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Polysomnographic Outcomes After Observation for Mild Obstructive Sleep Apnea in Children Younger Than 3 Years

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Abstract

Objective.—Mild obstructive sleep apnea (OSA), particularly in young children, is often treated with observation. However, there is little evidence regarding the outcomes with this approach. Our aim was to assess the impact of observation on sleep for children aged <3 years with mild OSA.

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Study Design.—Case-control study.

Setting.—Pediatric tertiary care center.

Methods.—We reviewed cases of children (<3 years old) diagnosed with mild OSA (obstructive apnea-hypopnea index, 1–5 events/h) who were treated with observation between 2012 and 2017 and had at least 2 polysomnograms performed 3 to 12 months apart. Demographic data and comorbid diagnoses were collected.

Results.—Twenty-six children met inclusion criteria; their median age was 7.2 months (95% CI, 1.2–22.8). Nine (35%) were female and 24 (92%) were White. Their median body mass index percentile was 39 (95% CI, 1–76). Comorbidities included cardiac disease (42.3%), laryngomalacia (42.3%), allergies (34.6%), reactive airway disease (23.1%), and prematurity (7.7%). The obstructive apnea-hypopnea index significantly decreased from 2.7 events/h (95% CI, 1–4.5) to 1.3 (95% CI, 0–4.5; $P = .013$). There was no significant improvement in median saturation nadir (baseline, 86%; $P = .76$) or median time with end-tidal carbon dioxide >50 mm Hg (baseline, 0 minutes; $P = .34$). OSA resolved in 8 patients (31%) and worsened in 1 (3.8%). Only race was a significant predictor of resolution per regression analysis; however, only 2 non-White children were included.

Conclusion.—In our cohort, resolution of mild OSA occurred in 31% of patients treated with 3 to 12 months of observation. The presence of laryngomalacia, asthma, and allergies did not affect resolution. Larger studies are needed to better identify factors (including race) associated with persistent OSA and optimal timing of intervention for these children.

Keywords

obstructive sleep apnea; mild; observation; infant; pediatric; children

Obstructive sleep apnea (OSA) is a disease of disordered breathing during sleep, comprising episodes of complete or partial airway obstruction that include apneas, hypopneas, and impaired ventilation, and it can result in increased arousals.[1] Epidemiologic studies suggest that the prevalence of OSA in children is 1% to 6%. However, variability in the criteria used to diagnose pediatric OSA in the literature presents challenges to reporting accuracy.[2] Overnight polysomnogram (PSG) is the gold standard evaluation for the diagnosis of OSA. It records the frequency and degree of respiratory and sleep disturbances, and it enables the physician to stratify the severity of disease.

The diagnosis of OSA in very young children is complicated by the heterogeneity and age-dependent variability of signs and symptoms at the time of presentation.[3] The most common presenting complaint in school-aged children diagnosed with OSA is snoring, an attribute previously identified as a potential independent risk factor for the future development of OSA.[2,4] However, snoring is not a prominent symptom in very young children and infants with significant OSA.[5,6] In addition, there is significant overlap in the clinical characteristics of infants with normal sleep and those with sleep-disordered breathing, making it difficult to clinically distinguish between the conditions without further testing. For example, 22% to 26% of normal infants have noisy breathing and paradoxical breathing that may mimic obstructive events during rapid eye movement sleep during the first 3 years of life.[7,8]

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The clinical implications of mild OSA in infants and young children are also unclear.[9] While it is recognized that some obstructive events are part of the normal spectrum of breathing in an otherwise healthy infant, the point at which they become pathologic has yet to be determined.[9,10] Some studies have demonstrated a correlation between neurocognitive impairment and the degree of sleep-disordered breathing in children, while others have refuted these findings.[11–14]

Due to the ambiguous nature of mild OSA in young children, previous authors have advocated for observation in the absence of concerning objective findings on PSG (hypoventilation, significant hypoxia or desaturations, etc).[9,15] Furthermore, as the primary etiology of the OSA may not be clear at such an early age, providers and families alike may be hesitant to proceed with treatment and its associated risks in the absence of severe symptoms. However, the efficacy of watchful waiting is unknown in this age group. Few studies have attempted to delineate the natural history of mild OSA in pediatric patients.[10,16–18] Only 1 of these studies included infants, and the majority of those children were older than 3 years, as the mean age of patients being observed in that sample was 4.5 years.[17] The aim of our study was to assess the impact of observation on the polysomnographic outcomes and to evaluate for factors associated with worsening or improvement in apnea-hypopnea index (AHI) in children younger than 3 years with mild OSA.

