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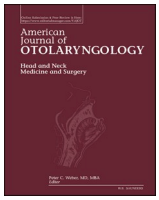

Adenoid regrowth and obesity in a longitudinal pediatric cohort

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ABSTRACT

Objective: This study aims to elucidate the association between pediatric obesity and revision adenoidectomy in a cohort with longitudinal care needs.
Methods: Data were collected via chart review from a tertiary pediatric hospital for patients who underwent adenoidectomy from January 2015 to January 2016 and had 2 years of post-operative follow-up. Demographic, surgical, and clinical data were analyzed using logistic and Cox regression models to identify factors affecting the likelihood and timing of revision adenoidectomy.
Results: Of 461 patients, 115 (24.9 %) were obese at primary intervention. Secondary intervention was performed for 136 patients (29.5 %), with a median interval of 29 months between procedures. In the logistic regression, predictors of revision included younger age at primary intervention (OR = 0.844, $p = 0.002$), adenoidectomy over adenotonsillectomy as the initial surgery (OR = 0.3, $p < 0.001$), higher BMI percentile (OR = 1.008, $p = 0.048$), and allergic rhinitis (OR = 1.722, $p = 0.039$). In the Cox regression, hazard was lower with adenotonsillectomy (HR = 0.358, $p < 0.001$), older age at initial surgery (HR = 0.858, $p = 0.001$), and GERD (HR = 0.595, $p = 0.05$), but higher with laryngomalacia/tracheomalacia (HR = 1.909, $p = 0.034$). BMI percentile was not associated with revision timing in the Cox model. Model concordance was 0.694.
Conclusion: Odds of revision adenoidectomy in this population are increased with higher BMI percentile, younger age at primary intervention, undergoing initial adenoidectomy rather than adenotonsillectomy, and various comorbidities, with differing time-dependent effects. These findings support the potential role of obesity-related inflammation in adenoid hypertrophy and individualized surgical decision-making in pediatric patients with sleep disordered breathing.

1. Introduction

Childhood obesity is one of the most well-studied comorbidities of pediatric obstructive sleep apnea (OSA), which causes a range of sleep, behavioral, and mood symptoms [1–3]. According to the Centers for Disease Control (CDC), the prevalence of childhood obesity in the United States is 12.7 % for 2–5 year olds, 20.7 % for 6–11 year olds, and 22.2 % for adolescents [4]. The prevalence of OSA is estimated to be 1–6 % in the general pediatric population but as high as 19–61 % among patients with obesity [5]. Upper airway obstruction in pediatric OSA is commonly attributed to adenoid and tonsillar hypertrophy (ATH) [2]. Obesity increases the risk of ATH by four to five times [6], potentially due to increased levels of inflammatory markers in obese patients, as anti-inflammatory agents can help mitigate lymphoid tissue growth [7–9].

First-line surgical treatment for pediatric OSA is adenotonsillectomy (AT) [10], but residual OSA remains a challenge. Residual OSA after surgery is associated with obesity [5,11,12], OSA severity [13], neurodevelopmental comorbidities [11], and craniofacial deformities [11]. Treatment for residual OSA may include revision adenoidectomy. Extant literature indicates that revision adenoidectomy rates vary from 0.6 to 2.6 % [14–17]. Revision surgery has been associated with age <5, middle ear disease, and primary adenoidectomy without tonsillectomy [18,19]. However, BMI has not been addressed to the same extent as these other factors.

Despite childhood obesity being a known risk factor for residual OSA, and previous studies exploring the associations of known residual OSA risk factors with revision surgery, no work has analyzed risks of revision adenoidectomy with respect to BMI. This study aims to elucidate the association between pediatric BMI percentile and the incidence

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of revision adenoidectomy in a cohort with ongoing specialty care management.

2. Methods

This retrospective cohort study examined patients undergoing adenoid removal surgery (either adenoidectomy or AT) at a tertiary care academic children's hospital. Patients received initial adenoid removal surgery between January 2015 and January 2016. Patients without two years of follow-up and patients with missing height and weight data were excluded.

We collected patient demographics, comorbidities, and indications for surgery. In addition, BMI at the time of initial and revision adenoidectomy were recorded. BMI percentiles for patients between the ages of 2 and 20 were calculated using the CDC BMI Percentile Calculator for Children and Teens. Patients were classified as obese if their BMI was greater than or equal to that of the 95th percentile for their age and sex. Children under two years of age were categorized as obese with BMI $\geq$19 at the time of surgery as this value correlates with a BMI >95th percentile according to the CDC calculator. This limit also closely aligns with the percentiles designated by the World Health Organization (WHO) BMI-for-Age Growth Charts. Because the impact of obesity is not well defined in children <2 years of age, these patients were compared categorically as obese or normal weight as above but excluded from further statistical analysis utilizing BMI percentile.

