Nature and Science of Sleep

Elevate Series

Open Access Full Text Article

ORIGINAL RESEARCH

Evaluating Positional Obstructive Sleep Apnea in Children: Prevalence, Characteristics, and Risk Factors

Qian Wang[1,2,*], Guimin Huang[3,*], Ruikun Wang[4], Zhilong Cao[5], Jieqiong Liang[1], Mengyao Li[1], Qinglong Gu[1,2]

[1]Department of Otolaryngology-Head and Neck Surgery, Capital Institute of Pediatrics, Beijing, People's Republic of China; [2]Graduate School of Peking Union Medical College, Beijing, People's Republic of China; [3]Child Health Big Data Research Center, Capital Institute of Pediatrics, Beijing, People's Republic of China; [4]Capital Institute of Pediatrics-Peking University Teaching Hospital, Beijing, People's Republic of China; [5]School of Software, Beihang University, Beijing, People's Republic of China

*These authors contributed equally to this work

Correspondence: Qinglong Gu, Department of Otolaryngology-Head and Neck Surgery, Capital Institute of Pediatrics, No. 2 Yabao Road, Chaoyang District, Beijing, 100020, People's Republic of China, Tel +86-13146836613, Email gql71@163.com

Purpose: This study investigates the prevalence, risk factors, and clinical characteristics of positional obstructive sleep apnea (POSA) among pediatric patients diagnosed with obstructive sleep apnea (OSA).

Patients and Methods: A total of 1,236 children aged 0 to 17 years who underwent nocturnal polysomnography (PSG) and completed the Sleep Questionnaire were included. After excluding those with an AHI <1, neurological or muscular disorders, or insufficient sleep time in specific positions, 908 patients remained: 158 with POSA and 750 with non-positional OSA (NPOSA). Propensity score matching (PSM) was applied at a 1:2 ratio, resulting in a final sample of 153 POSA and 306 NPOSA patients. Data analyses were performed using R software (version 4.2.3).

Results: The prevalence of POSA was 12.8%. After PSM, patients with POSA had a lower overall AHI (8.66 vs 10.30), REM-AHI (14.30 vs 17.40), and NREM-AHI (7.43 vs 8.77) compared to those with NPOSA. POSA patients also had a shorter total sleep time (411 vs 427 minutes), spent less time in the supine position (168 vs 225 minutes), and more time in non-supine positions (241 vs 202 minutes) than NPOSA patients. Additionally, while the supine AHI was higher in POSA patients (15.60 vs 10.30), the non-supine AHI was lower (5.00 vs 11.00) compared to NPOSA patients. The minimum oxygen saturation was slightly higher in POSA patients (0.88 vs 0.87). All differences were statistically significant ($P < 0.05$). Risk factors for POSA included mild OSA, allergic rhinitis, non-allergic rhinitis, and obesity.

Conclusion: The prevalence of POSA in children is lower than in adults, and its severity is less than that of NPOSA. Compared to NPOSA patients, POSA patients had significantly higher AHI during supine sleep and lower AHI during non-supine sleep. POSA patients also spent more time in non-supine positions, suggesting that avoiding supine sleep may help reduce apnea events. These findings highlight the importance of monitoring and managing sleep posture in POSA patients.

Keywords: obstructive sleep apnea, pediatric patients, risk factors, polysomnography, sleep position, sleep-disordered breathing

Introduction

Obstructive sleep apnea (OSA) in children is a condition secondary to partial or complete obstruction of the upper airway during sleep. The prevalence of pediatric OSA is between 1.2% and 5.7%.[1–3] Pediatric OSA is characterized by snoring, intermittent nocturnal oxygen desaturation, and sleep disruption.[4] The gold standard for diagnosing OSA in children is nocturnal polysomnography (PSG), which provides comprehensive monitoring of sleep patterns and respiratory function. The most common first-line treatment for pediatric OSA is adenotonsillectomy, a surgical procedure aimed at removing the tonsils and adenoids to alleviate airway obstruction.[2] For patients who are unable to undergo surgery or continue to experience OSA post-surgery, common treatments include continuous positive airway pressure (CPAP) therapy, medication, and other interventions.[5,6] Previous studies suggested that OSA was susceptible to body position, and there was

Received: 25 June 2024
Accepted: 20 September 2024
Published: 3 October 2024

proof that the severity of OSA was particularly worse when supine.[7,8] Sleeping in a supine position not only can result in decreased craniofacial volume[9] and lung volume[10] but also results in the inability of airway dilator muscles to compensate for airway collapsibility during an obstruction.[11] There is a dominant phenotype of OSA: positional obstructive sleep apnea (POSA). Usually, POSA is diagnosed by the criterion that the apnea-hypopnea index (AHI) is at least twice as great in the supine position compared with non-supine positions.[12] Some studies have stated that the prevalence of POSA in adult patients is 61%[13], while in pediatric patients the prevalence is smaller than in adults, at 19% to 48%.[14–16] Studies have shown that positional device therapy is an effective treatment for POSA. This therapy uses a chest strap with a cushion on the back to prevent children from sleeping in a supine position, which is particularly beneficial for children who cannot tolerate continuous positive airway pressure (CPAP) therapy.[16,17] Therefore, it is crucial to distinguish between pediatric POSA and non-positional OSA (NPOSA), as this differentiation can guide the selection of the most appropriate treatment. Properly identifying the type of OSA ensures that children receive the most effective and least invasive interventions tailored to their specific needs.[16,17] Most studies consistently show that POSA is a milder form of OSA in adult patients. POSA has less severe sleep apnea and more preserved sleep architecture than NPOSA.[18–20] In children, however, only a limited number of studies have examined the impact of POSA versus NPOSA on the severity of OSA and the alterations in sleep structures. Various studies indicate that the positional effect on OSA in children could be influenced by factors such as age, BMI, and the site of upper airway obstruction.[21–23] Nonetheless, the precise factors related to POSA in children remain unclear.

To address the current gap in awareness and the lack of guidelines for the diagnosis and treatment of POSA in children, the study aims to (1) investigate the prevalence of POSA, (2) compare the clinical characteristics between POSA and NPOSA, and (3) identify factors associated with POSA. Through these objectives, the study strives to enhance understanding of POSA in children and inform future clinical practices.

Materials and Methods

Study Participants

This cross-sectional study was conducted through the retrospective identification of pediatric patients via electronic medical record retrieval. Between January 2020 and May 2023, we included 1,236 children aged 0 to 17 years who underwent both a sleep questionnaire assessment and overnight polysomnography (PSG) at the Sleep Center of the Children's Hospital affiliated with the Capital Institute of Pediatrics. The data integrity of this study was fully ensured, as all included patients provided complete questionnaire responses and PSG data, with no missing information. Participants were diagnosed with OSA based on an Apnea-Hypopnea Index (AHI) ≥1, confirmed by nocturnal polysomnography (PSG).[24] All subjects were referred to the Sleep Medicine Center of our hospital by pediatricians and otolaryngologists. None of them had a history of craniofacial abnormalities, preterm birth, adenoidectomy, or tonsillectomy.

