**Predictors of OSA following Adenotonsillectomy in Children with Down Syndrome**

**Abstract**

**Objectives:** Given that 30-50% of children with Down syndrome have persistent obstructive sleep apnea (OSA) after adenotonsillectomy, we evaluated whether demographic, clinical and polysomnographic factors predicted persistent OSA and OSA severity after adenotonsillectomy.

**Design:** Retrospective study.

**Setting:** Secondary care hospital.

**Participants:** Retrospective review of 32 children with the diagnosis of DS and OSA by polysomnography type 1 who underwent adenotonsillectomy, from January 2010 to December 2018.

**Main outcome and measure:** Non-parametric analysis was used to compare pre and postoperative factors, regression was used to model persistent OSA and OSA severity.

**Results:** Thirty-two children were included (17 male, median age 10.00 ± 8.00 years, median body mass index z-score 0.89 ± 1.25). Overall, adenotonsillectomy resulted in a significant improvement in median obstructive apnea-hypopnea index (oAHI) from 7.5 ± 8.95 to 4.40 ± 4.38 events per hour (p<0.001) and in median OSA-18 score from 85.00 ± 12.00 to 61 ± 37.75 (p<0.001). Persistent OSA was found in 56.25% of the children. Univariate regression suggests that postoperative OSA-18 score predicted persistent OSA after adenotonsillectomy. Preoperative oAHI, preoperative oxygen desaturation rate, pre and postoperative OSA-18 scores correlated with OSA severity after adenotonsillectomy. However, in a multivariate model only the postoperative OSA-18 score was able to predict OSA severity after adenotonsillectomy.

**Conclusions:** Although adenotonsillectomy results in a significant improvement of OSA in children with Down syndrome, more than half of the children had persistent OSA. The postoperative OSA-18 score predicted both persistent OSA and OSA severity after adenotonsillectomy.

**Key Words:** Obstructive sleep apnea, Pediatric, Adenotonsillectomy, Down syndrome, Persistent, OSA Severity

**Key Points:**

**-** Adenotonsillectomy results in a significant improvement of OSA in children with Down syndrome.

- More than half of children with Down syndrome have persistent OSA after adenotonsillectomy.

- Preoperative polysomnographic data failed to predict persistent OSA and OSA severity after adenotonsillectomy.

- Demographic and clinical factors failed to predict persistent OSA and OSA severity after adenotonsillectomy.

- Postoperative OSA-18 score predicted both persistent OSA and OSA severity after adenotonsillectomy.

**Introduction**

Obstructive Sleep Apnea (OSA) is a sleep-breathing disorder affecting approximately 1-4% of the general pediatric population. The prevalence of OSA in children with Down Syndrome (DS) is significantly higher with reported prevalences of 30-70%1. This increased prevalence is a consequence not only, as in the general pediatric population of enlarged adenoids and tonsils but also of multiple anatomical and functional factors including maxillary and mandibular hypoplasia, narrow nasopharynx, small oropharynx, relative macroglossia, obesity, gastroesophageal reflux and generalized hypotonia1-2. OSA, in this particular population has been associated with significant morbidity leading to cardiovascular problems, impaired cognition, behavior problems and lower quality of life (QOL)3-5. Adenotonsillectomy (T&A) is considered the first-line surgical therapy for OSA6. Nevertheless, in children with DS, T&A alone is often not sufficient since persistent OSA was shown to be present in almost half of the children7. Given the emergence of new tools that enable a more tailored treatment of children with OSA, namely Drug Induced Sleep Endoscopy (DISE) and DISE-oriented procedures it becomes relevant to try to identify the children whose response to the current therapy is unsatisfactory. In this study we sought to evaluate whether demographic, clinical and polysomnographic factors could predict persistent OSA and OSA severity after T&A, thus identifying the subset of children who might benefit from further treatment.

**2. Materials and Methods**

**2.1 Patients**

Approval from [removed for blind peer review] Institutional Review Board was obtained. A retrospective chart review was performed which identified all children with the diagnosis of DS and OSA who had adenotonsillectomy performed from January 2010 to December 2018. A full clinical examination was performed in every child, body mass index (BMI) z-score calculated (a BMI z-score greater than 1.65 was considered as fulfilling obese criteria) and tonsil size scored according to Brodsky score.