Indications for surgery were grouped into clinically relevant categories. Adenotonsillar hypertrophy (ATH) and adenoid hypertrophy (AH) were all included within the category of ATH as an indication for surgery. Eustachian tube dysfunction (ETD), recurrent otitis media (OME), and other middle ear pathologies were grouped into a single category called middle ear disease. Patients were identified via current procedural terminology (CPT) codes (42820, 42821, 42830, 42831). Monopolar cautery was utilized for all surgical procedures.

The univariate analysis of the associations between BMI percentile and other variables utilized Pearson correlation coefficients for continuous variables and point-biserial correlation for binary variables. Paired *t*-tests were used to compare paired measurements of continuous variables. A logistic regression model was generated to evaluate the factors that may increase or decrease risk of requiring a revision procedure after initial adenoidectomy. Some clinically related comorbidities were combined to reduce risk of overfitting, avoid collinearity, and increase the power of each parameter: asthma and reactive airway disease, hypotonia and cerebral palsy, and OSA and sleep-disordered breathing were all collapsed into single variables after verifying that they were not significant in univariate analysis.

In a multivariable Cox regression analysis, one variable was stratified due to a notable violation of the proportional hazards assumption. A modest residual deviation remained but was deemed acceptable, along with one additional variable with mild non-proportionality, to maintain interpretability and avoid overfitting. Statistical analysis was performed in RStudio v2024.12.1 + 563. This study was approved by the Vanderbilt Institutional Review Board.

3. Results

Of 1948 patients originally identified with CPT codes, 1410 were lost to follow-up and 54 were excluded because only their revision adenoidectomies were performed within the time frame of interest. This left 484 patients, 23 of whom were excluded due to lack of height and weight data. The remaining 461 patients are composed of 267 (58.4 %) males and 194 (41.6 %) females (Table 1). There were 115 (24.9 %) obese patients and 346 (75.1 %) non-obese patients at the time of first intervention. The most common comorbidities in the cohort were allergic rhinitis (45.8 %), asthma (38.2 %), and gastro-esophageal reflux disease (GERD, 34.9 %). The most common indications for the first intervention included ATH (90.7 %), middle ear disease (54.9 %), and recurrent tonsillitis or other infection (29.5 %), among others (Table 2). Age at first intervention ranged from 9 months to 16 years, with a mean and standard deviation of 4.2 ± 2.9 years.

Table 1
Cohort demographics and primary intervention.

$N = 146$	n (%)
Sex	
Male	267 (58.4)
Age	
At primary intervention	4.2 ± 2.9 years old
At secondary intervention	6.2 ± 3.3 years old
Race[a]	
Asian	3 (0.7)
American Indian or Alaskan Native	2 (0.4)
Black or African American	83 (18.2)
Hispanic/Latino/a	18 (3.9)
Middle Eastern or North African	6 (1.3)
Unknown/Not reported	23 (5.0)
White	334 (73.1)
Ethnicity	
Hispanic, Latino/a, or Spanish Origin	59 (12.9)
Not Hispanic, Latino/a, or Spanish Origin	229 (50.1)
Unknown/Not reported	169 (37.0)
Primary intervention type	
Adenoidectomy	205 (44.5)
Adenotonsillectomy	256 (55.5)

[a] Categories are not mutually exclusive.

Secondary intervention was performed on 136 of these patients (29.5 %). The interval between primary and secondary intervention ranged from 4 months to 8 years, with a median of 29 months (IQR 20–46 months). Indications for revision included ATH (74.5 %), OSA (41.6 %), and middle ear disease (41.6 %). Among patients who underwent a revision procedure, BMI percentile was significantly higher at the time of secondary intervention (66.3 ± 31.8) than at the time of primary intervention (62.3 ± 34.1, $p = 0.009$). Furthermore, a longer interval between the two procedures was associated with increased BMI percentile at secondary intervention ($p = 0.002$, $r = 0.265$). However, BMI percentile at primary intervention had no significant relationship with the time interval between procedures. Older age was also correlated with higher BMI percentile at the time of both primary and secondary interventions ($r = 0.237$, $p < 0.001$; $r = 0.233$, $p = 0.007$, respectively).

