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ORIGINAL ARTICLE

Association Between Childhood Obesity and Obstructive Sleep Apnea Severity

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ABSTRACT

Background: Childhood obesity is an increasingly important risk factor for obstructive sleep apnea (OSA). Although adenotonsillar hypertrophy remains a common cause of pediatric OSA, excess adiposity may worsen upper airway collapsibility and increase disease severity.

Objective: To determine the association between childhood obesity and the severity of obstructive sleep apnea among children undergoing polysomnographic evaluation.

Material and Methods: This cross-sectional analytical study was conducted at a Tertiary Care Hospital in Pakistan from April 2023 to September 2023 and included 135 children aged 5–17 years who presented with symptoms suggestive of sleep-disordered breathing. Anthropometric measurements were recorded, and obesity was defined as body mass index-for-age at or above the 95th percentile. All participants underwent overnight polysomnography. OSA severity was classified using obstructive apnea-hypopnea index (OAHI): no OSA/minimal disease <1 event/hour, mild OSA 1–4.9 events/hour, moderate OSA 5–9.9 events/hour, and severe OSA ≥10 events/hour. The association between obesity and moderate-to-severe OSA was analyzed using chi-square testing and multivariable logistic regression.

Results: Of 135 children, 57 (42.2%) were obese and 78 (57.8%) were non-obese. The mean age was 11.1 +/- 3.3 years, and 78 (57.8%) were male. Moderate-to-severe OSA was observed in 37 obese children (64.9%) compared with 26 non-obese children (33.3%). Severe OSA was more frequent among obese children than non-obese children, 35.1% versus 12.8%, respectively. Median OAHI was significantly higher in obese children than non-obese children, 9.6 events/hour versus 3.8 events/hour. Obesity was significantly associated with moderate-to-severe OSA on unadjusted analysis (odds ratio 3.70; 95% CI 1.80-7.60; p<0.001). After adjustment for age, sex, and tonsillar hypertrophy, obesity remained an independent predictor of moderate-to-severe OSA (adjusted odds ratio 3.12; 95% CI 1.48-6.59; p=0.003).

Conclusion: Childhood obesity was significantly associated with greater OSA severity. Obese children had higher OAHI values, lower oxygen saturation nadirs, and greater odds of moderate-to-severe OSA. Routine assessment of obesity status should be incorporated into the evaluation and risk stratification of children with suspected OSA.

Keywords: Childhood obesity; obstructive sleep apnea; pediatric sleep-disordered breathing; apnea-hypopnea index; polysomnography; BMI percentile.

INTRODUCTION

Obstructive sleep apnea is a common pediatric sleep-related breathing disorder characterized by repeated partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxemia, sleep fragmentation, and altered ventilation[1,2]. Pediatric OSA differs from adult OSA in clinical presentation, pathophysiology, and diagnostic thresholds, and even relatively low apnea-hypopnea index values may be clinically important in children[1,2].

Childhood obesity has emerged as a major public health concern and is commonly defined using age- and sex-specific BMI percentiles, with obesity corresponding to BMI-for-age at or above the 95th percentile[3]. The rising prevalence of pediatric obesity has changed the clinical phenotype of childhood OSA. Traditionally, adenotonsillar hypertrophy was considered the dominant risk factor, particularly in younger children; however, obesity is increasingly recognized as an important contributor, especially among older children and adolescents[4,5].

The relationship between obesity and OSA is biologically plausible. Fat deposition around the upper airway can narrow the pharyngeal lumen, while central adiposity may reduce lung volumes and increase upper airway collapsibility during sleep[6,7]. Obesity-related systemic inflammation, insulin resistance, and altered leptin signaling may further contribute to ventilatory instability and cardiometabolic consequences[7,8].

Several studies have reported that overweight and obese children have a higher prevalence of OSA and more severe polysomnographic abnormalities than children with healthy weight[4,7,9]. However, the magnitude of this association may vary by age, pubertal status, ethnicity, tonsillar size, and referral population[9,10]. Clarifying the relationship between obesity and OSA severity is important because children with obesity may require closer perioperative monitoring, objective follow-up after adenotonsillectomy, and combined treatment strategies including weight management[1,6].

The present study was conducted to evaluate the association between childhood obesity and OSA severity among 135 children undergoing polysomnography for suspected sleep-disordered breathing.