Exclusion criteria were as follows: (1) individuals with an AHI<1: we excluded children with an Apnea-Hypopnea Index (AHI) of less than 1 because an AHI below this threshold does not meet the clinical criteria for Obstructive Sleep Apnea (OSA). (2) those with neurological or muscular disorders: patients with neurological or muscular disorders were excluded because these conditions could independently affect sleep architecture and respiratory function. Including such individuals could introduce confounding variables, making it difficult to isolate the specific impact of POSA on sleep and associated factors. (3) patients whose recorded sleep time in a supine and non-supine position was less than 30 minutes: this criterion to ensure that the diagnosis of POSA was based on sufficient data. A minimum of 30 minutes of sleep in both supine and non-supine positions is necessary to accurately assess the positional influence on OSA. Insufficient data could lead to misclassification and reduce the validity of the study's findings.

The research protocol was reviewed and approved by the ethics committee of the Capital Institute of Pediatrics, under the reference number SHERLL2021034. This study was conducted following the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Informed consent was obtained from all study participants and their parents/guardians before the commencement of the study.

Collection of Clinical Data

During our data collection process, the gender and age of each participant were carefully recorded by a pediatrician and a researcher, both of whom were rigorously trained and certified. Additionally, we documented their weight and height to calculate their body mass index (BMI) before undergoing polysomnography, in line with the guidelines from the "Overweight and Obesity Screening Table for School-age Children and Adolescents", according to the health industry standard WS/T 586–2018.[25] Obesity was defined as having a BMI above the 95th percentile for the child's age and gender.[26]

Definition and Diagnosis of Allergic Rhinitis and Non-Allergic Rhinitis

Allergic rhinitis (AR) is defined as a nasal condition caused by IgE-mediated inflammation following exposure to allergens. The diagnosis is based on clinical history, physical examination, and positive results from specific IgE tests or skin prick tests for common allergens.[27] We collected this information through the sleep questionnaire by asking, "Has your child been tested for allergens and diagnosed with allergic rhinitis?"

In contrast, non-allergic rhinitis is diagnosed based on the presence of nasal symptoms such as nasal congestion, rhinorrhea, and sneezing, rather than allergy triggers. It is typically confirmed by a negative result on specific IgE testing or skin prick tests. However, some localized allergic rhinitis cases may go unrecognized. Therefore, nasal cytology has been suggested as a highly useful tool for describing and diagnosing non-allergic rhinitis.[28] We collected this information through the sleep questionnaire by asking, "Has your child been tested for allergens and diagnosed with non-allergic rhinitis? "

Questionnaire

The sleep questionnaire used in the study includes 6 questions related to clinical comorbidities of pediatric OSA[29–33] and 22 PSQ items.[34,35] The 6 questions are as follows: '1. Does your child have enlarged tonsils? 2. Does your child have enlarged adenoids? 3. Has your child been tested for allergens and diagnosed with allergic rhinitis? 4. Has your child been tested for allergens and diagnosed with non-allergic rhinitis? 5. Have your child diagnosed with bronchial asthma? 6. Have your child been diagnosed with a neuromuscular disease?'

The English Version of PSQ was developed by Chervin et al, and the Chinese version was translated by Tai et al[34,35] The PSQ comprises 22 questions that probe a variety of symptoms including snoring characteristics (duration, intensity, frequency), episodes of apnea, mouth breathing, the presence of enuresis, excessive daytime sleepiness, headaches, weight and height percentiles, as well as symptoms of hyperactivity-impulsivity and inattention.[35] Please see Table S1 for details. The questionnaire was administered to parents before child's admission for overnight polysomnography. Parents were instructed to fill out the questionnaire based on their observations of their child's symptoms and behaviors. Typically, completing the questionnaire required between 10 to 20 minutes. The data from the questionnaires were collected jointly by a physician and a researcher, ensuring thorough and consistent data collection throughout the study.

Polysomnography

Patients in our study were referred to the sleep laboratory for polysomnography (PSG) by pediatricians and otolaryngologists within our hospital when they were suspected of having OSA. All participating children underwent overnight PSG at the Sleep Medicine Center of the Children's Hospital affiliated with the Capital Pediatric Research Institute. Each child was accompanied by their parents in the same room during the procedure. Polysomnography was conducted using a computerized system (Alice 6, Philips Respironics Inc., Murrysville, Pennsylvania), which included a 6-channel electroencephalograph with bilateral frontal, central, and occipital leads, an electrocardiogram, and airflow monitoring through both a nasal pressure cannula and a thermistor. Respiratory effort was assessed using thoracic and abdominal inductive plethysmography, while oxygen saturation was measured with pulse oximetry via a finger probe. PSG data were manually scored by certified sleep technologists following the guidelines established by the American Academy of Sleep Medicine (AASM) in 2007.[36] Additionally, the scoring data were reviewed by pediatric sleep physicians to ensure

the accuracy and consistency of the results. Sleep positions were manually determined at 30-second intervals. All PSG data were collected by two designated doctors and researchers.

The AHI was calculated as the average number of apneas and hypoventilation per hour of sleep. The severity of OSA was categorized based on AHI values: primary snoring (AHI < 1), mild (1 ≤ AHI < 5), moderate (5 ≤ AHI < 10), and severe (AHI ≥ 10).[24]

The oxygen desaturation index (ODI) was defined as the number of oxygen desaturations (≥ 3%) per hour of sleep, and the minimum SpO2 referred to the lowest value of blood oxygen saturation measured during the entire sleep period.[37]

POSA was defined according to Cartwright et al as AHI supine being at least twice as high as AHI non-supine. NPOSA was characterized by AHI when sleeping in the supine position less than twice that in non-positional positions.[12]

Statistical Analysis

All statistical analyses were conducted using R software (version 4.2.3). Descriptive statistics, including means with standard deviations for continuous variables and frequencies with percentages for categorical variables, were computed. Nonparametric testing using the Wilcoxon rank sum test for variables that are not normally distributed. The chi-square test was used to assess the independence of categorical variables, while the Wilcoxon rank sum test with a continuity correction was employed for between-group comparisons. Enhanced analytical accuracy was achieved using the compare Groups package. Logistic regression models were utilized to estimate odds ratios (ORs) and their 95% confidence intervals (CIs), with coefficients transformed using the exp () function.

To control for confounding factors and selection bias, propensity score matching (PSM) was applied to the entire cohort of children diagnosed with OSA. Propensity scores were derived from logistic regression analysis of covariates, including gender and age, and used for one-to-two nearest-neighbor matching with a caliper value of 0.2 standard deviations of the logit of the propensity score. To evaluate the effectiveness of the matching process, we generated a balance plot (Figure S1). The plot indicates that the matching process successfully reduced the standardized mean differences for gender and age, achieving a well-balanced comparison between the matched groups. A significance level of 0.05 was used.