Increased BMI percentile was negatively correlated with indications of middle ear disease ($r = -0.136$, p = 0.007) and adenotonsillar hypertrophy ($r = -0.142$, $p = 0.005$) for the primary intervention. Trisomy 21 was associated with higher BMI percentile ($r = 0.1$, $p = 0.048$), and cerebral palsy was associated with lower BMI percentile ($r = -0.101$, $p = 0.047$). There were no other significant associations between BMI percentile and indications or comorbidities (Table 2).

In a multiple logistic regression model, there were several significant factors affecting the odds of undergoing a revision procedure (Table 3). If the primary intervention was an adenotonsillectomy rather than an adenoidectomy, the odds of needing a revision were significantly lower (OR = 0.3, $p < 0.001$). Additionally, increased age at primary intervention was associated with decreased odds of requiring revision (OR = 0.844, $p = 0.002$). Patients with increased BMI had significantly higher odds of undergoing revision (OR = 1.008, $p = 0.048$), as did patients with allergic rhinitis (OR = 1.722, $p = 0.039$).

In Kaplan-Meier univariate analysis of the time interval between procedures, patients whose primary intervention was an adenoidectomy had a greater probability of revision over time compared to those who initially underwent adenotonsillectomy ($p < 0.001$, Fig. 1a). Patients who underwent initial surgery at age 5 or younger had earlier onset of revision need compared to those whose primary intervention was performed at over 5 years of age (p < 0.001, Fig. 1b). There was no statistically significant difference in time to revision between obese and non-obese groups (Fig. 1c).

Table 2
BMI percentile, comorbidities, and indications.

	n (%)	BMI percentile n = 94 (mean ± SD)	r	*P*-value
Comorbidities (n = 461)				
Asthma	176 (38.2)	59.7 ± 35.2	−0.003	0.957
Allergic rhinitis	211 (45.8)	62.9 ± 32.6	0.080	0.118
Cerebral palsy	16 (3.5)	42.0 ± 38.8	−0.101	0.047*
Craniofacial abnormality	101 (21.9)	63.4 ± 35.1	0.055	0.279
Trisomy 21	31 (6.7)	73.5 ± 27.8	0.100	0.048*
GERD	161 (34.9)	56.1 ± 34.6	−0.074	0.144
Hypotonia	82 (17.8)	58.6 ± 36.1	−0.015	0.773
Laryngomalacia/ tracheomalacia	69 (15.0)	60.6 ± 34.4	0.009	0.862
Reactive airway disease	76 (16.5)	60.5 ± 33.2	0.007	0.883
Indications for primary intervention (n = 461)				
ATH	418 (90.7)	55.9 ± 35.1	−0.136	0.007**
Middle ear disease	253 (54.9)	55.0 ± 34.6	−0.142	0.005**
OSA	133 (28.9)	63.0 ± 34.4	0.061	0.230
Recurrent tonsillitis or other infection	136 (29.5)	59.5 ± 33.8	−0.006	0.094
Sleep disordered breathing	104 (22.6)	57.3 ± 32.9	−0.042	0.414
Indications for secondary intervention (n = 137)				
ATH	102 (74.5)	66.2 ± 31.2	−0.008	0.931
Middle ear disease	57 (41.6)	68.1 ± 32.0	0.048	0.579
OSA	57 (41.6)	66.8 ± 33.5	0.013	0.884
Recurrent tonsillitis or other infection	51 (37.2)	61.0 ± 30.8	−0.128	0.140
Sleep disordered breathing	31 (22.6)	69.1 ± 31.1	0.048	0.585

Abbreviations – BMI: body mass index, SD: standard deviation, GERD: gastroesophageal reflux disease, ATH: adenotonsillar hypertrophy, OSA: obstructive sleep apnea.
BMI percentile does not include patients < 2 years old.
* $p < 0.05$.
** $p < 0.01$.

Table 3
Multilinear regression, factors associated with revision.