**2.2 Overnight polysomnography**

All children had a full overnight type 1 polysomnography (PSG) in the sleep laboratory before and after T&A. Children with a postoperative PSG more than 6 months apart from surgery were excluded. The preoperative and postoperative PSG metrics were collected using Philips Alice 5 G3 Respironics. Signals included electroencephalogram (6 channels); eletrooculogram (2 channels), submental electromyogram, tibial electromyogram, electrocardiogram. Airflow was measured with a nasal cannula and thermistor. Respiratory effort was assessed with thoracic and abdominal inductive plethysmography. Oxygen saturation was measured by a finger probe connected to a pulse oximeter and end-tidal CO2 was obtained in all patients. Snoring was detected by a microphone at the suprasternal notch and body position was monitored with a sensor position and an infrared video camera. Polysomnograms were scored and interpreted by a European Sleep Research Society certified physician according to the American Academy of Sleep Medicine (AASM) guidelines for children8. A diagnosis of OSA was established with an obstructive apnea-hypopnea index (oAHI) ≥ 1 event per hour. OSA severity was defined by oAHI. Mild OSA was defined as an oAHI ≥ 1 and < 5 events per hour, moderate OSA as an oAHI ≥ 5 and < 10 events per hour and severe OSA as an oAHI ≥ 10 events per hour. Persistent OSA after T&A was defined in patients with moderate to severe OSA as a postoperative oAHI ≥ 5 events per hour, and in patients with mild OSA as a postoperative oAHI ≥ 1 event per hour9. The saturation nadir was defined as the lowest oxygen saturation during a respiratory event.

**2.3 OSA-18 Questionnaire**

All caregivers completed the OSA-18 questionnaire validated for the Portuguese population10 before T&A and 3 months after. This instrument assesses sleep-breathing disorder related quality of life (QOL) by caregivers in five domains: sleep disturbance, physical symptoms, emotional symptoms, daytime function and caregiver concerns; each item is given a score from 1-7 in which 1 means “never” and 7 means “always”. Scores < 60 suggest a mild impact on health related QOL, scores between 60 and 80 a moderate impact and scores >80 a large impact11.

**2.4 Data analysis**

Statistical analysis was performed with IBM SPSS statistics version 24. Data was presented as median values and interquartile range. Pre and postoperative variables were compared by Wilcoxon Signed rank test and Mann-Whitney U test; correlations were calculated using Spearman’s Correlation coefficient. Univariate logistic regression was used to model persistent OSA. Multiple linear regression was used to model postoperative AHI. Statistical significance was accepted at *p*<0.05.

**3. Results**

**3.1 Pre and postoperative data**

This study included 32 children, 17 male (53.1%) with the diagnosis of both DS and OSA who underwent T&A. The median age at surgery was 10.00 ± 8.00 years and the median BMI z-score 0.89 ± 1.25. Overall, 50% were obese (n=16) and 59.4% had tonsillar hypertrophy (n=19) defined by a Brodsky score >2. The median oAHI improved from 7.50 ± 8.95 events per hour to 4.40 ± 4.38 after T&A (p<0.001). Also, the median saturation nadir, the mean asleep oxygen saturation, the respiratory distress index (RDI) and the oxygen desaturation index (ODI) significantly improved after T&A (Table 1). OSA severity before T&A was as follows, mild= 6/32 (18.8%); moderate= 13/32 (40.6%); severe= 13/32 (40.6%). After surgery, the percentage of children with moderate (56.3%) and severe OSA (15.6%) significantly decreased (p<0.001), whereas the percentage of children with mild OSA (56.3%) naturally increased (p<0.001; Figure 1). Persistent OSA after T&A was present in 56.25% of children, and if the AASM criteria are applied none of the children achieved cure (oAHI < 1 event per hour). On the QOL questionnaire the median OSA-18 score was 85.00 ± 12.00 before T&A and 61.00 ± 37.75 after T&A (p<0.001; Table 1).

