Adenotonsillectomy for Obstructive Sleep Apnea in Children with Complex Chronic Conditions

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Abstract
Objective. To estimate the prevalence of persistent obstructive sleep apnea postadenotonsillectomy in children with complex chronic conditions.

Study Design. A retrospective cohort study.

Setting. The Hospital for Sick Children Sleep Laboratory.

Subjects. Children ≤18 years of age who had moderate to severe obstructive sleep apnea and had polysomnography pre- and postadenotonsillectomy.

Methods. Medical and polysomnographic data were reviewed. The prevalence of persistent obstructive sleep apnea postadenotonsillectomy was determined for the following groups: no complex chronic conditions, single-system complex chronic conditions, and multisystem complex chronic conditions.

Results. We reviewed data of 133 (84 male) children. Their mean (standard deviation) age was 5.5 (3.8) years. The persistent obstructive sleep apnea rate postadenotonsillectomy was highest in children with multisystem complex chronic conditions (57%), intermediate in children with single-system complex chronic conditions (29%), and lowest in children without complex chronic conditions (15%), P = .0004. The odds (confidence interval) of having persistent obstructive sleep apnea postadenotonsillectomy was 7.42 (2.16-25.51) times higher in children with multisystem complex chronic conditions vs no complex chronic conditions and 3.35 (1.16-9.64) times higher in children with multisystem complex chronic conditions vs single-system complex chronic conditions.

Conclusions. Although adenotonsillectomy is considered first-line therapy in healthy children older than 2 years for the treatment of obstructive sleep apnea, there is a significantly greater risk of persistent obstructive sleep apnea postadenotonsillectomy in children with complex chronic conditions. Therefore, other surgical procedures or nonsurgical management may need to be considered as first-line treatment for this cohort.

Keywords
adenotonsillectomy, children, sleep apnea, obstructive sleep apnea, surgery, complex chronic conditions

Medical and technological advances have allowed an increasing number of children with complex chronic conditions (CCCs) to survive.1,2 A CCC is one that lasts more than 12 months, involves 1 or several organ systems, and requires a high level of specialty care.3 Children with CCCs are diagnostically heterogeneous and require multiple interventions (ie, frequent hospitalizations, intensive community care services, medical technology at home) to maintain their health.4,5 Even though they represent less than 1% of the pediatric population, children with CCCs account for over one-third of pediatric health care costs because they are at high risk of morbidity and mortality.6-10

Obstructive sleep apnea (OSA) is the most common type of sleep-disordered breathing (SDB) in children. OSA affects 2% to 4% of healthy children,11 and this prevalence in children with underlying comorbidities such as trisomy 21, Chiari malformations, and neuromuscular disease is significantly higher, reaching over 50%.12-14 OSA is characterized by repeated episodes of partial or complete upper airway obstruction associated with oxygen desaturation and/or sleep fragmentation. In healthy children, the most common etiological factor for OSA is adenotonsillar hypertrophy, and adenotonsillectomy (AT) is curative more than

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70% of the time.\textsuperscript{15-17} Success rates are significantly lower in children with underlying comorbidities (eg, trisomy 21, craniofacial abnormalities, obesity).\textsuperscript{18-20} Outcomes following AT in children with CCCs have not been investigated, and predictors of persistent OSA following AT have not been elucidated. The aim of this study was to determine the prevalence of persistent OSA post-AT in children with CCCs and to identify predictors of persistent postoperative OSA to help guide selection of surgical candidates.

Materials and Methods

Subjects

This study was approved by the Research Ethics Board at the Hospital for Sick Children (SickKids), University of Toronto, Canada (REB #1000045745). The sleep laboratory database at SickKids was retrospectively reviewed. We reviewed data for children referred to the sleep laboratory between May 1, 2007, and December 31, 2015. Inclusion criteria for the review were as follows: children younger than 18 years who had moderate to severe OSA that required AT and had polysomnography (PSG) pre- and post-AT. Children who had other surgical procedures other than AT or those who had been initiated on noninvasive therapy in between the 2 PSGs were excluded from the review.