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MATERIAL AND METHODS

This was a cross-sectional analytical study conducted at a tertiary care hospital in Pakistan from April 2023 to September 2023. Children presenting with symptoms suggestive of sleep-disordered breathing were enrolled consecutively. A total of 135 children aged 5–17 years were included. Children were included if they had one or more symptoms suggestive of OSA, including habitual snoring, witnessed apneas, restless sleep, mouth breathing, daytime sleepiness, morning headache, poor school performance, or behavioral symptoms, and underwent overnight polysomnography. Children were excluded if they had craniofacial syndromes, neuromuscular disease, Down syndrome, chronic lung disease, congenital heart disease, previous adenotonsillectomy, current use of continuous positive airway pressure, or incomplete polysomnographic data.

Anthropometric assessment: Weight was measured using a calibrated scale and height was measured using a stadiometer. BMI was calculated as weight in kilograms divided by height in meters squared. BMI-for-age percentile was determined according

to age- and sex-specific reference charts. Children with BMI-for-age at or above the 95th percentile were classified as obese, while those below the 95th percentile were classified as non-obese.

Clinical assessment: A structured proforma was used to record age, sex, presenting symptoms, duration of snoring, witnessed apnea, daytime sleepiness, mouth breathing, allergic rhinitis, asthma history, and tonsillar grade. Tonsillar hypertrophy was defined as grade III or IV tonsils on clinical examination.

Polysomnography: All participants underwent attended overnight polysomnography. Recorded parameters included electroencephalography, electro-oculography, chin and limb electromyography, airflow, thoracoabdominal movement, oxygen saturation, body position, snoring, and heart rate. Respiratory events were scored according to standard pediatric sleep-scoring criteria. The obstructive apnea-hypopnea index was calculated as the number of obstructive apneas, mixed apneas, and hypopneas per hour of total sleep time.

OSA severity classification: OSA severity was classified as follows: no OSA/minimal disease: OAHI <1 event/hour; mild OSA: OAHI 1-4.9 events/hour; moderate OSA: OAHI 5-9.9 events/hour; and severe OSA: OAHI >=10 events/hour. For regression analysis, moderate and severe OSA were combined as moderate-to-severe OSA.

Statistical analysis: Data were analyzed using standard statistical methods. Quantitative variables were expressed as mean +/- standard deviation or median with interquartile range. Qualitative variables were expressed as frequency and percentage. The chi-square test was used for categorical variables. The independent samples t-test or Mann-Whitney U test was used for continuous variables where appropriate. Logistic regression was performed to determine the independent association between obesity and moderate-to-severe OSA after adjustment for age, sex, and tonsillar hypertrophy. A p-value <0.05 was considered statistically significant.

RESULTS

The study included 135 children. The mean age was 11.1 +/- 3.3 years. There were 78 males (57.8%) and 57 females (42.2%). Obesity was present in 57 children (42.2%), while 78 children (57.8%) were non-obese.

Habitual snoring was the most common presenting symptom, reported in 120 children (88.9%). Witnessed apnea was reported in 73 children (54.1%), mouth breathing in 81 children (60.0%), and daytime sleepiness in 52 children (38.5%). Tonsillar hypertrophy was present in 75 children (55.6%).

Obese children had significantly worse polysomnographic parameters compared with non-obese children. Median OAHI was 9.6 events/hour in the obese group compared with 3.8 events/hour in the non-obese group. Mean oxygen saturation nadir was lower in obese children.

Table 1: Baseline characteristics of included children

Variable	Total n=135	Non-obese n=78	Obese n=57	p-value
Age, years, mean +/- SD	11.1 +/- 3.3	10.8 +/- 3.4	11.6 +/- 3.2	0.18
Male sex, n (%)	78 (57.8)	42 (53.8)	36 (63.2)	0.28
BMI z-score, mean +/- SD	1.29 +/- 1.03	0.62 +/- 0.78	2.21 +/- 0.46	<0.001
Habitual snoring, n (%)	120 (88.9)	67 (85.9)	53 (93.0)	0.21
Witnessed apnea, n (%)	73 (54.1)	36 (46.2)	37 (64.9)	0.03
Mouth breathing, n (%)	81 (60.0)	44 (56.4)	37 (64.9)	0.32
Daytime sleepiness, n (%)	52 (38.5)	25 (32.1)	27 (47.4)	0.07
Tonsillar hypertrophy grade III/IV, n (%)	75 (55.6)	41 (52.6)	34 (59.6)	0.42