**3.2 Factors predicting persistent OSA and OSA severity after T&A**

Regression suggests that the postoperative OSA-18 score predicted persistent OSA after T&A on univariate logistic regression (p=0.022, Table 2). No other studied factor was shown to predict persistent OSA after T&A. A significant correlation was found between OSA severity after T&A and preoperative oAHI (p=0.003), preoperative ODI (p=0.016), preoperative OSA-18 score (p=0.001) and postoperative OSA-18 score (p<0.001; Table 3). Multiple linear regression controlling for age, sex, tonsillar hypertrophy, BMI z-score, preoperative oAHI, preoperative saturation nadir, preoperative mean asleep oxygen saturation, preoperative RDI, preoperative ODI and pre and postoperative OSA-18 scores revealed that only the postoperative OSA-18 score predicted OSA severity after T&A (p=0.029, R2 = 0.705; Table 4).

**4. Discussion**

These results indicate that T&A significantly improved OSA severity in children with DS, however more than half of the children still showed persistent OSA after surgery. This data is in accordance with previous reports. Maris *et al.* 7 found a significant improvement of OSA severity but also that 47,1% of children had persistent OSA after surgical treatment. In their study only 22 patients out of 34 underwent T&A, whereas the remaining underwent isolated tonsillectomy or adenoidectomy. Moreover, the definition of persistent OSA differs from our study since it is defined as a postoperative oAHI ≥ 5 events per hour, irrespective of the preoperative oAHI. This poses an inherent limitation given that patients with mild OSA (oAHI < 5 events per hour) could never be considered as having persistent OSA after surgery. The definition of cure also differs between studies; Maris *et al.*7considers a normal oAHI as having less than 2 events per hour. Taking into consideration this cut-off, in our study, instead of having none of the patients achieving cure we could verify that 15.6% (5 patients) could be considered as cured, similarly to the 17.6% reported by Maris *et al*.7 The same cut-off was also used by Shete *et al.*12andNerfeldt and Sundelin13who reported cure rates of 18% and 15.2%, respectively. Ingram *et al.* 14 reported the cure rate of T&A using both criteria and found that 12% of children had an oAHI < 1 event per hour and 21% had an oAHI < 2 events per hour. In this study 52% of children also had an oAHI > 5 events per hour, the closest approximation to our definition of persistent OSA. Another recent study by Best *el al.*15 reported a cure rate of 10.8%, using the most stringent criteria of oAHI < 1 event per hour. The differences found between our study and the studies by Ingram *et al.* 14 and Best *at al.*15 could be explained by the different sample sizes. In opposition to all of the above-mentioned studies and ours, Abdel-Aziz *et al.*16 reported a normalization of oAHI (oAHI < 1 event per hour) in 72% of children after T&A. Nevertheless, the majority of children in this study had mild OSA, which constitutes a major difference. In addition, also overweight and obese children were excluded and the BMI z-score cut-off used to define those categories not mentioned.

Regarding the results of the OSA-18 questionnaire it became obvious that OSA has a large impact in the QOL of children with DS. T&A significantly improved these children’s QOL, however it is important to notice that even after T&A OSA has a moderate impact in the QOL. Nerfeldt and Sundelin13 and Sudarsan *et al.*17 reported similar results, nevertheless in smaller and more heterogeneous samples, respectively. Moreover, Bergeron *et al.*5alsoshowed that persistent OSA after T&A poses a moderate impact in the QOL of children with DS, regardless of OSA severity.