The health care record was reviewed to collect data on baseline patient demographics, anthropometrics (age, sex, body mass index [BMI], height, weight), and pre- and post-AT PSG results. The primary medical diagnosis was collected from the medical records and classified and coded according to the International Classification of Diseases, 10th Revision. From the collected data on medical diagnosis, the CCC was defined using the Feudtner et al\textsuperscript{13} classification system: a diagnosis lasting more than 12 months, involving 1 or several organ systems, and requiring a high level of specialty care. The CCCs for each patient were then further categorized as having single or multiple system involvement. Children were divided a priori into 3 groups: (1) non-CCC group: healthy children and children with a primary medical diagnosis that did not meet a CCC definition; (2) single-system CCC group: children with a CCC diagnosis involving a single system; and (3) multisystem CCC group: CCC diagnoses that involve 2 or more system categories.

Polysomnography

All participants underwent 2 standard level 1 overnight baseline PSGs before and after AT using the Natus SleepWorks (Pleasanton, California) data acquisition and analysis system. Sleep architecture and respiratory data were assessed,\textsuperscript{21} and information was obtained from PSG and scored according to the American Academy of Sleep Medicine (AASM) scoring guidelines by a registered polysomnographic technician.\textsuperscript{22} Respiratory events were scored according to the respiratory rules for children in the AASM guidelines.\textsuperscript{22} Recorded respiratory data included counts and indices of the following events: obstructive apneas, obstructive hypopneas, central apneas, and mixed apneas during sleep. The obstructive apnea-hypopnea index (OAHI) was defined as the number of obstructive apneas, hypopneas, and mixed apneas per hour. OSA severity was graded according to OAHI. OAHI of <2 was considered normal, OAHI from ≥2 to <5 was mild OSA, OAHI from ≥5 to <10 was moderate OSA, and OAHI ≥10 was considered severe OSA.\textsuperscript{16,23-25} In our center as part of standard of practice in relation to the risks vs benefits of the surgery, children were considered for AT when the OAHI was ≥5 events per hour of sleep, whereas medical therapy such as nasal steroids was indicated for those with mild OSA (OAHI from ≥2 to <5 events per hour of sleep). AT was considered to have failed when the OAHI remained ≥5 events per hour of sleep postsurgery. We did not use lower cutoff values for OAHI to determine success of AT as it has been shown that only 30% of healthy children with OSA will have OAHI improved below 1 event per hour of sleep (cured) post-AT, and several centers have used OAHI ≥5 events per hour to be the criterion for AT failure.\textsuperscript{17}

Study Outcome Measures

The primary outcome measure for the study was the prevalence of persistent moderate to severe OSA post-AT. The identification of predictors of persistent OSA post-AT was the secondary outcome measure.

Statistical Analysis

Descriptive statistics were used to summarize the study population. Baseline characteristics, primary diagnosis frequency, and PSG results were reported as mean with standard deviation (SD) for normally distributed continuous variables and median with interquartile range for nonnormally distributed variables. Frequencies and percentages were used to summarize categorical variables. Analysis of variance (ANOVA) was used to analyze baseline PSG results among the different CCC groups. This same comparison was performed using the Kruskal-Wallis test for OAHI.

For the primary analysis, descriptive statistics were used to determine the prevalence of persistent OSA post-AT. Children with a significant improvement in OAHI post-AT (OAHI post-AT <5 events per hour of sleep) were compared to those who did not improve post-AT (OAHI post-AT ≥5 events per hour of sleep); Student t test, Wilcoxon rank sum, and χ² testing were used for normally distributed continuous variables, nonnormally distributed continuous variables, and categorical variables, respectively. For comparisons between PSG data pre- and post-AT, paired t tests were used for all variables except OAHI; Wilcoxon matched-pairs signed rank-sum test was used for this comparison.

For the analysis of the secondary outcome, univariate and multivariable logistic regression was used to evaluate the effect of the covariates (age, sex, BMI, time from AT to follow-up PSG, primary diagnosis, and baseline OAHI) on the primary outcome variable: persistent OSA post-AT. Statistical significance was set at \( P < .05 \). All statistical
analyses were performed using SAS version 9.3 (SAS Institute, Cary, North Carolina).

Results

Baseline Characteristics of the Study Population

A total of 133 children (84 [63%] male) were included in this study with a mean age (SD) of 5.5 (3.8) years and mean (SD) BMI of 19.8 (6.8) kg/m². Patient demographics are summarized in Table 1. Forty-seven (35%) patients did not have a CCC, whereas 86 (65%) children had a CCC diagnosis. Of the patients without a CCC, 17 of 47 had primary diagnoses that were not classified as CCCs. Obesity was the most common diagnosis (n = 7, 41%), followed by airway disease, including asthma and bronchiectasis (n = 3, 17%). The other diagnoses included (n = 1, 6%, in each of the following): cataracts, autism, Goldenhar syndrome, thrombocytopenia, hemolytic uremic syndrome, failure to thrive, and congenital ear malformation.