Results

Prevalence of Positional Obstructive Sleep Apnea in Children

The study initially included 1,236 children aged 0 to 17 years. After applying exclusion criteria, 908 cases were retained, consisting of 158 cases meeting the criteria for POSA and 750 cases classified as NPOSA (Figure 1). The overall prevalence of POSA within the study population was found to be 12.8%. The mean age of patients with NPOSA/POSA was 5.49±2.71 years/5.84±2.78 years, with 63.1% (n=473)/61.4% (n=97) being male. And the BMI of patients with NPOSA/POSA was 16.8±3.41/18.0±4.18 kg/cm^2 (Table S2). Following PSM at a 1:2 ratio between POSA and NPOSA groups, 153 POSA cases were matched with 306 NPOSA cases (Figure 1). Among these children after PSM, 102 had mild OSA, 186 had moderate OSA, and 171 had severe OSA. The prevalence of POSA after matching was 52% in the mild OSA group, 29% in the moderate OSA group, and 26.9% in the severe OSA group (Figure 2). Post-matching BMI classification identified 37 cases as wasting, 238 as normal, 74 as overweight, and 110 as obese. The prevalence of POSA across BMI categories post-matching was 24.3% in wasting individuals, 31.5% in normal BMI, 33.8% in overweight, and 40% in obese individuals (Figure 3).

Comparative Analysis of Polysomnography Data in Positional and Non-Positional Obstructive Sleep Apnea

Following PSM, a comparative analysis of PSG data between the NPOSA and POSA groups unveiled significant differences. The NPOSA group demonstrated a notably higher AHI compared to the POSA group, and both non-rapid eye movement apnea-hypopnea index (NREM-AHI) and rapid eye movement apnea-hypopnea Index (REM-AHI) were notably higher in the NPOSA group. Furthermore, the NPOSA group exhibited a lower Minimum SpO2 in contrast to the

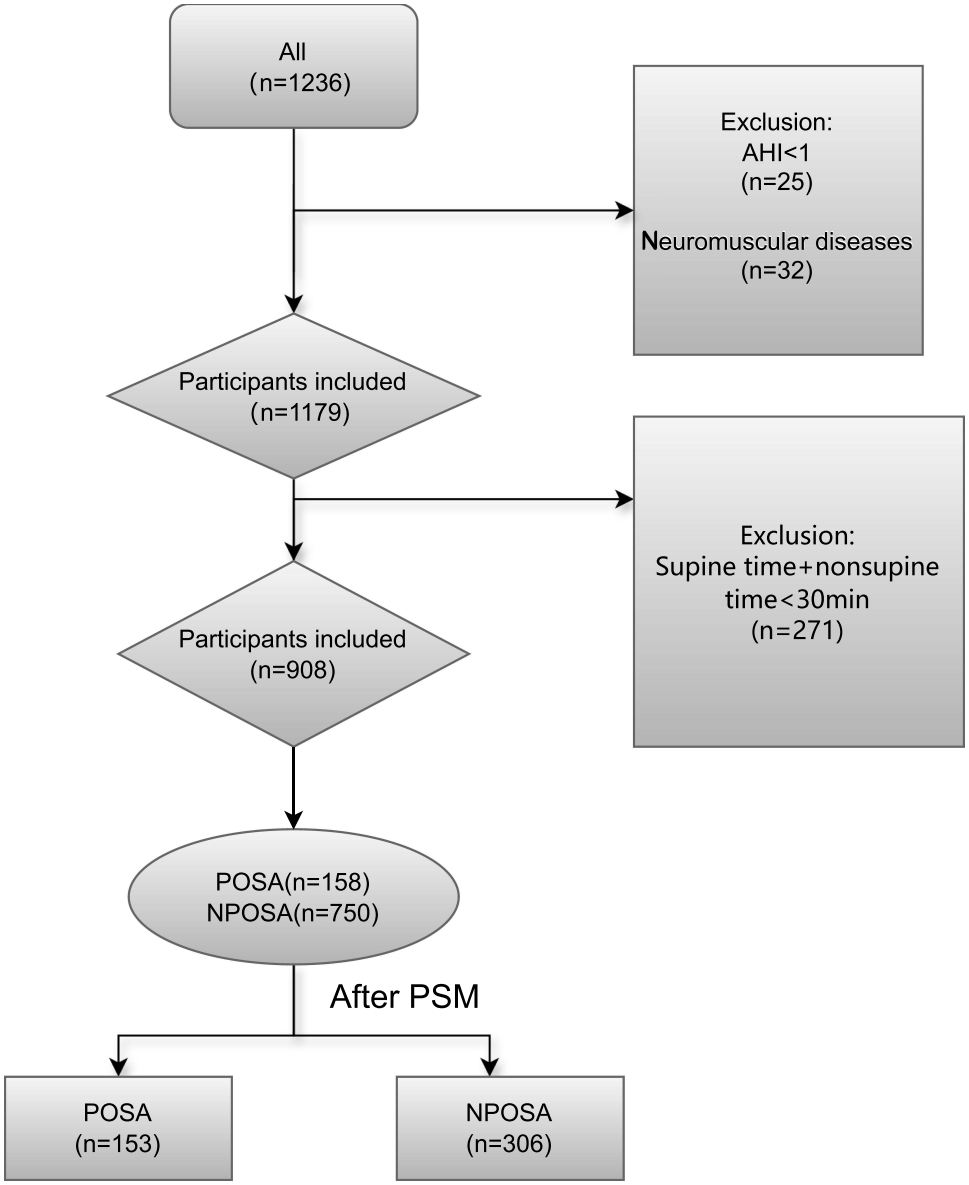

Figure 1 Flowchart of data inclusion.

POSA group. The supine AHI was lower in the NPOSA group than in the POSA group, while the non-supine AHI was lower in the POSA group compared to the NPOSA group. Total sleep time (TST) was found to be shorter in the POSA group than in the NPOSA group. Additionally, N (non-rapid eye movement sleep) 1%, N2%, and REM (rapid eye movement sleep) % were higher in the NPOSA group compared to the POSA group, whereas N3% was lower in the NPOSA group compared to the POSA group. TST in the supine position was higher in the NPOSA group than in the POSA group, while TST in the non-supine position was lower in the NPOSA group compared to the POSA group ($p<0.05$), as outlined in Table 1.

Prevalence Variations of Positional and Non-Positional Obstructive Sleep Apnea Post-Propensity Score Matching

The chi-square test revealed a notable increase in the prevalence of POSA within the mild OSA population compared to NPOSA. Conversely, patients diagnosed with severe OSA exhibited a higher prevalence of NPOSA compared to POSA. Furthermore, individuals with non-allergic rhinitis and allergic rhinitis illustrated a higher prevalence of POSA in