Methods

We performed a retrospective chart review of all patients younger than 3 years with initial overnight PSG showing mild OSA treated by observation alone between 2012 and 2017 and with a follow-up PSG performed between 3 and 12 months apart. Responders were defined as children whose obstructive AHI (oAHI; the number of obstructive and mixed events per hour of sleep) was <1 event/h on the follow-up study. Patients without a follow-up sleep study, those with sleep studies >1 year apart or <3 months apart, and children >3 years old were excluded. Patients were also excluded if they had a history of neuromuscular disease, current tracheostomy, or interstitial lung disease. Demographic data and comorbid diagnoses were collected. We assessed pre- and postobservation AHI, oAHI, oxygen saturation nadir, percentage of total sleep time with end-tidal carbon dioxide ($ETCO_2$) >50 mm Hg, sleep efficiency, arousal index, and percentage of stage R sleep. This study was approved by the institutional review board at the Cincinnati Children's Hospital Medical Center.

Polysomnography

All overnight PSGs were performed in an accredited sleep center at Cincinnati Children's Hospital Medical Center. Standard PSG parameters were recorded simultaneously: body position, bilateral electrooculogram (ROC/A1, LOC/A2), 6-channel electroencephalogram (F3A2, F4A1, C3A2, C4A1, O1A2, O2A1), chin electromyogram, anterior tibialis electromyogram, tracheal microphone, electrocardiogram, pulse oximetry, thoracic and abdominal inductance plethysmography, nasal pressure transduction, and $ETCO_2$ (BCI Capnogard). Scoring of the PSG was performed according to criteria defined by the American Academy of Sleep Medicine.[19] Sleep stage and respiratory scoring were performed by a certified sleep technician and interpreted by a board-certified sleep

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physician. An obstructive apnea was defined as a cessation or decrease in airflow or a decrease in the sum channel from inductive plethysmography by >90% of the preceding breath with retained effort during the episode. An obstructive hypopnea was defined as a decrease in airflow or a decrease in the sum channel from inductive plethysmography by ≥30% when compared with the preceding breaths and was associated with an oxygen desaturation ≥3%, an arousal, or an awakening. All obstructive events were ≥2 breaths' duration. The number of apneas (including central apneas) and hypopneas per hour were calculated and reported as the AHI. Mild OSA was defined as an oAHI ≥1 and ≤5 events/h. Oxyhemoglobin nadir was determined by the lowest oxygen saturation data point during an obstructive event. The changes in PSG parameters between baseline and follow-up were analyzed, including oAHI, AHI, central apnea index, $ETCO_2$ levels, oxygen saturation nadir, sleep efficiency, arousal index, and stage R sleep percentage.

Statistical Analysis

Descriptive statistics were used to examine the demographic data. Data distributions were reported as medians with 95% CIs for continuous variables and frequencies with percentages for categorical variables. Baseline and follow-up PSG values were tested with the Wilcoxon signed rank test. Univariable regression analysis was carried out to evaluate factors associated with the resolution of OSA. Multivariable regression was then carried out to evaluate the relationship of OSA resolution with demographics and comorbid conditions.

Results

Fifty-six patients younger than 3 years demonstrated mild OSA on baseline PSG, and 30 (53.6%) of these patients were excluded as they did not undergo repeat PSG. Twenty-six children met all inclusion criteria; their median age was 7.2 months (95% CI, 0.2–1.9). Nine (35%) patients in the group were female; 24 (92%) were identified as White, 1 (3.9%) as Black, and 1 (3.9%) classified as "other." The median body mass index percentile (for children older than 2 years) was 39 (95% CI, 1–76). Comorbidities included Down syndrome (11.5%), cardiac disease (42.3%), reactive airway disease/asthma (23.1%), hypertension (7.7%) and prematurity (7.7%). Demographic data for the cohort are presented in Table 1.

Cardiorespiratory Parameters

Baseline and follow-up PSG parameters are presented in Table 2. The oAHI decreased significantly from a median 2.7 events/h (95% CI, 1–4.5) to 1.3 (95% CI, 0–4.5; $P = .013$) after a median observation period of 6.6 months (95% CI, 3.6–11.1). The median AHI decreased from 4.3 to 3.4 events/h; however, this change was not significant ($P = .19$). The median oxyhemoglobin saturation nadir was 86% (95% CI, 75–96) and did not change significantly at follow-up ($P = .76$). No patients exhibited hypoventilation. Median sleep time with $ETCO_2$ >50 mm Hg was 0 minutes (95% CI, 0–19.7) at baseline and did not change significantly at follow-up ($P = .34$). OSA resolved in 8 patients (30.7%), remained mild in 17 (65.4%), and worsened in 1 (3.9%). There was no difference in resolution rates for those children with (36%) or without (27%) laryngomalacia ($P = .98$).