Table 2: Polysomnographic parameters according to obesity status

Parameter	Non-obese n=78	Obese n=57	p-value
Total sleep time, minutes, mean +/- SD	405 +/- 52	396 +/- 58	0.34
Sleep efficiency, %, mean +/- SD	84.6 +/- 8.5	82.9 +/- 9.1	0.26
OAHI, events/hour, median (IQR)	3.8 (1.6-7.5)	9.6 (4.7-18.2)	<0.001
Oxygen desaturation index, median (IQR)	4.1 (1.9-8.8)	10.8 (5.2-19.4)	<0.001
Oxygen saturation nadir, %, mean +/- SD	89.2 +/- 4.8	84.8 +/- 6.2	<0.001
Arousal index, events/hour, median (IQR)	8.6 (5.1-13.4)	13.9 (8.2-21.5)	0.002

Table 3: Distribution of OSA severity according to obesity status

OSA severity	Non-obese n=78	Obese n=57	Total n=135
No OSA/minimal disease	17 (21.8%)	4 (7.0%)	21 (15.6%)
Mild OSA	35 (44.9%)	16 (28.1%)	51 (37.8%)
Moderate OSA	16 (20.5%)	17 (29.8%)	33 (24.4%)
Severe OSA	10 (12.8%)	20 (35.1%)	30 (22.2%)
Moderate-to-severe OSA	26 (33.3%)	37 (64.9%)	63 (46.7%)

Table 4: Predictors of moderate-to-severe OSA on logistic regression

Variable	Adjusted odds ratio	95% CI	p-value
Obesity	3.12	1.48-6.59	0.003
Age >=12 years	1.42	0.68-2.96	0.35
Male sex	1.29	0.61-2.73	0.50
Tonsillar hypertrophy grade III/IV	2.21	1.05-4.66	0.037

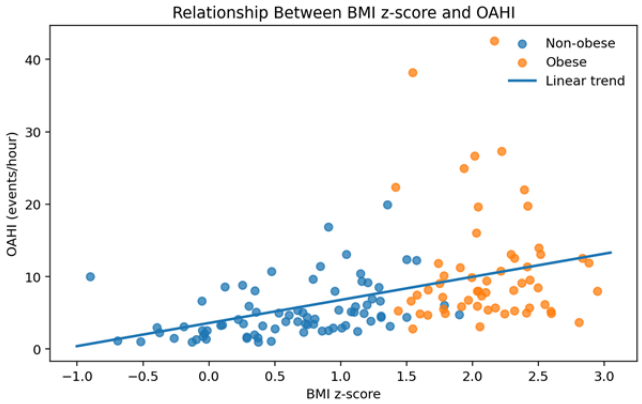

Figure 2: Relationship between BMI z-score and OAHI.

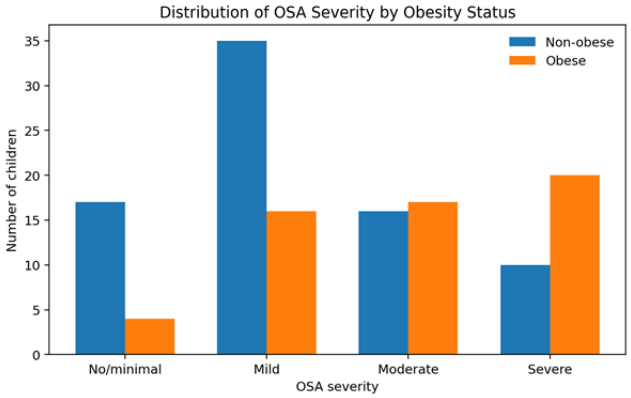

Figure 1: Distribution of OSA severity by obesity status.

Moderate-to-severe OSA was present in 63 children (46.7%). Among obese children, 37 (64.9%) had moderate-to-

severe OSA, compared with 26 (33.3%) among non-obese children. Severe OSA was observed in 20 obese children (35.1%) and 10 non-obese children (12.8%).

Chi-square analysis showed a significant association between obesity status and OSA severity category (p=0.001). Obese children had 3.70 times higher odds of moderate-to-severe OSA than non-obese children (95% CI 1.80-7.60; p<0.001).

Multivariable analysis: After adjustment for age, sex, and tonsillar hypertrophy, obesity remained significantly associated with moderate-to-severe OSA. Tonsillar hypertrophy was also independently associated with moderate-to-severe OSA.