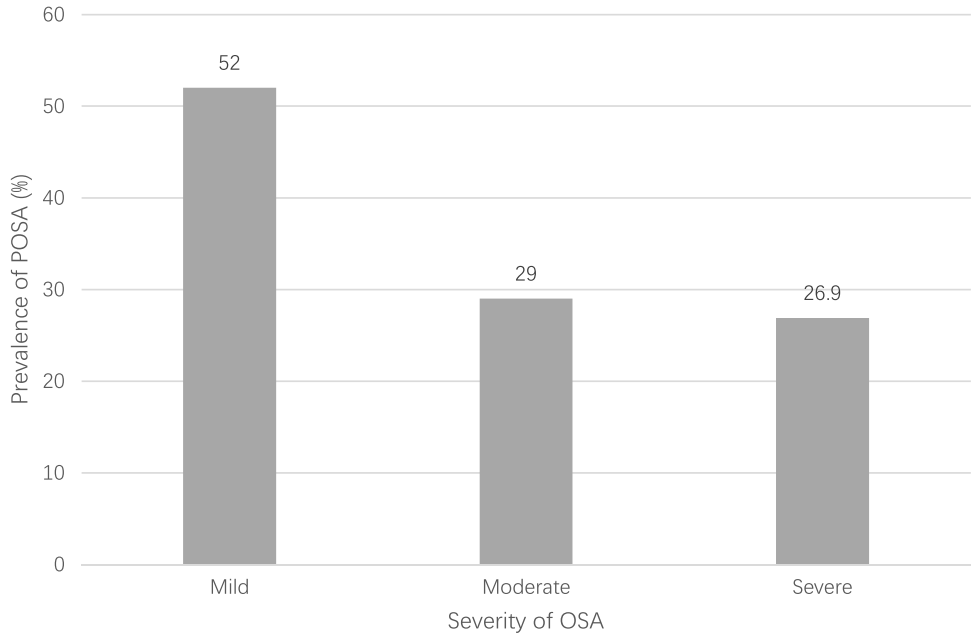

Figure 2 The prevalence of Positional Obstructive Sleep Apnea in different severity groups of Obstructive Sleep Apnea after PSM.

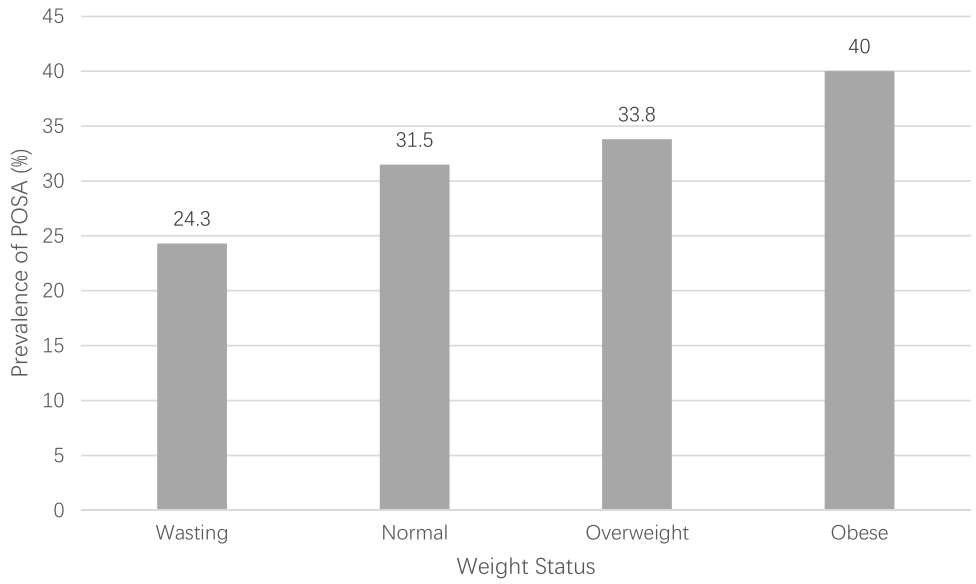

Figure 3 The prevalence of Positional Obstructive Sleep Apnea in different BMI groups of Obstructive Sleep Apnea after PSM.

comparison to NPOSA. Lastly, in patients with obesity, the prevalence of POSA was higher than that of NPOSA, although this comparison was not statistically significant (Table 2). The proportion of patients with a history of bronchial asthma did not significantly differ between the NPOSA and POSA groups (Table S3).

Risk Factors for POSA in Children

After PSM, OSA severity, non-allergic rhinitis, allergic rhinitis, and BMI were each included as variables in the logistic regression model for POSA. In the univariate analysis, allergic rhinitis, non-allergic rhinitis, and mild OSA were significant risk factors for POSA, with OR values greater than 1 and P-values less than 0.05, while obesity was not significant. In the multivariate analysis, Model 1 (adjusted for AHI) showed that allergic rhinitis, non-allergic rhinitis,

Table 1 Comparison of POSA and NPOSA on PSG Data After PSM

Variables	NPOSA	POSA	P-value
n	306	153	
*Age, y	5.96± (2.70)	5.97± (2.72)	0.971
**Male, n (%)	189 (61.8)	96 (62.7)	0.919
*BMI, kg/m^2	17.4± (4.05)	18.0± (4.18)	0.135
*TST, min	427± (47.4)	411± (58.4)	0.002
*N1, %	3.53± (1.94)	3.02± (2.25)	0.017
*N2, %	45.4± (13.2)	42.5± (17.1)	0.064
*N3, %	35.4± (12.4)	40.2± (16.8)	0.002
*REM, %	15.6± (5.95)	14.3± (5.88)	0.02
*AHI, events/h	10.3± (5.67)	8.66± (6.49)	0.008
*REM-AHI, events/h	17.4± (13.9)	14.3± (14.1)	0.024
*NREM-AHI, events/h	8.77± (5.08)	7.43± (5.79)	0.015
*Total time in supine position, min	225± (85.7)	168± (94.2)	<0.001
*Total time in non-supine position, min	202± (81.9)	241± (97.0)	<0.001
*Supine-time/nonsupine-time	1.70± (1.89)	1.09± (1.31)	<0.001
*Supine AHI, events/h	10.3± (6.53)	15.6± (12.9)	<0.001
*Non-supine AHI	11.0± (7.93)	5.00± (3.99)	<0.001
*Supine AHI/ Non-supine-AHI	1.04± (0.46)	3.57± (3.37)	<0.001
*Minimum SpO2	0.87± (0.06)	0.88± (0.05)	0.001
*ODI, events/h	8.11± (6.03)	7.34± (6.26)	0.209

Notes: All populations were derived from propensity score matched populations. * indicates that the data is expressed as mean ± standard; P: p-value; ** indicates that the data is expressed as the number of individuals (percentile of the total number). TST: total sleep time; N1%, N2%, N3%, REM%: the percentage of stage 1, 2, 3 and rapid eye movement sleep in total sleep time; Minimum SpO2: Minimum Peripheral Capillary Oxygen Saturation; ODI: oxygen desaturation index.

Abbreviations: PSM, propensity score matching; BMI, body mass index; AHI, apnea-hypopnea index; REM-AHI: the AHI of rapid eye movement sleep; NREM-AHI: the AHI of non-rapid eye movement sleep.

Table 2 Chi-Square Test for POSA and NPOSA Among Different Groups After PSM

	NPOSA	POSA	X^2	P-value
OSA severity			20.659	<0.001
Mild, n (%)	49 (16.0)	53 (34.6)		
Moderate, n (%)	132 (43.1)	54: (35.3)		
Severe, n (%)	125 (40.8)	46 (30.1)		
Non-allergic rhinitis			5.7267	0.017
Yes, n (%)	188 (61.4)	112 (73.2)		
No, n (%)	118 (38.6)	41 (26.8)		
Allergic rhinitis			4.3776	0.036
Yes, n (%)	125 (40.8)	79 (51.6)		
No, n (%)	181 (59.2)	74 (48.4)		

(*Continued*)

Table 2 (Continued).