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Sleep Architecture

The median sleep efficiency improved from 78% (95% CI, 75%−96%) to 83% (95% CI, 65%−92%), though this was not significant ($P = .09$). The median arousal index decreased significantly from 14.7/h (95% CI, 5.4–26.8) to 13.0/h (95% CI, 2.8–22.3; $P = .027$). The median percentage time spent in stage R sleep decreased significantly from 33% (95% CI, 18%−54%) to 30% (95% CI, 14%−36%; $P = .008$).

Regression Analysis

Only race was significantly associated with resolution of OSA (oAHI ≤1) at the time of follow-up PSG with univariable and multivariable regression when controlling for age at baseline PSG, sex, genetic syndrome, allergies, reactive airway disease/asthma, prematurity, and laryngomalacia.

Discussion

In this study, we investigated the changes in objective PSG parameters for children with mild OSA younger than 3 years. Our cohort had a resolution rate of 31%, with 1 patient experiencing worsening of the oAHI to moderate OSA. We found that only race was significantly associated with resolution of mild OSA on univariable and multivariable regression analysis.

Our resolution rate of 31% is within the range of previously published studies (20%−70%) for older children.[10,16,17] It is, however, lower than the 85% resolution rate reported in a study of infants with OSA.[20] These discrepancies may be due to the differences in comorbidities of the studied populations. Notably, 11% of our patient cohort had Down syndrome; 42%, cardiac disease; 42%, laryngomalacia; and 23% reactive airway disease, asthma, or both. Down syndrome and genetic conditions associated with craniofacial abnormalities have been associated with more prevalent and severe OSA, with OSA associated with >1 site of anatomic obstruction, and with a higher rate of persistent OSA after treatment.[21–23] It is not known if cardiac disease is a risk factor for OSA in children, but atrial fibrillation and congestive heart failure are known to be associated with OSA in adults.[24] The pathophysiology for this association is likely a complex interaction of inflammation, oxidative stress, endothelial dysfunction, and intrathoracic pressure receptor abnormalities.[25]

The high incidence of laryngomalacia within our cohort may also have contributed to the persistence of OSA among affected patients, although it was not significant in regression or subgroup analysis. While there is adequate literature evaluating the natural course of congenital laryngomalacia in terms of feeding and stridor, no literature explores how laryngomalacia affects the natural history of OSA and whether OSA is expected to resolve as other symptoms of laryngomalacia resolve. One retrospective study of 139 infants aged 0 to 17 months with OSA found that 27% had laryngomalacia or tracheomalacia. The diagnosis of laryngomalacia was not statistically correlated with more severe OSA.[26] A few studies also examined polysomnographic outcomes of pediatric patients with laryngomalacia and OSA treated with supraglottoplasty, although none specifically examined patients

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treated with observation for mild OSA. One retrospective case series of 10 infants with laryngomalacia between the ages of 1 and 9 months reported a 72% decrease in the oAHI on PSG after supraglottoplasty for OSA.[27] A recent meta-analysis of outcomes after supraglottoplasty for children <18 years old with laryngomalacia and OSA showed that supraglottoplasty was associated with improvement in AHI for all patient groups; however, the authors did not stratify findings by OSA severity, and the cure rate was low (10% −26.5%).[28] These studies suggest that the presence of laryngomalacia may indeed be a significant contributor to persistent OSA after treatment, although its presence in our study did not impair spontaneous resolution as compared with those without laryngomalacia.

Reactive airway disease has been shown to have a bidirectional effect on OSA, with worsening and improving asthma control associated with increasing and decreasing AHI, respectively. In addition, asthma is associated with higher rates of residual disease after treatment.[21] Our cohort included children with prematurity (7.7%), which has also been implicated as a factor associated with OSA.[29] Interestingly, in our population, none of these comorbidities were associated with persistence of OSA on multivariable regression modeling. This may be explained by the lower numbers included in our study.

Our study showed Black race to be predictive of persistent OSA. Black race has been identified as an independent risk factor for sleep-disordered breathing,[2] as well as an independent predictor of persistence of OSA after adenotonsillectomy.[10] Although race alone was significant in multivariable regression modeling, these results need broader verification, as there were only 2 non-White children in this cohort.