Of the patients with a CCC diagnosis, 49 patients had a single-system CCC and 37 children had a CCC involving 2 or more systems. The CCC primary diagnoses for the study cohort are summarized in Table 2. In the study cohort, the CCC diagnoses were distributed across 8 CCC system categories. The 3 most common categories for CCC diagnosis were (1) other congenital or genetic defect (n = 44, 51%), (2) neurologic and neuromuscular (n = 11, 13%), and (3) hematologic or immunologic (n = 11, 13%). In the other congenital or genetic defect category, there were 20 children with a diagnosis of trisomy 21.

Preoperative Polysomnography

All patients had a PSG prior to AT. A total of 114 of 133 (85.7%) children in the cohort had severe OSA pre-AT. The results of the pre-AT PSGs are summarized in Table 1. The only significantly different PSG variable between children without a CCC, children with a single-system CCC, and children with a multisystem CCC was a significantly lower minimum oxygen saturation (P = .003) in the multisystem CCC group (Table 1).

Prevalence of Persistent OSA Post-AT

All children in the study cohort underwent a repeat baseline PSG after AT. The median (range) time that the PSG was performed following AT was 215.5 (50-633) days. As expected following AT, there were significant improvements in OAHI (median [range] from 19.2 [5.0-146.0] to 6.7 [0-78.0], P < .0001), SaO₂ nadir (mean [SD] from 77.1 [13.2] to 85.1 [11.5], P < .0001), arousal index (mean [SD] from 20.4 [14.6] to 13.3 [10.4], P < .0001), and sleep efficiency (mean [SD] from 83.2 [12.1] to 86.4 [11.0], P = .004). PSG results for the subgroups post-AT are summarized in Table 1. Overall, 121 children had improvement of OAHI number, whereas 12 patients experienced a lack of
improvement or deterioration in their OAHI post-AT. In the post-AT PSG, the multisystem CCC group had significantly lower minimum oxygen saturation ($P = .0002$) and higher OAHI ($P = .003$) than the other groups (Table 1). In all 3 CCC groups, the median OAHI improved post-AT: from 18.7 to 5.4 in children with multisystem CCC, from 18.8 to 1.5 in children with single-system CCC, and from 23.8 to 1.2 in children without CCC.

In the overall study cohort, the repeat PSG post-AT showed persistence of OSA (OAHI ≥ 5 events per hour of sleep) in 42 (32%) children (see Table 3). The rate of persistent OSA following AT was highest in children with multisystem CCC (38%, $n = 14/37$), intermediate in children with single-system CCC (16%, $n = 8/49$), and lowest in children without CCC (8.5%, $n = 4/47$), $P = .003$. Looking specifically at the CCC disease category, of the 44 children with an other congenital of genetic defect, 30 (68%) failed AT. Furthermore, of the 20 children with trisomy 21 in the cohort, 10 (50%) failed AT.

### Predictors of Persistent OSA Post-AT

The results of the univariate analysis for persistent OSA post-AT are presented in Table 4. Presence of a multisystem CCC ($P = .003$) and oxygen saturation (SaO$_2$) nadir at baseline ($P = .007$) were significant predictors of AT failure. The odds of having persistent OSA post-AT were 7.50