	NPOSA	POSA	X^2	P-value
BMI groups			3.9132	0.271
Wasting, n (%)	28 (9.2)	9 (5.9)		
Normal, n (%)	163 (53.3)	75 (49.0)		
Overweight, n (%)	49 (16.0)	25 (16.3)		
Obesity, n (%)	66 (21.6)	44 (28.8)		

Notes: All populations were derived from propensity score matched populations.
X^2: Chi-square value; P: p-value.
Abbreviations: PSM, propensity score matching; BMI, body mass index.

Table 3 Logistic Regression Models Displaying Odds Ratios and Confidence Intervals for POSA

	Univariate		Model 1		Model 2		Model 3	
	OR (95% CI)	P-Value	OR (95% CI)	P-Value	OR (95% CI)	P	OR (95% CI)	P-Value
OSA severity								
Moderate-severe OSA	1		-	-	-		1	
Mild OSA	2.78(1.77, 4.38)	<0.001	-	-	-		2.86(1.81, 4.55)	<0.001
Comorbidities								
Non-rhinitis	1		1		-			-
Rhinitis	1.71(1.13, 2.64)	0.013	1.64(1.07, 2.55)	0.026	-			-
Non-allergic rhinitis	1		1		-			-
Allergic rhinitis	1.55(1.05, 2.29)	0.029	1.49(1.00, 2.23)	0.0495	-			-
Weight status								
Non-obesity	1		1		1			-
Obesity	1.47(0.94, 2.28)	0.09	1.66(1.05, 2.62)	0.03	1.6(1.81, 4.55)	0.047		-

Notes: All populations were derived from propensity score matched populations. Univariate analysis refers to the examination of a single variable in isolation to understand its relationship with an outcome variable. Model 1 involves a statistical model adjusted for AHI. Model 2 includes adjustments for AHI, allergic rhinitis, and rhinitis. Model 3 adjusts for allergic rhinitis, rhinitis, and BMI.

and obesity were significant risk factors for POSA (OR > 1, P < 0.05). Model 2 (adjusted for AHI, allergic rhinitis, and non-allergic rhinitis) indicated that obesity became a significant risk factor for POSA (OR > 1, P < 0.05). Model 3 (adjusted for allergic rhinitis, non-allergic rhinitis, and BMI) revealed that mild OSA was a significant risk factor for POSA (OR > 1, P < 0.05). These findings suggested that allergic rhinitis, non-allergic rhinitis, obesity, and mild OSA were significant risk factors for POSA in children when controlled for different variables (Table 3).

Discussion

This cross-sectional study delved into the prevalence and characteristics of POSA in children, shedding light on potential risk factors associated with this condition. Among our pediatric participants diagnosed with OSA, we identified the POSA of prevalence as 12.8%. We employed PSM in our analysis to mitigate potential biases. Consequently, we opted to utilize the matched data for our analyses, ensuring a more balanced and reliable comparison between the two groups. Following PSM, we observed variations in POSA prevalence across different OSA severity levels, notably peaking in the

mild OSA group. Our analysis of PSG parameters unveiled differences between children with POSA and NPOSA, with POSA associated with a lower AHI compared to NPOSA. Particularly noteworthy was our observation of a significant correlation between POSA and mild OSA. Additionally, we found a significant association between POSA and non-allergic rhinitis as well as allergic rhinitis. Particularly, after considering the influence of mild OSA, non-allergic rhinitis, and allergic rhinitis, we observed a significant correlation between obesity and POSA. These findings suggested that non-allergic rhinitis, allergic rhinitis, and obesity may have increased the risk of POSA in pediatric patients.

Our study revealed a pediatric POSA prevalence of 12.8%, contrasting with Verhelst et al's findings of 19%.[14] The variance could stem from multiple factors. First, our exclusion criteria differed from those of Verhelst et al. We excluded children who had undergone adenotonsillectomy (AT) and those with neurological or muscular disorders, while their study included children with persistent postoperative OSA and conditions like Down syndrome. Additionally, geographical differences between the studies (our research was conducted in China, whereas Verhelst et al' s study was conducted in Belgium) may account for variations in genetic, environmental, and lifestyle factors, potentially leading to different POSA prevalence rates. Finally, our study has a larger sample size (1,236 participants) compared to the 171 participants in Verhelst et al' s study, which may provide a more representative estimate of POSA prevalence. When we categorized patients based on the severity of OSA, we consistently observed that the proportion of patients with POSA was highest among those with mild OSA and lowest among those with severe OSA. Similar to findings by Verhelst et al,[14] who reported POSA in children as milder. This finding also consistent with what is typically observed in several studies, where POSA always be more prevalent in mild cases of OSA.[38,39] Our results indicated a significant correlation between mild OSA and POSA (OR=2.78, 95% CI: 1.77–4.38, $p<0.001$), indicating that children with mild OSA are more likely to develop POSA, a milder form of OSA in children.

Our study results showed that the NPOSA group had higher percentages of N1, N2, and REM sleep stages compared to the POSA group, while the POSA group had higher percentages of N3 sleep stages. These findings had significant physiological and clinical implications. Firstly, the higher N1 and N2 percentages in the NPOSA group, and with the lower N3 percentages, suggest that these children spend more time in lighter sleep stages. This could lead to poorer sleep quality, adversely affecting their daytime functioning and overall health. Prolonged time in lighter sleep stages may indicate disruptions in deeper sleep stages, negatively impacting restorative sleep.[40] The POSA group had a significantly higher percentage of N3 indicating that children with NPOSA may spend less time in deep sleep, which could potentially affect the secretion of growth hormones and subsequently impact growth and development.[41,42] Studies in children have shown that REM sleep time in POSA patients is shorter than that in NPOSA patients,[14] which is consistent with our study. Our study indicated that the REM sleep duration for children with NPOSA (15.6 ± 5.95) was slightly longer than that of children with POSA (14.3 ± 5.88), the respiratory events during REM sleep (AHI: 17.4 ± 13.9) were significantly higher in children with NPOSA compared to those with POSA (14.3 ± 14.1). This may demonstrate that children with NPOSA experience more severe respiratory events during REM sleep, which could more easily affect the maturation of brain structure and function, potentially impacting emotional and cognitive development.[43,44]

In our study, we observed that the AHI in the NPOSA group exceeded that of the POSA group, indicating a greater occurrence of breathing pauses and low ventilation episodes among children with NPOSA. This trend aligns with other findings from adult and pediatric studies, where it was also observed that the AHI in the NPOSA population exceeded that of the POSA group.[14,45] Moreover, the Minimum SpO2 was lower in the NPOSA group, suggesting more frequent nighttime desaturations, which points to a potentially more severe intermittent hypoxic state in these children. The clinical implications of these findings are substantial. Higher AHI and lower Minimum SpO2 in NPOSA children imply a more severe form of obstructive sleep apnea, which can lead to significant health issues. Intermittent hypoxemia, characterized by repeated episodes of oxygen deprivation and restoration, is associated with oxidative stress, systemic inflammation, and increased sympathetic activity. These factors can contribute to long-term health complications such as impaired endothelial function, elevated blood pressure, and increased risk for cardiovascular and metabolic disorders.[46–48] In terms of daily life, the increased severity of OSA in the NPOSA group may result in more pronounced daytime symptoms, including excessive daytime sleepiness, impaired cognitive function, and behavioral problems. Chronic sleep disruption and intermittent hypoxia can affect children's mood, academic performance, and overall quality of life. Additionally, the more frequent

and severe episodes of desaturation observed in NPOSA children could impact their growth and development, potentially leading to long-term developmental delays.[49,50]