Variable	aOR	95 % CI	P-value
Female sex (vs male)	0.845	[0.516, 1.375]	0.498
Age >5 years old at primary intervention (vs ≤5)	0.844	[0.752, 0.937]	0.002**
BMI percentile at primary intervention	1.008	[1.000, 1.015]	0.048*
Adenotonsillectomy (vs adenoidectomy)	0.300	[0.156, 0.563]	<0.001***
Comorbidities			
Asthma/reactive airway disease	1.382	[0.805, 2.367]	0.239
Allergic rhinitis	1.722	[1.031, 2.895]	0.039*
Craniofacial abnormality	0.857	[0.425, 1.666]	0.657
GERD	0.537	[0.279, 1.003]	0.056
Laryngomalacia/tracheomalacia	1.990	[0.920, 4.270]	0.078
Neuromuscular diagnosis	2.251	[0.835, 5.976]	0.103
Trisomy 21	1.937	[0.632, 5.760]	0.237
Indications for primary intervention			
ATH	1.267	[0.531, 3.289]	0.607
Middle ear disease	0.903	[0.509, 1.596]	0.727
OSA/Sleep disordered breathing	1.310	[0.644, 2.682]	0.457
Recurrent tonsillitis or other infection	0.953	[0.523, 1.719]	0.874

Abbreviations – BMI: body mass index, aOR: adjusted odds ratio, CI: confidence interval, GERD: gastroesophageal reflux disease, ATH: adenotonsillar hypertrophy, OSA: obstructive sleep apnea.
BMI percentile does not include patients < 2 years old.
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

On multivariable Cox proportional hazards analysis (Table 4), four variables significantly impacted time to revision. Patients whose primary intervention was an adenotonsillectomy rather than an adenoidectomy had a decreased risk of undergoing revision over time (HR = 0.358, $p < 0.001$). Older age at primary intervention was associated with lower hazard (HR = 0.858, $p = 0.001$). Patients with GERD had decreased hazard (HR = 0.595, $p = 0.05$) while patients with laryngomalacia or tracheomalacia had increased hazard (HR = 1.909, $p = 0.034$). The concordance of the model was 0.694.

4. Discussion

The primary goal of this study was to evaluate the association of pediatric BMI with revision adenoid surgery in a population with at least two years of post-operative follow-up, a group enriched for comorbid conditions and longitudinal care needs. As the prevalence of obesity is dramatically increasing in the U.S. pediatric population [4], it is necessary to investigate the potential impact of this inflammatory disease on adenoid regrowth and surgical burden. At the time of initial surgery, the obesity rate among our patients was 24.9 %; this is higher than the US average rate of pediatric obesity, which is 19.7 % [4]. In our cohort, higher BMI percentile was associated with increased risk of revision in the logistic regression, along with allergic rhinitis, younger age at primary intervention, and having undergone adenoidectomy without tonsillectomy. However, BMI percentile and allergic rhinitis were not significant in the Cox regression model, suggesting a less time-dependent effect on the need for revision. In contrast, laryngomalacia/tracheomalacia and GERD were not significant in the logistic regression but were associated with increased hazard and decreased hazard, respectively, in the Cox regression, indicating a possible time-dependent effect on adenoid regrowth after primary intervention.

The incidence of revision adenoidectomy in our cohort (29.5 %) was substantially higher than that found in many comparable studies, which typically report rates of up to 2.5 % [14–17]. This discrepancy may be influenced by a patient population that requires longitudinal follow-up, which selects for the most medically complex patients with high comorbidity rates. For instance, an 2017 study done at a tertiary care center found a revision rate of 2.5 %, but featured a cohort with a 0.3 % rate of GERD compared to our 34.9 %, and a 2.4 % rate of allergic rhinitis compared to our 45.8 % [20]. Our patient population also has a higher incidence of obesity than the general US pediatric population, which we have found to increase the risk of adenoid regrowth in this group. Another study used records from a multistate hospital system,