**Table 1.** Complex Chronic Condition Primary Diagnoses for the Study Cohort.

<table>
<thead>
<tr>
<th>Categories (n = 86)</th>
<th>Subcategories</th>
</tr>
</thead>
</table>
| Other congenital or genetic defect, n = 44 (51%) | Chromosomal anomalies (n = 23)  
- Trisomy 21 (n = 20)  
- Wolf-Hirschhorn syndrome (n = 2)  
- Pallister-Killian syndrome (n = 1)  
Bone and joint anomalies (n = 16)  
- Achondroplasia (n = 9)  
- Craniosynostosis (n = 5)  
- Spondyloepiphyseal dysplasia (n = 1)  
- Osteopetrosis (n = 1)  
Other congenital anomalies (n = 4)  
- Prader-Willi syndrome (n = 3)  
- Other congenital malformation syndromes, not elsewhere classified (n = 1)  
Diaphragm and abdominal wall (n = 1) |
| Neurologic and neuromuscular, n = 11 (13%) | Infante cerebral palsy (n = 1)  
Epilepsy (n = 1)  
Central nervous system degeneration and diseases (n = 2)  
Brain and spinal cord malformations (n = 7)  
- Chiari malformation (n = 4)  
- Congenital hydrocephalus (n = 2)  
- Lissencephaly (n = 1) |
| Hematologic or immunologic, n = 11 (13%) | Hereditary anemias (sickle cell disease) (n = 11)  
Cardiomyopathies (n = 1)  
Heart and great vessel malformations (n = 7)  
- Tetralogy of Fallot (n = 2)  
- Congenital pulmonary valve stenosis (n = 1)  
- Complete heart block (n = 1)  
- Scimitar syndrome (n = 1)  
- Congenital aortic insufficiency (n = 1)  
- Atrioventricular septal defect (n = 1) |
| Cardiovascular, n = 8 (9.3%) | Extreme prematurity (n = 4)  
Respiratory diseases (n = 3)  
Hypoxic-ischemic encephalopathy (n = 1) |
| Premature and neonatal, n = 8 (9.3%) | Neoplasms (n = 2)  
Chronic renal failure (n = 1) |
| Malignancy, n = 2 (2.3%) | Endocrine disorders (n = 1) |
| Renal and urologic, n = 1 (1.2%) | |
Results of the multivariable regression analysis are presented in Table 5. After controlling for age, sex, BMI, mean and nadir SaO\textsubscript{2} at baseline, arousal index, highest CO\textsubscript{2} at baseline, baseline OAHI, and time from AT to follow-up PSG, the presence of multisystem CCC was the only significant predictor of failed AT \((P = .01)\). The odds of having persistent OSA post-AT were 7.42 (95% CI, 2.16-25.51) times higher if the child had a multisystem CCC vs no CCC. Similarly, the odds of having persistent OSA post-AT remained significantly higher for children with multisystem CCC compared to single-organ CCC (OR, 3.35; 95% CI, 1.16-9.64).

### Table 3. Demographic, Anthropometric, and Clinical Variable Cohort Comparisons between Those with Persistent OSA vs Resolved OSA Post-AT.

<table>
<thead>
<tr>
<th>Results</th>
<th>Successful AT Groups ((n = 91))</th>
<th>Failed AT Group ((n = 42))</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>5.6 (3.5)</td>
<td>5.4 (4.3)</td>
<td>.85</td>
</tr>
<tr>
<td>Male/female, No.</td>
<td>56/35</td>
<td>28/14</td>
<td>.57</td>
</tr>
<tr>
<td>Weight, mean (SD), kg</td>
<td>25.9 (18.8)</td>
<td>27.4 (22.9)</td>
<td>.69</td>
</tr>
<tr>
<td>Height, mean (SD), cm</td>
<td>110.2 (24.9)</td>
<td>102.9 (30.7)</td>
<td>.15</td>
</tr>
<tr>
<td>BMI, mean (SD), kg/m(^2)</td>
<td>19.5 (6.6)</td>
<td>20.5 (7.0)</td>
<td>.42</td>
</tr>
<tr>
<td>No. of healthy children with non-CCC diagnosis</td>
<td>40</td>
<td>7</td>
<td>.0004</td>
</tr>
<tr>
<td>CCC involving 1 system category, No.</td>
<td>35</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>CCC involving (\geq) 2 system categories, No.</td>
<td>16</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>OAHI at baseline pre-AT, median (range)</td>
<td>17.2 (5.97-6)</td>
<td>25.7 (5.6-146)</td>
<td>.06</td>
</tr>
</tbody>
</table>

Abbreviations: AT, adenotonsillectomy; BMI, body mass index; CCC, complex chronic condition; OAHI, obstructive apnea-hypopnea index; 

\(^a\)Independent \(t\) test for continuous variables and \(\chi^2\) test for categorical variables. For OAHI, Wilcoxon rank-sum test was used.