Kim et al[7] investigated the AHI was significantly higher in the supine position compared to the non-supine position among children with OSA. According to the results of this study, we found that the AHI in the supine position was higher than that in the non-supine position in both the NPOSA group and the POSA group. This suggests that the supine position may increase apneic events by making the upper airway more susceptible to obstruction. In addition, we observed that the AHI of POSA patients was significantly higher than that of NPOSA patients during supine sleep, while the AHI of POSA patients was lower than that of NPOSA patients during non-supine sleep. These results indicate that compared with patients with NPOSA, sleep apnea in POSA patients is more severe in the supine position but relatively mild in the non-supine position. Verhelst et al[14] noted that children with POSA sleep longer in the supine position. However, our study showed that patients with POSA spent more time sleeping in the non-supine position than patients with NPOSA, while patients with NPOSA spent more time sleeping in the supine position than patients with POSA. Additionally, among POSA patients, we found that they experienced more severe apnea events in the supine position (AHI was significantly higher in the supine position compared to the non-supine position). POSA patients also spent more time in non-supine sleep than in the supine position. These findings suggest that avoiding the supine position during sleep, likely due to the body's self-protective mechanisms, may help reduce apnea events in POSA patients, highlighting the importance of monitoring and managing sleep posture in POSA patients. There is already research showing that positional therapy is effective for children with POSA.[17] Our study suggests that positional therapy may have potential benefits for children with POSA as well. Future research should further investigate the specific effects of positional therapy in pediatric POSA patients.

We conducted a survey using the sleep questionnaire and all participants had undergone PSG. We explored to analyze the relationship between each item on the questionnaires and POSA. Our findings indicate that the proportion of POSA is significantly higher in patients with non-allergic rhinitis and allergic rhinitis compared to those without them, and this difference was statistically significant. Further logistic regression analysis suggests that the presence of non-allergic rhinitis and allergic rhinitis in OSA patients may increase the risk of POSA. Rhinitis, whether allergic or non-allergic, involves inflammation and congestion of the nasal passages, which can contribute to upper airway resistance and obstruction during sleep, thereby increasing the likelihood of developing obstructive sleep apnea.[51,52] Individuals with rhinitis may experience nasal congestion, swelling of the nasal mucosa, and increased nasal secretions, all of which can exacerbate upper airway obstruction during sleep. When combined with positional factors, such as sleeping in the supine position, nasal congestion may further impede airflow, leading to a higher prevalence of POSA among patients with non-allergic rhinitis of OSA. Previous studies have shown that nasal inflammation and congestion associated with allergic rhinitis can significantly impact sleep quality and respiratory patterns during sleep.[31,53] When sleeping in the supine position, the head is horizontal to the body, the blood in the head returns slowly, and the nasal mucosal blood vessels will continue to be congested, aggravating nasal congestion. Additionally, nasal secretions can easily block the posterior meatus when sleeping in the supine position. In contrast, sleeping on the side can alleviate the problems of a narrow pharyngeal cavity and poor ventilation caused by the supine position, and when sleeping on the side, the nasal cavities on both sides alternately show good ventilation on one side. This may cause OSA patients to have more severe sleep apnea and hypopnea in the supine position than in the non-supine position, so allergic rhinitis and non-allergic rhinitis have more severe sleep apnea and hypopnea in the supine position than in the non-supine position may be risk factors for POSA. The specific physiological mechanism is still unclear, and future research needs to continue exploring this issue.

The relationship between obesity and POSA remains controversial. Previous studies have reported that children with POSA have a higher prevalence of obesity,[14] but a study showed that no relation between obesity with POSA.[54] Obese patients may be more susceptible to the effects of body position differences and gravity on fat deposition around the upper airway. Studies in adults indicate that low BMI predicts POSA,[55] and POSA is more common in patients with less obesity. Our findings are consistent with other studies that found higher prevalence rates of POSA in obese children.[14,15] Dayyat et al[21] found that obese children had a higher AHI in the supine position than in the prone position. Obese children may be more susceptible to the effects of positional differences and gravity on fat deposition around the upper respiratory tract. When obese children lie supine, their upper airway is more prone to collapse, increasing the risk of

obstruction. This effect is intensified in patients with non-allergic rhinitis and allergic rhinitis due to nasal and pharyngeal congestion. Side sleeping can alleviate this pressure and improve breathing. In our univariate analysis, we found that mild OSA, allergic rhinitis, and non-allergic rhinitis were significantly associated with POSA, while obesity was not statistically significant. However, after conducting a multivariate analysis, the relationship between obesity and POSA became significant ($P < 0.05$). This result is consistent with other studies finding that obesity is related to POSA in children.[14,15] In univariate analysis, each variable is considered independently, without accounting for interactions with other variables, which can sometimes obscure significant associations. However, in multivariate analysis, multiple variables and their interactions are considered simultaneously, revealing how individual factors combine with others to influence outcomes. For instance, while univariate analysis did not identify obesity as a significant risk factor for POSA, multivariate analysis showed that when controlling for mild OSA, allergic rhinitis, and non-allergic rhinitis—factors that may interact with obesity—the impact of obesity on POSA becomes evident. This suggests that these conditions may mask the relationship between obesity and POSA in univariate analysis. The findings underscore the importance of considering related factors, such as mild OSA, allergic rhinitis, and non-allergic rhinitis, in comprehensive patient assessments to accurately evaluate the risk of POSA associated with obesity.

Strengths and Limitations

Several strengths in this study. First, the study employs an objective screening method, using overnight PSG to confirm OSA, ensuring an unbiased assessment beyond subjective symptoms. Second, the use of propensity score matching (PSM) helps mitigate potential confounding factors and selection bias, enhancing the credibility of comparisons between the POSA and NPOSA groups. Third, the study collected data from 1,236 pediatric OSA patients between January 2020 and May 2023. The large sample size and extended time span, along with the fact that the single center is a renowned children's hospital in China that attracts a diverse patient population from various regions, enhance the representativeness and generalizability of the findings. Finally, the study benefits from a consistent diagnostic system, professionally trained and stable sleep technicians, and standardized equipment, ensuring the reliability of the data collected.

However, several limitations should be noted. First, the study's retrospective design may limit its ability to establish causal relationships between POSA and other variables. Second, reliance on parental reports for certain data, such as questionnaire-reported symptoms, may lead to recall bias or subjective interpretation. Third, the study's cross-sectional nature provides only a snapshot of POSA prevalence and associated factors at a specific point in time, lacking insights into the condition's trajectory and long-term outcomes. Future studies with prospective designs (cohort-study) and long-term follow-up are necessary to address these limitations and validate our findings in broader and more diverse populations. Additionally, the single-center design in China may limit the generalizability of the results. Cultural differences, healthcare practices, and genetic backgrounds could affect the prevalence and characteristics of POSA, making the findings less applicable to other regions or ethnic groups. To validate and expand upon these findings, further research in diverse settings, including multi-center studies across various geographical locations and ethnic groups, is needed to determine whether the observed associations and risk factors for POSA are consistent across different populations.