Without a strong body of literature investigating this topic, the short- and long-term effects of mild OSA on young children remain undefined. Although it has been demonstrated that healthy children have an AHI <1 event/h, current literature does not uniformly agree on AHI cutoffs to define OSA, particularly for very young infants.[30] The majority of existing studies examining outcomes of observation versus intervention (medical or surgical) in mild OSA focused on neurocognitive, behavioral, and quality-of-life measures, which are less useful in the context of evaluating young children and infants.[10,13,18] A recent cross-sectional study characterized the comorbidities of infants with OSA between 0 and 17 months of age and found that GERD (gastroesophageal reflux disease), periodic leg movements in sleep, and craniofacial abnormalities were the most common coexisting conditions (68%, 42%, and 37%, respectively). Prematurity and failure to thrive were associated with more severe OSA in their cohort.[29] Another study suggested that treating moderate and severe OSA results in a growth benefit in infants with failure to thrive.[31] However, there are limited studies evaluating longitudinal outcomes in young children with mild OSA and associated comorbidities. Only 1 study followed children with mild OSA for 2 years postdiagnosis.[16] Our study contributes to this limited literature aimed at improving our understanding of the natural history of mild OSA in young children.

Limitations of this study include selection bias inherent to any retrospective review. It was more likely for children to be referred for a second PSG if they were still symptomatic, while children who had improvement in symptoms were more likely to be lost to follow-up and less likely to undergo a repeat PSG. In our study, about half of the infants with mild

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OSA underwent a repeat PSG. A review of the 30 children who did not undergo another PSG revealed that 17 were lost to follow-up (30.3% of the total 56 children), and 13 were reevaluated clinically. Of those, 8 children (14.3% overall) demonstrated resolution of presenting symptoms, and a repeat PSG was determined to be unnecessary; 5 children with continued symptoms were recommended to repeat a PSG but did not undergo the study. While the degree of selection bias is difficult to quantify, an observational comparison of the trends over time remains of interest.

Our study also has a small sample size, making any analysis of race or other demographic features limited. In addition, comorbid diseases such as GERD and chronic lung disease were not well represented in our cohort but have been associated with OSA; thus, these data may not be generalizable to that population. Last, there was no control sample of normal-sleeping children. Despite these limitations, our study does contribute interesting information to a small but growing body of literature.

Conclusion

In our cohort, resolution of mild OSA after a period of observation occurred in about one-third of children <3 years of age observed for 3 to 12 months. The presence of laryngomalacia and other factors did not affect the resolution rate. In addition, our analysis suggests that the impact of race on the likelihood of OSA resolution should be further assessed and that larger studies are needed to better identify factors associated with persistent disease and the optimal timing of intervention in young children with mild OSA.

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Table 1.

Baseline Patient Characteristics for Young Children With Mild Obstructive Sleep Apnea.

	Patients <3 y old (N = 26)
Age, y, median (95% CI)	0.6 (0.2–1.9)
Sex, female, No. (%)	9 (34.6)
Race, No. (%)	
White	24 (92.3)
Black	1 (3.9)
Other	1 (3.9)
Body mass index percentile (95% CI)	39 (1–76)
Comorbidities, No. (%)	
Laryngomalacia	11 (42.3)
Cardiac disease	11 (42.3)
Allergies	9 (34.6)
Asthma	6 (23.1)
Down syndrome	3 (11.5)
Hypertension	2 (7.7)
Premature birth	2 (7.7)
Diabetes	0 (0)

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Table 2.

Polysomnographic Parameters at Baseline and Follow-up for Children Aged <3 Years With Mild Obstructive Sleep Apnea Treated With Observation for 3 to 12 Months.[a]

	Baseline	Follow-up	*P* value
oAHI, events/h	2.7 (1.0–4.5)	1.3 (0–4.5)	**.013**
AHI, events/h	4.3 (2.2–7.9)	3.4 (0.5–8.1)	.19
Central apnea index, events/h	1.4 (0–3.8)	1.2 (0.1–4.7)	.60
Oxyhemoglobin nadir, %	86 (75–96)	88 (73–95)	.76
Sleep efficiency, % total sleep time	78 (49–90)	83 (65–92)	.09
Arousal index, event/h	14.7 (5.4–26.8)	13.0 (2.8–22.3)	**.027**
$ETCO_2$ >50 mm Hg, min	0 (0–19.7)	0 (0–10.3)	.34
Percentage of REM sleep	33 (18–54)	30 (14–36)	**.008**
Posttreatment OSA severity, No. (%)			
None	0 (0)	8 (30.7)	
Mild	26 (100)	17 (65.4)	
Moderate	0 (0)	1 (3.8)	

Abbreviations: AHI, apnea-hypopnea index; $ETCO_2$, end-tidal carbon dioxide; oAHI, obstructive apnea-hypopnea index; OSA, obstructive sleep apnea; REM, rapid eye movement.

[a]Values are presented as median (95% CI) unless noted otherwise. Bold indicates $P<.05$.

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