A positive relationship was observed between increasing BMI z-score and increasing OAHI. The correlation between BMI z-score and OAHI was moderate and statistically significant (r=0.46; p<0.001), suggesting that higher adiposity was associated with greater OSA severity.

DISCUSSION

In this study of 135 children evaluated for suspected sleep-disordered breathing, childhood obesity was significantly associated with greater OSA severity. Obese children had higher median OAHI, higher oxygen desaturation index, lower oxygen saturation nadir, and a higher frequency of moderate-to-severe OSA compared with non-obese children. After adjustment for age, sex, and tonsillar hypertrophy, obesity remained an independent predictor of moderate-to-severe OSA.

The findings are consistent with previous pediatric studies showing that obesity increases the risk and severity of OSA[11-13]. Kohler et al. reported that obesity increased the risk of sleep-related upper airway obstruction in children, while other studies have shown a positive association between BMI z-score and OSA severity, particularly among older children and adolescents[11,12]. A recent study of children with obesity also demonstrated that increasing obesity class was associated with higher odds of severe OSA[13].

Several mechanisms may explain this association. Excess adipose tissue around the neck and pharyngeal structures can reduce upper airway caliber and increase collapsibility during sleep[14]. Abdominal and thoracic adiposity may reduce functional residual capacity, thereby decreasing caudal traction on the upper airway and predisposing to obstruction[14,15]. Obesity is also associated with systemic inflammation, oxidative stress, insulin resistance, and leptin dysregulation, which may impair respiratory control and contribute to intermittent hypoxemia[16,17].

The present study also showed that obese children had lower oxygen saturation nadirs and higher oxygen desaturation indices. These findings suggest that obesity may not only increase the number of obstructive respiratory events but may also worsen their physiological consequences. Intermittent hypoxemia and sleep fragmentation are clinically important because they have been linked to neurocognitive, behavioral, cardiovascular, and metabolic complications in children[16-19].

Tonsillar hypertrophy was also independently associated with moderate-to-severe OSA in this study. This supports the concept that pediatric OSA is multifactorial. In younger children, adenotonsillar hypertrophy may dominate, whereas in older children and adolescents, obesity-related airway and ventilatory factors may become more prominent[1,20]. The coexistence of obesity and adenotonsillar hypertrophy may therefore identify a subgroup at particularly high risk of clinically significant OSA.

The clinical implications are important. First, children with obesity and symptoms such as habitual snoring, witnessed apnea, mouth breathing, or daytime sleepiness should be prioritized for objective sleep evaluation. Second, obese children diagnosed with OSA may require more comprehensive treatment than adenotonsillectomy alone. Weight management, treatment of allergic rhinitis, positive airway pressure therapy, orthodontic evaluation, and careful follow-up may be needed depending on severity and residual symptoms[1,6,21]. Third, because residual OSA after adenotonsillectomy is more common in obese children, repeat polysomnography should be considered when symptoms persist or when baseline disease is moderate to severe[21,22].

The results are also relevant to perioperative planning. Severe OSA and obesity are both associated with increased risk of postoperative respiratory complications, making preoperative PSG and postoperative monitoring important in selected high-risk children[1,6,22]. Early recognition allows clinicians to counsel families more appropriately and plan multidisciplinary care.

The strength of this study was the use of polysomnography-based OSA classification rather than symptom-based diagnosis alone. The study also adjusted for tonsillar hypertrophy, which is an important confounder in pediatric OSA. However, some limitations should be noted. The cross-sectional design does not prove causality. The study was conducted among children referred for suspected sleep-disordered breathing; therefore, the findings may not represent the general pediatric population. Pubertal stage, waist circumference, neck circumference, and socioeconomic factors were not included in the final regression model. Finally, treatment outcomes were not evaluated.

Despite these limitations, the study supports a clinically meaningful association between childhood obesity and OSA severity. These findings reinforce the need for integrated evaluation of sleep, growth, airway anatomy, and weight status in children presenting with symptoms of sleep-disordered breathing.

CONCLUSION

Childhood obesity was significantly associated with increased severity of obstructive sleep apnea among children undergoing polysomnography. Obese children had higher OAHI values, more oxygen desaturation, lower oxygen saturation nadirs, and higher odds of moderate-to-severe OSA. Screening for OSA should be emphasized in obese children with snoring or other symptoms of sleep-disordered breathing. Multidisciplinary management, including weight optimization and sleep-focused treatment, may improve outcomes in this high-risk population.

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