Conclusion

The prevalence of POSA in children is lower than in adults, and its severity is less than that of NPOSA. Compared to NPOSA patients, POSA patients had significantly higher AHI during supine sleep and lower AHI during non-supine sleep. POSA patients also spent more time in non-supine positions, suggesting that avoiding supine sleep may help reduce respiratory events. These findings highlight the importance of monitoring and managing sleep posture in POSA patients. Future research should focus on exploring the potential benefits of positional therapy for these patients.

Funding

This study was funded by the National Natural Science Foundation of China (Grant 72004142), Capital's Funds for Health Improvement and Research (CFH 2024-2G-1135), Beijing Municipal Natural Science Foundation (7232010), and

Key Program of Capital's Funds for Health Improvement and Research (2022-1-2101). The funders had no role in the design of the study and collection, analysis, and interpretation of data and in writing the manuscript.

Disclosure

All authors report no conflicts of interest in this work.

References

1. Bixler EO, Vgontzas AN, Lin H-M, et al. Sleep disordered breathing in children in a general population sample: prevalence and risk factors. *Sleep*. 2009;32(6):731–736. doi:10.1093/sleep/32.6.731
2. Marcus CL, Brooks LJ, Ward SD, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*. 2012;130(3):e714–e755. doi:10.1542/peds.2012-1672
3. Li AM, So HK, Au CT, et al. Epidemiology of obstructive sleep apnoea syndrome in Chinese children: a two-phase community study. *Thorax*. 2010;65(11):991–997. doi:10.1136/thx.2010.134858
4. Marcus CL. Sleep-disordered breathing in children. *Am J Respir Crit Care Med*. 2001;164(1):16–30. doi:10.1164/ajrccm.164.1.2008171
5. Rana M, August J, Levi J, Parsi G, Motro M, DeBassio W. Alternative approaches to adenotonsillectomy and continuous positive airway pressure (CPAP) for the management of pediatric obstructive sleep apnea (OSA): a review. *Sleep Disord*. 2020;2020(1):7987208. doi:10.1155/2020/7987208
6. Zhang Y, Leng S, Hu Q, et al. Pharmacological interventions for pediatric obstructive sleep apnea (OSA): network meta-analysis. *Sleep Med*. 2024;116:129–137. doi:10.1016/j.sleep.2024.01.030
7. Kim HY, Dhong H-J, Lee J-K, Chung S-K, Jung S-C. Sleep quality and effects of position on sleep apnea in East Asian children. *Auris Nasus Larynx*. 2011;38(2):228–232. doi:10.1016/j.anl.2010.07.005
8. Walter LM, Dassanayake DU, Weichard AJ, Davey MJ, Nixon GM, Horne RS. Back to sleep or not: the effect of the supine position on pediatric OSA: sleeping position in children with OSA. *Sleep Med*. 2017;37:151–159. doi:10.1016/j.sleep.2017.06.014
9. Saigusa H, Suzuki M, Higurashi N, Kodera K. Three-dimensional morphological analyses of positional dependence in patients with obstructive sleep apnea syndrome. *Anesthesiology*. 2009;110(4):885–890. doi:10.1097/ALN.0b013e31819b5d57
10. Squier SB, Patil SP, Schneider H, Kirkness JP, Smith PL, Schwartz AR. Effect of end-expiratory lung volume on upper airway collapsibility in sleeping men and women. *J Appl Physiol*. 2010;109(4):977–985. doi:10.1152/japplphysiol.00080.2010
11. Takahashi S, Ono T, Ishiwata Y, Kuroda T. Effect of changes in the breathing mode and body position on tongue pressure with respiratory-related oscillations. *Am J Orthod Dentofacial Orthop*. 1999;115(3):239–246. doi:10.1016/S0889-5406(99)70324-0
12. Cartwright RD. Effect of sleep position on sleep apnea severity. *Sleep*. 1984;7(2):110–114. doi:10.1093/sleep/7.2.110
13. Garg H, Er XY, Howarth T, Heraganahally SS. Positional sleep apnea among regional and remote Australian population and simulated positional treatment effects. *Nat Sci Sleep*. 2020;12:1123–1135. doi:10.2147/NSS.S286403
14. Verhelst E, Clinck I, Deboutte I, et al. Positional obstructive sleep apnea in children: prevalence and risk factors. *Sleep Breath*. 2019;23:1323–1330. doi:10.1007/s11325-019-01853-z
15. Selvadurai S, Voutsas G, Massicotte C, et al. Positional obstructive sleep apnea in an obese pediatric population. *J Clin Sleep Med*. 2020;16(8):1295–1301. doi:10.5664/jcsm.8496
16. Tholen K, Meier M, Kloor J, Friedman N. Persistent OSA in obese children: does body position matter? *J Clin Sleep Med*. 2021;17(2):227–232. doi:10.5664/jcsm.8902
17. Xiao L, Baker A, Voutsas G, et al. Positional device therapy for the treatment of positional obstructive sleep apnea in children: a pilot study. *Sleep Med*. 2021;85:313–316. doi:10.1016/j.sleep.2021.07.036
18. Al Oweidat K, Toubasi AA, Albtoosh AS, et al. Comparing the characteristics of positional and nonpositional sleep apnea patients among the Jordanian population. *Ann Thorac Med* 2022;17(4):207–213.
19. Wang X, Luo J, Huang R, Yi X. Preliminary study on clinical characteristics of Chinese patients with positional obstructive sleep apnea. *Sleep Breath*. 2021;1–8.
20. Kim KT, Cho YW, Kim DE, Hwang SH, Song ML, Motamedi GK. Two subtypes of positional obstructive sleep apnea: supine-predominant and supine-isolated. *Clin Neurophysiol*. 2016;127(1):565–570. doi:10.1016/j.clinph.2015.06.009
21. Dayyat E, Maarafeya MM, Capdevila OS, Kheirandish-Gozal L, Montgomery-Downs HE, Gozal D. Nocturnal body position in sleeping children with and without obstructive sleep apnea. *Pediatric Pulmonol*. 2007;42(4):374–379. doi:10.1002/ppul.20590
22. Cuhadaroglu C, Keles N, Erdamar B, et al. Body position and obstructive sleep apnea syndrome. *Pediatric Pulmonol*. 2003;36(4):335–338. doi:10.1002/ppul.10366
23. Zhang X-W, Li Y, Zhou F, Guo C, Huang Z-T. Association of body position with sleep architecture and respiratory disturbances in children with obstructive sleep apnea. *Acta oto-Laryngologica*. 2007;127(12):1321–1326. doi:10.1080/00016480701242451
24. Hsu WC, Kang KT, Yao CCJ, et al. Evaluation of upper airway in children with obstructive sleep apnea using cone-beam computed tomography. *Laryngoscope*. 2021;131(3):680–685. doi:10.1002/lary.28863
25. Liu Z, Sun L, Zhang Y, et al. The prevalence of underweight and obesity in Chinese children and adolescents with major depressive disorder and relationship with suicidal ideation and attempted suicide. *Front Psychiatry*. 2023;14:1130437. doi:10.3389/fpsyt.2023.1130437
26. Chen W, Chang M-H. New growth charts for Taiwanese children and adolescents based on World Health Organization standards and health-related physical fitness. *Pediatr Neonatol*. 2010;51(2):69–79. doi:10.1016/S1875-9572(10)60014-9
27. Brown TJ. Diagnosis and management of allergic rhinitis in children. *Pediatr Ann*. 2019;48(12):e485–e488. doi:10.3928/19382359-20191111-01
28. Poddighe D, Gelardi M, Licari A, Del Giudice MM, Marseglia GL. Non-allergic rhinitis in children: epidemiological aspects, pathological features, diagnostic methodology and clinical management. *World J Methodol*. 2016;6(4):200. doi:10.5662/wjm.v6.i4.200
29. Tagaya M, Nakata S, Yasuma F, et al. Relationship between adenoid size and severity of obstructive sleep apnea in preschool children. *Int J Pediatr Otorhinolaryngol*. 2012;76(12):1827–1830. doi:10.1016/j.ijporl.2012.09.010