### Table 4. Univariate Analysis Results for Persistent OSA Post-AT.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Odds Ratio (95% CI)</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.0 (0.92-1.11)</td>
<td>.85</td>
</tr>
<tr>
<td>Male vs female</td>
<td>0.80 (0.37-1.72)</td>
<td>.57</td>
</tr>
<tr>
<td>BMI</td>
<td>0.97 (0.92-1.03)</td>
<td>.42</td>
</tr>
<tr>
<td>OAHI at baseline</td>
<td>0.99 (0.98-1.01)</td>
<td>.18</td>
</tr>
<tr>
<td>Mean SaO\textsubscript{2} at baseline</td>
<td>1.05 (0.94-1.17)</td>
<td>.39</td>
</tr>
<tr>
<td>SaO\textsubscript{2} nadir at baseline</td>
<td>1.04 (1.01-1.07)</td>
<td>.007</td>
</tr>
<tr>
<td>Arousals index at baseline</td>
<td>0.99 (0.97-1.02)</td>
<td>.88</td>
</tr>
<tr>
<td>Highest CO\textsubscript{2} at baseline</td>
<td>0.97 (0.94-1.004)</td>
<td>.09</td>
</tr>
<tr>
<td>Time from AT to follow-up PSG</td>
<td>1.0 (0.99-1.01)</td>
<td>.23</td>
</tr>
<tr>
<td>Primary diagnosis (\geq) 2 CCCs vs non-CCC</td>
<td>7.50 (2.67-21.07)</td>
<td>.003</td>
</tr>
<tr>
<td>(\geq) 2 CCCs vs 1 CCC</td>
<td>3.28 (1.34-8.06)</td>
<td>.23</td>
</tr>
</tbody>
</table>

Abbreviations: AT, adenotonsillectomy; BMI, body mass index; CCC, complex chronic condition; CO\textsubscript{2}, carbon dioxide; OAHI, obstructive apnea-hypopnea index; OSA, obstructive sleep apnea; PSG, polysomnography; REM, rapid eye movement; SaO\textsubscript{2}, oxygen saturation; TST, total sleep time.

### Table 5. Multivariable Regression Analysis Results for Persistent OSA Post-AT.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Odds Ratio (95% CI)</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.96 (0.84-1.11)</td>
<td>.60</td>
</tr>
<tr>
<td>Male vs female</td>
<td>0.60 (0.23-1.56)</td>
<td>.29</td>
</tr>
<tr>
<td>BMI</td>
<td>0.99 (0.93-1.06)</td>
<td>.83</td>
</tr>
<tr>
<td>OAHI at baseline</td>
<td>0.99 (0.95-1.02)</td>
<td>.40</td>
</tr>
<tr>
<td>Mean SaO\textsubscript{2} at baseline</td>
<td>0.85 (0.65-1.12)</td>
<td>.25</td>
</tr>
<tr>
<td>SaO\textsubscript{2} nadir at baseline</td>
<td>1.04 (0.99-1.08)</td>
<td>.14</td>
</tr>
<tr>
<td>Arousals index at baseline</td>
<td>1.01 (0.97-1.06)</td>
<td>.60</td>
</tr>
<tr>
<td>Highest CO\textsubscript{2} at baseline</td>
<td>0.98 (0.95-1.02)</td>
<td>.39</td>
</tr>
<tr>
<td>Time from AT to follow-up PSG</td>
<td>1.0 (0.99-1.01)</td>
<td>.35</td>
</tr>
<tr>
<td>Primary diagnosis (\geq) 2 CCCs vs non-CCC</td>
<td>7.42 (2.16-25.51)</td>
<td>.01</td>
</tr>
<tr>
<td>(\geq) 2 CCCs vs 1 CCC</td>
<td>3.35 (1.16-9.64)</td>
<td>.23</td>
</tr>
</tbody>
</table>

Abbreviations: AT, adenotonsillectomy; BMI, body mass index; CCC, complex chronic condition; CO\textsubscript{2}, carbon dioxide; OAHI, obstructive apnea-hypopnea index; OSA, obstructive sleep apnea; PSG, polysomnography; REM, rapid eye movement; SaO\textsubscript{2}, oxygen saturation; TST, total sleep time.

(95% confidence interval [CI], 2.67-21.07) times higher in children with multisystem CCC vs no CCC. Similarly, the odds of having persistent OSA post-AT were also significantly higher for children with multisystem CCC compared to single-organ CCC (odds ratio [OR], 3.28; 95% CI, 1.34-8.06).

Results of the multivariable regression analysis are presented in Table 5. After controlling for age, sex, BMI, mean and nadir SaO\textsubscript{2} at baseline, arousal index, highest CO\textsubscript{2} at baseline, baseline OAHI, and time from AT to follow-up PSG, the presence of multisystem CCC was the only significant predictor of failed AT \((P = .01)\). The odds of having persistent OSA post-AT were 7.42 (95% CI, 2.16-25.51) times higher if the child had a multisystem CCC vs no CCC. Similarly, the odds of having persistent OSA post-AT remained significantly higher for children with multisystem CCC compared to single-organ CCC (OR, 3.35; 95% CI, 1.16-9.64).

