and allergic status would contribute to disease variability.

Objective

Main objective: To assess the association between the degree of adenotonsillar hypertrophy, age, body mass index (BMI) and allergic status with the severity of obstructive sleep apnea (OSA), measured by the apnea-hypopnea index (AHI) in the Macedonian pediatric population.

Specific objectives:

1. To analyze the distribution of OSA by severity (mild, moderate, severe) in relation to adenoid and tonsillar extent.
2. To examine the association of AHI with age and BMI categories (normal weight, overweight, obesity).
3. To assess the impact of allergic conditions (allergic rhinitis, asthma, positive SPT) on the severity of OSA.

Material and methods

Study Design

This study presents preliminary findings from an ongoing prospective doctoral research project designed to evaluate determinants of pediatric obstructive sleep apnea (OSA) severity. The study is conducted at the University Clinic for Respiratory Diseases in Children Kozle, Skopje. Participants were consecutively recruited among children referred for evaluation of suspected sleep-disordered breathing. The present report includes the first 26 enrolled subjects.

The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from parents or legal guardians prior to participation.

Subjects

The study included children aged 3 to 14 years, who were referred for assessment due to suspected sleep-disordered breathing.

Inclusion criteria were: presence of snoring or symptoms of nocturnal upper airway obstruction.

Exclusion criteria: Children with craniofacial anomalies, neuromuscular disorders, or genetic syndromes were excluded.

This paper is part of a broader study of OSA in children and refers exclusively to the subgroup with adenotonsillar hypertrophy. This subgroup was selected for preliminary analysis due to its high prevalence and characteristic pathophysiological relevance.

Clinical evaluation

All subjects underwent a standard history and physical examination. Demographic data (age, sex), anthropometric measurements (height, weight), and body mass index (BMI) were recorded. BMI categories were determined according to CDC percentile charts [18].

Allergic status was assessed through clinical history and confirmed by skin prick testing (SPT). Data regarding allergic rhinitis, asthma, recurrent upper respiratory infections, and family history of OSA were collected.

Tonsil size was assessed according to the Brodsky scale (grade I–IV), and adenoid obstruction was assessed endoscopically (grade I–IV according to percentage of nasopharyngeal obstruction) [19–22]. Based on the findings, the subjects were divided into four groups: without hypertrophy, with adenoid, with tonsillar, and with combined adenotonsillar hypertrophy.

Parents answered questions to assess symptoms of obstructive apnea, daytime sleepiness, and behavior. The analysis included the Pediatric Sleep Questionnaire (PSQ), which is used as a preliminary screening tool and has been adapted for the needs of this study. PSQ results were analyzed as a total score and by domains ('snoring/breathing problems, daytime sleepiness, behavior/attention') [23–25].

Cardiorespiratory polygraphy

The subjects underwent overnight cardiorespiratory polygraphy on the ALICE Sleepware G3 system (Philips Respironics, USA), with monitoring of airflow, oxygen saturation, pulse, thoracic and abdominal movements [25].

The apnea-hypopnea index (AHI) was calculated as the number of apnea and hypopnea episodes per hour of sleep. OSA severity was classified as: Normal: AHI < 1 event/hour, Mild ($1 < AHI \leq 5$), Moderate ($5 < AHI \leq 10$), and Severe (AHI > 10).

Statistical analysis

Statistical analysis was performed using the IBM SPSS Statistics program, version 28.0.

Descriptive statistics were used to present demographic and clinical data, with continuous variables expressed as mean ± standard deviation and categorical variables as number and percentage. Normality was assessed using the Shapiro–Wilk test. Spearman correlation analysis was used to assess the relationship between continuous parameters (e.g., age, BMI, PSQ score, and apnea-hypopnea index – AHI).

For comparison of categorical data, such as the degree of adenotonsillar hypertrophy, gender, and allergic status in relation to OSA severity, the χ^2 test (Chi-square) or Fisher's exact test was used when the number of cases was small. The level of statistical significance was set at $p < 0.05$.

Due to the exploratory nature of this preliminary analysis, no formal sample size calculation was performed. The present report represents the initial phase of an ongoing prospective doctoral cohort, with continued recruitment planned for future multivariate modeling.

Results

Demographic and anthropometric characteristics

A total of 26 children aged 3 to 14 years were included in the study (mean age 6.41 ± 2.49, median 6.55 years). According to the age distribution, preschool children (3–6 years) were represented by 10 (38.5%), and school children (6–12 years) by 16 (61.5%), while there were no adolescents in the sample. In terms of gender, males dominated with 18 children (69.2%), compared to 8 females (30.8%).

The average BMI was 17.61 kg/m² (median 16.49). According to BMI categories (according to CDC percentiles), the majority of children had normal body weight (n=9; 34.6%), followed by underweight (n=7; 26.9%), overweight (n=6; 23.1%), and obese (n=4; 15.4%). Overall, 38.5% of the sample had an elevated BMI (above the 85th percentile).

Allergic rhinitis was present in 50% (n=13), asthma in 19.2% (n=5), and nasal obstruction in 84.6% (n=22). A positive family history of snoring/OSA was reported in 30.8% (n=8).

Table 1. Demographics and baseline clinical characteristics Total (N=26).