30. Liu J, Wu Y, Wu P, Xu Z, Ni X. Analysis of the impact of allergic rhinitis on the children with sleep disordered breathing. *Int J Pediatr Otorhinolaryngol*. 2020;138:110380. doi:10.1016/j.ijporl.2020.110380
31. Zheng M, Wang X, Ge S, et al. Allergic and non-allergic rhinitis are common in obstructive sleep apnea but not associated with disease severity. *J Clin Sleep Med*. 2017;13(8):959–966. doi:10.5664/jcsm.6694
32. Faverio P, Zanini U, Monzani A, et al. Sleep-disordered breathing and chronic respiratory infections: a narrative review in adult and pediatric population. *Int J Mol Sci*. 2023;24(6):5504. doi:10.3390/ijms24065504
33. Katz ES, D'Ambrosio CM. Management of sleep disordered breathing in children with neuromuscular disease. In: *Sleep in Childhood Neurological Disorders*. New York: Demos Medical; 2011:275–294.
34. Li H, Tai J, Feng G, et al. Analysis of sleep quality and related factors among children in Beijing. *Zhonghua er bi yan hou tou Jing wai ke za zhi*. 2019;54(6):416–420. doi:10.3760/cma.j.issn.1673-0860.2019.06.004
35. Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. *Sleep Med*. 2000;1(1):21–32. doi:10.1016/s1389-9457(99)00009-x
36. Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events: deliberations of the sleep apnea definitions task force of the American academy of sleep medicine. *J Clin Sleep Med*. 2012;8 (5):597–619. doi:10.5664/jcsm.2172
37. Pulmonology SoP, Pediatrics SoOSASJ. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*. 2002;109(4):704–712.
38. Akbay MO, Gurkan CG, Ak AE, Sarac S. Exploring the impact of positional sleep apnea in a Turkish population: unveiling the untold story. *Med Sci Monit*. 2023;29:e941425–941421. doi:10.12659/MSM.941425
39. Heinzer R, Petitpierre NJ, Marti-Soler H, Haba-Rubio J. Prevalence and characteristics of positional sleep apnea in the HypnoLaus population-based cohort. *Sleep Med*. 2018;48:157–162. doi:10.1016/j.sleep.2018.02.011
40. Carskadon MA, Dement WC. Normal human sleep: an overview. *Princ and Pract Sleep Med*. 2005;4(1):13–23.
41. Born J, Wilhelm IJ. System consolidation of memory during sleep. *Psychol Res*. 2012;76:192–203. doi:10.1007/s00426-011-0335-6
42. Walker MP. The role of sleep in cognition and emotion. *Ann N Y Acad Sci*. 2009;1156(1):168–197. doi:10.1111/j.1749-6632.2009.04416.x
43. Peever J, Fuller PM. The biology of REM sleep. *Curr Biol*. 2017;27(22):R1237–R1248. doi:10.1016/j.cub.2017.10.026
44. Siegel JM. The REM sleep-memory consolidation hypothesis. *Science*. 2001;294(5544):1058–1063. doi:10.1126/science.1063049
45. Rissanen M, Oksenberg A, Töyräs J, Myllymaa S, Leppänen T. Total durations of respiratory events are modulated within REM and NREM sleep by sleeping position and obesity in OSA patients. *Sleep Med*. 2021;81:394–400. doi:10.1016/j.sleep.2021.02.020
46. Dewan NA, Nieto FJ, Somers VK. Intermittent hypoxemia and OSA: implications for comorbidities. *Chest*. 2015;147(1):266–274. doi:10.1378/chest.14-0500
47. Wali SO, Abaalkhail B, AlQassas I, Alhejaili F, Spence DW, Pandi-Perumal SR. The correlation between oxygen saturation indices and the standard obstructive sleep apnea severity. *Ann Thorac Med*. 2020;15(2):70–75. doi:10.4103/atm.ATM_215_19
48. Rashid NH, Zaghi S, Scapuccin M, Camacho M, Certal V, Capasso R. The value of oxygen desaturation index for diagnosing obstructive sleep apnea: a systematic review. *Laryngoscope*. 2021;131(2):440–447. doi:10.1002/lary.28663
49. Hunter SJ, Gozal D, Smith DL, et al. Effect of sleep-disordered breathing severity on cognitive performance measures in a large community cohort of young school-aged children. *Am J Respir Crit Care Med*. 2016;194(6):739–747. doi:10.1164/rccm.201510-2099OC
50. Nieminen P, Löppönen T, Tolonen U, Lanning P, Knip M, Löppönen H. Growth and biochemical markers of growth in children with snoring and obstructive sleep apnea. *Pediatrics*. 2002;109(4):e55–e55. doi:10.1542/peds.109.4.e55
51. Wongvilairat S, Assanasen P, Banhiran W, Tantilipikorn P, Bunnag C. The prevalence of high risk of obstructive sleep apnea in patients with allergic rhinitis. *Asian Pac J Allergy Immunol*. 2022;40(3):205–209. doi:10.12932/AP-141218-0458
52. Kalpaklıoğlu AF, Kavut AB, Ekici M. Asthma, immunology. Allergic and nonallergic rhinitis: the threat for obstructive sleep apnea. *Ann Allergy Asthma Immunol*. 2009;103(1):20–25. doi:10.1016/S1081-1206(10)60138-X
53. Liu J, Zhang X, Zhao Y, Wang Y. The association between allergic rhinitis and sleep: a systematic review and meta-analysis of observational studies. *PLoS One*. 2020;15(2):e0228533.
54. Wu Y, Zheng L, Cui G, Xu Z, Ni X. Subtypes of obstructive sleep apnea in children and related factors. *J Clin Sleep Med*. 2022;18(10):2397–2404. doi:10.5664/jcsm.10124
55. Wali SO, AlQassas I, Qanash S, et al. The prevalence of positional obstructive sleep apnoea in a sample of the Saudi population. *J Epidemiol Global Health*. 2023;13(1):129–139. doi:10.1007/s44197-023-00089-1