The postoperative OSA-18 score was the only studied factor shown to predict both persistent OSA and OSA severity after T&A, the latter of which in a multivariate model. This constitutes an interesting finding since OSA-18 after T&A can be a tool of great value to predict the need for future treatments such as continuous positive airway pressure (CPAP) or DISE-oriented procedures. In addition, it can also be useful for centers in which polysomnography is not widely available. In a bivariate analysis, preoperative oAHI, preoperative ODI and preoperative OSA-18 score correlated with OSA severity after T&A, however this did not have independent predictive power in the presence of other factors. Concerning age, gender, tonsil hypertrophy, BMI z-score and other polysomnographic variables, these were not associated with either persistent OSA or OSA severity after T&A. A systematic review by Farhood *et al.*18 *and* Maris *et al.*7support these findings by concluding that no associations were found between preoperative oAHI, BMI or age with persistent OSA or OSA severity after T&A. On the other hand, Ingram *et al.*14reported that the only factor to be associated with OSA severity after T&A was the preoperative mean asleep oxygen saturation, a fact we could not prove in our sample. Several studies in healthy children also report that BMI negatively influences T&A outcomes19-20, nevertheless no study was capable of showing that in children with DS7,14,21-22, pointing out that factors such as hypotonia and craniofacial anatomy are probably more relevant in these children. Prospective studies lack and are necessary to better understand these findings in children with DS and OSA.

Our study has some limitations inherent to the retrospective design, which may bias clinical data. Furthermore, the current definition of cure and persistent OSA after T&A lack standardization. A more uniform data report would enable investigators to improve comparability and research outcomes.

In conclusion, this study demonstrated that T&A significantly improves OSA severity in children with DS, however more than half of the children still have persistent OSA. The preoperative oAHI, preoperative ODI and preoperative OSA-18 score correlate with OSA severity after T&A; nevertheless the postoperative OSA-18 score was the only factor to be able to predict both persistent OSA and OSA severity after T&A.

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| --- | --- | --- | --- |
|  | Preoperative | Postoperative | *p* value |
| Age median – years | 10.00 ± 8.00 | 11.00 ± 9.00 | 0.002\* |
| Gender (male) – n (%) | 17 (53.1%) | - | - |
| BMI z-score median | 0.89 ± 1.25 | 0.90 ± 1.07 | 0.807 |
| Tonsils (Brodsky 2 and 3) – n (%) | 19 (59.4%)3 | - | - |
| oAHI median – events/hour | 7.50 ± 8.95 | 4.40 ± 4.38 | <0.001\*\* |
| Saturation nadir median – % | 90.00 ± 3.00 | 90.50 ± 2.00 | 0.002\* |
| Mean Asleep oxygen saturation median – % | 96.00 ± 4.00 | 97.00 ± 1.00 | 0.033\* |
| ODI median – events/hour | 9.05 ± 9.55 | 4.90 ± 5.30 | <0.001\*\* |
| RDI median – events/hour | 8.00 ± 9.93 | 4.10 ± 4.70 | <0.001\*\* |
| OSA-18 score median | 85.00 ± 12 | 61.00 ± 37.75 | <0.001\*\* |
| Persistent OSA after adenotonsillectomy – n (%) |  | 18 (56.25%) |  |

Table 1. Pre and postoperative demographic, clinical and polysomnographic data.

Differences between groups for age, BMI z-score, preoperative oAHI, preoperative saturation nadir, preoperative mean asleep oxygen saturation, preoperative ODI, preoperative RDI, preoperative OSA-18 score, postoperative OSA-18 score were calculated using Wilcoxon signed-rank test.

Abbreviations: oAHI, obstructive apnea-hypopnea index; BMI, body mass index; ODI, oxygen desaturation index; RDI, respiratory distress index; OSA, obstructive sleep apnea.

\*p<0.05, \*\*p<0.001

Figure 1. Changes in OSA severity after adenotonsillectomy.

Abbreviations: OSA, obstructive sleep apnea.

\*\*p<0.001

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| --- | --- | --- |
|  | Persistent OSA | |
|  | Β (95% CI) | *p* value |
| Age median – years | 0.163 (0.987 to 1.404) | 0.069 |
| Gender (male) – n (%) | 0.606 (0.115 to 1.976) | 0.232 |
| Tonsils (Brodsky 2 and 3) – n (%) | -1.243 (0.066 to 1.265) | 0.099 |
| BMI z-score median | -0.009 (0.526 to 1.866) | 0.977 |
| Preoperative oAHI median – events/hour | -0.109 (0.961 to 1.296) | 0.151 |
| Preoperative saturation nadir median – % | 0.024 (0.828 to 1.268) | 0.822 |
| Preoperative mean Asleep oxygen saturation median – % | -0.092 (0.684 to 1.215) | 0.528 |
| Preoperative ODI median – events/hour | 0.051 (0.950 to 1.165) | 0.326 |
| Preoperative RDI median – events/hour | 0.039 (0.930 to 1.163) | 0.494 |
| Preoperative OSA-18 score median | 0.024 (0.961 to 1.092) | 0.455 |
| Postoperative OSA-18 score median | 0.053 (1.008 to 1.104) | 0.022\* |

Table 2. Simple logistic regression analysis: demographic, clinical and polysomnographic factors associated with persistent OSA.