### Discussion

To our knowledge, this is the first study to evaluate outcomes following AT in children with CCC. We demonstrated that having a CCC diagnosis was a strong predictor of persistent OSA post-AT. According to this study, 15% of healthy children, 29% of children with single-system CCC, and 57% of children with multisystem CCC had persistent OSA post-AT. Whereas our AT results in healthy children are consistent with previous reports,\(^{17}\) the persistence of OSA in children with CCC is disappointing.

It is now well recognized that untreated OSA in children is associated with poor weight gain, poor daytime and school function, neurocognitive dysfunction, and cardiovascular consequences.\(^{26-31}\) Untreated OSA in children also has been found to be associated with decreased health-related quality of life\(^{32}\) as well as an increase in health care resource utilization.\(^{33,34}\)
Therefore, current clinical practice guidelines from the American Academy of Pediatrics (AAP) recommend AT as first-line therapy in healthy children older than 2 years for the treatment of OSA, but these guidelines excluded children with underlying conditions such as trisomy 21 and craniofacial anomalies for whom a higher prevalence of OSA and failed AT has been reported. However, these reports were limited in numbers, and they were specific to the underlying primary disease. On other hand, the AT success rate is not known in the groups that have diseases with more than 1 system involvement (ie, with complex medical conditions), which all share similar functional limitations, increased health resource use, and increased morbidities and mortality risks.

Children with CCCs have significant comorbidities that are risk factors for AT failure, but the relationship between CCC and AT failure has never been formally evaluated. The high rate of persistent OSA in children with multisystem CCC in our study questions whether AT should be the first line of therapy in this cohort of children. Our cohort included a diverse population of children representing 8 different CCC categories. The most common diagnosis of CCC was congenital/genetic defects (44%).

Due to the small sample size, the present study was not powered to investigate each individual CCC category as a predictor of persistent OSA. However, this would be an important area of future study. Although the wide confidence intervals reflect the relatively small sample size, the presence of a CCC diagnosis as a strong predictor of persistent OSA indicates the clinical relevance and importance of CCCs in surgical decisions. The management of OSA in children diagnosed with CCC is complex and requires careful balancing of risks and benefits. It is important to highlight other limitations to the study. We only included children with an OSA diagnosis who underwent AT, and therefore we did not evaluate the prevalence of OSA in CCC in comparison to non-CCC patients who were referred for evaluation. Furthermore, the patients included were children who had PSGs both pre- and post-AT, which might add a potential selection bias as children with complex medical conditions are more likely to undergo pre- and postoperative PSG than are healthy children. In addition, the wide time range for postoperative PSG might have influenced the results of the PSGs and the OSA severity classifications. Several groups with a single diagnosis were identified to have a higher risk for OSA as mentioned, but OSA risk is not known in the CCC group despite the logical assumption of the increased risk. Furthermore, we did not look at the outcome of those children with moderate to severe OSA who did not undergo AT or received other medical/surgical managements. It is important to have future reports investigating AT alternatives in CCC children. Finally, it will be interesting to know the AT-related complications in CCC vs non-CCC patients and the predictors for that. We were unable to evaluate this given the fact that a significant proportion of children had AT outside our institution.

Conclusions

Although AT is considered first-line therapy in healthy children older than 2 years for the treatment of OSA, there is a significantly greater risk of persistent OSA post-AT in children with CCCs compared to children without CCCs. Therefore, other surgical procedures or nonsurgical management may need to be considered first-line treatment for this cohort of medically complex children.

Author Contributions

Reshma Amin, designing the study, assisting data collection, interpreting the data, writing the manuscript and approving the final version of the manuscript; Theresa Holler, reviewing the data and results, revising the manuscript critically and approving the final version of the manuscript; Indra Narang, assisting in the design of the study, interpreting the data, reviewing the data, critically revising the manuscript and approving the final version of the manuscript; Sharon L. Cushing, reviewing the results, revising the manuscript critically and approving the final version of the manuscript; Suhail Al-Saleh, designing the study, detailed review of the data and statistical results, writing the manuscript and approving the final version of the manuscript.

Disclosures

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