Parameter	Average value ± SD / n (%)
Age (years)	6.41 ± 2.49
Age group 3-6 years 6-12 years 12-14 years	 10 (38.5%) 16 (61.5%) 0 (0 %)
Gender Male Female	 18 (69.2%) 8 (30.8%)
BMI (kg/m²)	17.61 ± 3.77
BMI category Underweight Normal weight Overweight Obesity	 7 (26.9 %) 9 (34.6 %) 6 (23.1 %) 4 (15.4 %)
Family history of snoring/OSA	8 (30.8 %)
Allergic rhinitis	13 (50%)
Asthma	5 (19.2%)
Recurrent Upper Respiratory Infections/Otitis	10 (38.5 %)
Nasal obstruction	22 (84.6 %)

ENT findings

Adenoid and/or tonsillar hypertrophy were common in the sample. Tonsillar hypertrophy (Grade III–IV) was found in 7.7% of children. Adenoid hypertrophy (Grade III–IV) in 23.1%. Combined adenotonsillar hypertrophy in 50% of the subjects. Only 19.2% had normal ENT findings.

Table 2. Distribution according to ORL findings.

Group	n (%)
No hypertrophy	5 (19.2 %)
Tonsillar hypertrophy only	2 (7.7%)
Adenoid hypertrophy only	6 (23.1%)
Combined adenotonsillar hypertrophy	13 (50%)

Pediatric sleep questionnaire (PSQ)

The mean PSQ total score was 10.69 ± 2.41 (median 10.5). A positive screening result (PSQ_total ≥ 8) was observed in 80.8% of participants (n=21).

Polysomnographic findings (OSA severity)

According to the Apnea–Hypopnea Index (AHI), the distribution of OSA severity was as follows: normal in 1 child (3.8%), mild in 23 (88.5%), moderate in 2 (7.7%). Overall, mild obstructive sleep apnea was the predominant form in this population. Mean AHI = 2.88/hour, median 2.65/hour

The predominance of mild OSA limited variability across severity strata.

Table 3. Distribution by severity of OSA.

Category	n (%)
No OSA (AHI<1)	1 (3.8 %)
Mild OSA (1<AHI≤5)	23 (88.5 %)
Moderate (5<AHI≤10)	2 (7.7%)
Severe (AHI>10)	0 (0%)

Correlation analysis

Spearman analysis demonstrated a moderate positive correlation between PSQ total score and AHI ($\rho = 0.443$, $p = 0.023$). No significant correlations were observed between AHI and age ($\rho = -0.191$, $p = 0.35$) or BMI ($\rho = 0.130$, $p = 0.53$).

Analyses of categorical variables (association with OSA severity)

χ^2, Fisher and Kruskal–Wallis analyses showed no statistically significant differences in AHI by: hypertrophy group, adenoid or tonsillar extent, BMI category, SPT status, allergic rhinitis or asthma. (All $p > 0.05$) Differences between groups were minimal, which is partly due to the small sample size and the predominance of mild cases.

Discussion

This preliminary analysis demonstrated no significant association between adenotonsillar hypertrophy, age, BMI, or allergic status and objective OSA severity measured by AHI. In contrast, symptom burden assessed using the PSQ showed a moderate and statistically significant correlation with disease severity.

Although adenotonsillar hypertrophy remains the principal etiological factor in pediatric OSA, accumulating evidence indicates that anatomical size alone does not reliably predict physiological severity. Our findings support the concept of pediatric OSA as a multifactorial disorder in which airway collapsibility, neuromuscular control, inflammatory mechanisms, and craniofacial characteristics may influence obstruction beyond static lymphoid tissue enlargement.

Neither BMI nor allergic status was significantly associated with AHI in this cohort. While obesity is an established contributor to OSA in adolescents and adults, its role in younger pediatric populations—particularly in predominantly mild cases—may be less pronounced. Similarly, allergic inflammation may contribute primarily to symptomatic nasal obstruction rather than measurable apnea severity.

The significant association between PSQ score and AHI highlights the clinical utility of structured symptom-based screening tools. In settings where full polysomnography is not readily available, validated questionnaires may assist in identifying children at risk for clinically relevant OSA.

Reliance solely on adenotonsillar size for clinical decision-making may therefore oversimplify the pathophysiological complexity of pediatric OSA. A more integrative assessment incorporating anatomical, functional, and symptom-based parameters may improve risk stratification.

Despite its exploratory nature, this study has methodological strengths, including prospective design, standardized grading of hypertrophy, objective respiratory assessment, and predefined statistical methodology. However, the absence of significant associations should be interpreted cautiously, as the study may be underpowered to detect small-to-moderate effect sizes.

Multivariate modeling was not feasible due to sample size constraints; ongoing recruitment will allow assessment of independent predictors and interaction effects in a larger cohort.

Overall, these findings reinforce the multifactorial nature of pediatric OSA and underscore the need for comprehensive analytic models in future studies.

This study has several limitations. First, the sample size was small and represents preliminary data, limiting statistical power and generalizability. Second, the predominance of mild OSA cases may have reduced the ability to detect associations across severity strata.

Third, the use of cardiorespiratory polygraphy rather than full attended polysomnography may underestimate certain sleep parameters. Finally, this was a single-center study, and multivariate modeling was not feasible given the sample size.

Future research with larger, multicenter cohorts and comprehensive multivariate analyses is warranted to clarify the independent and interactive effects of anatomical and systemic factors on OSA severity.

Conclusion

In this preliminary pediatric cohort, obstructive sleep apnea severity was not significantly associated with adenotonsillar hypertrophy grade, age, BMI, or allergic status. These findings reinforce the concept that pediatric OSA severity cannot be explained solely by anatomical enlargement of lymphoid tissue. Symptom-based screening with the Pediatric Sleep Questionnaire demonstrated meaningful correlation with objective disease burden and may serve as a valuable clinical tool.

These preliminary findings underscore the need for integrative models incorporating anatomical, functional, and inflammatory parameters in future pediatric OSA research.

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