Abbreviations: oAHI, obstructive apnea-hypopnea index; BMI, body mass index; ODI, oxygen desaturation index; RDI, respiratory distress index; OSA, obstructive sleep apnea.

\*p<0.05

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| --- | --- |
|  | Postoperative oAHI |
| Age median – years | *p*= 0.106 / r = 0.291 |
| Gender (male) – n (%) | *p*= 0.815 |
| Tonsils (Brodsky 2 and 3) – n (%) | *p* = 0.803 |
| BMI z-score median | *p*= 0.621 / r = 0.091 |
| Preoperative oAHI median – events/hour | *p*= 0.003\* / r = 0.512 |
| Preoperative saturation nadir median – % | *p*= 0.878 / r = -0.028 |
| Preoperative mean asleep oxygen saturation median – % | *p*= 0.447 / r = -0.139 |
| Preoperative ODI median – events/hour | *p*= 0.016\* / r = 0.424 |
| Preoperative RDI median – events/hour | *p* = 0.050 / r= 0.374 |
| Preoperative OSA-18 score median | *p* = 0.001\* / r= 0.562 |
| Postoperative OSA-18 score median | *p*< 0.001\*\* / r= 0.682 |

Table 3. Correlation and association between postoperative oAHI and demographic, clinical and polysomnographic data.

Correlation between postoperative oAHI and the variables age, BMI z-score, preoperative oAHI, preoperative saturation nadir, preoperative mean asleep oxygen saturation, preoperative ODI, preoperative RDI, preoperative OSA-18 score, postoperative OSA-18 score was calculated using Spearman’s rank correlation coefficient. Association between postoperative oAHI and the variables gender and tonsils were calculated using Mann-Whitney U test.

Abbreviations: oAHI, obstructive apnea-hypopnea index; BMI, body mass index; ODI, oxygen desaturation index; RDI, respiratory distress index; OSA, obstructive sleep apnea.

\*p<0.05, \*\*p<0.001

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| --- | --- | --- |
|  | Postoperative oAHI | |
|  | Β (95% CI) | *p* value |
| Age median – years | -0.008 (-0.357 to 0.340) | 0.960 |
| Gender (male) – n (%) | 0.388 (-2.018 to 2.794) | 0.737 |
| Tonsils (Brodsky 2 and 3) – n (%) | 1.184 (-1.249 to 3.618) | 0.318 |
| BMI z-score median | -0.489 (-1.509 to 0.530) | 0.324 |
| Preoperative oAHI median – events/hour | 0.227 (-0.492 to 0.947) | 0.513 |
| Preoperative saturation nadir median – % | 0.263 (-0.326 to 0.851) | 0.358 |
| Preoperative mean Asleep oxygen saturation median – % | -0.280 (-0.798 to 0.237) | 0.268 |
| Preoperative ODI median – events/hour | 0.118 (-0.474 to 0.710) | 0.678 |
| Preoperative RDI median – events/hour | -0,150 (-0.954 to 0.654) | 0.654 |
| Preoperative OSA-18 score median | 0.006 (-0.135 to 0.147) | 0.926 |
| Postperative OSA-18 score median | 0.114 (0.013 to 0.215) | 0.029\* |

Table 4. Multiple linear regression analysis: demographic, clinical and polysomnographic factors associated with postoperative oAHI.a

Abbreviations: oAHI, obstructive apnea-hypopnea index; BMI, body mass index; ODI, oxygen desaturation index; RDI, respiratory distress index; OSA, obstructive sleep apnea.

a R2 = 0.705

\*p<0.05