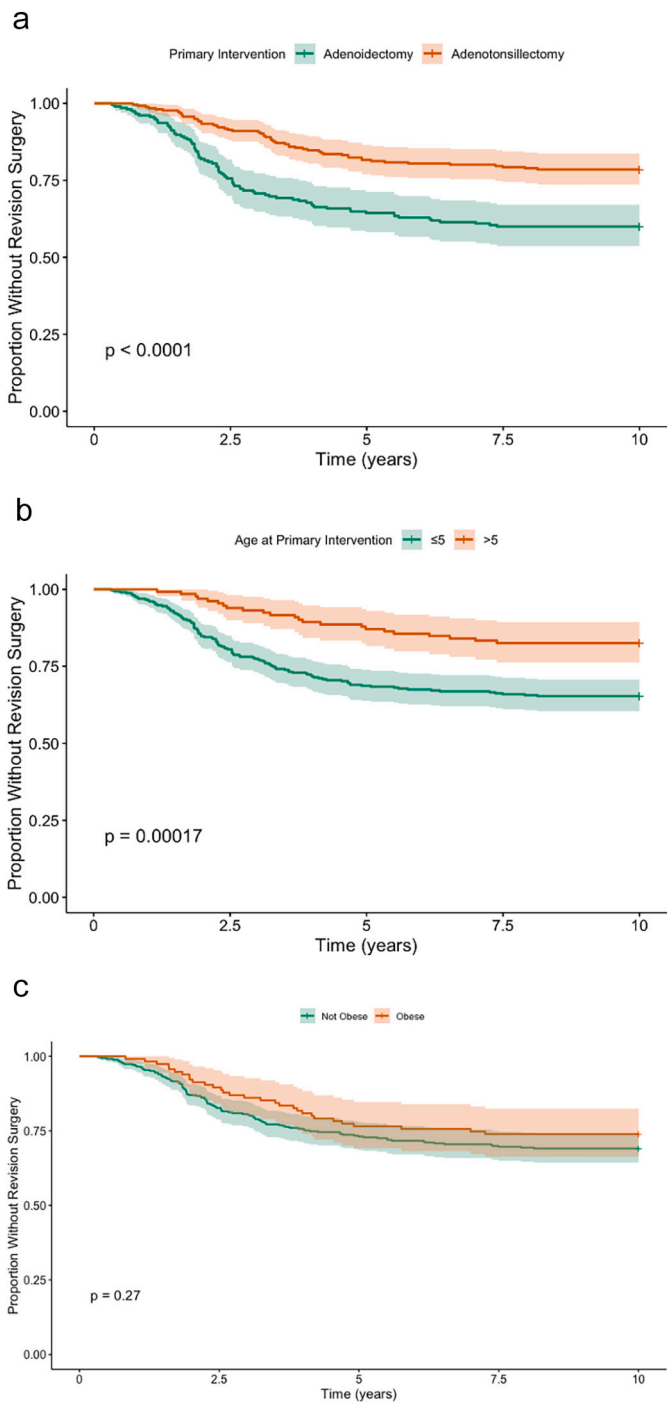

Fig. 1. Revision incidence between various subgroups.
Kaplan-Meier plots of the incidence of revision adenoidectomy with the cohort divided by (a) primary intervention type, (b) age, and (c) obesity status.

with their results reflecting a subset of the general US population, and found a revision rate of 1.3 % [21]. Because of the comorbidity burden of our cohort, we are able to more thoroughly examine the effect of comorbid conditions on adenoid regrowth, as these factors are prevalent at rates that lend them statistical power. While potentially not generalizable to the typical pediatric population, our results emphasize the importance of long-term follow-up, prognostic counseling, and potentially inform decision-making in the care of those pediatric patients with identified risk factors.

Other studies investigating risk factors for revision adenoidectomy have found no link between obesity and revision incidence [20],

Table 4
Multivariable Cox regression predicting time to revision procedure.

Variable	HR	95 % CI	P-value
Adenotonsillectomy (vs adenoidectomy)	0.358	[0.21, 0.612]	<0.001***
Age > 5 years old at primary intervention (vs ≤5)	0.858	[0.781, 0.942]	0.001**
BMI percentile at primary intervention	1.006	[1.000, 1.012]	0.051
Female sex (vs male)	0.843	[0.563, 1.263]	0.408
Comorbidities			
Allergic rhinitis	1.501	[0.998, 2.259]	0.051
Asthma/reactive airway disease	1.279	[0.832, 1.968]	0.263
Craniofacial abnormality	0.887	[0.519, 1.515]	0.661
GERD	0.595	[0.354, 0.999]	0.05*
Laryngomalacia/tracheomalacia	1.909	[1.049, 3.473]	0.034*
Neuromuscular diagnosis	1.819	[0.870, 3.801]	0.112
Trisomy 21	1.511	[0.645, 3.537]	0.342
Indications for primary intervention			
ATH	1.271	[0.603, 2.68]	0.528
Middle ear disease	0.892	[0.563, 1.413]	0.625
OSA/Sleep disordered breathing	1.235	[0.665, 2.296]	0.504
Recurrent tonsillitis or other infection	0.946	[0.578, 1.548]	0.824

Abbreviations – BMI: body mass index, HR: hazard ratio, CI: confidence interval, GERD: gastroesophageal reflux disease, ATH: adenotonsillar hypertrophy, OSA: obstructive sleep apnea.
BMI percentile does not include patients < 2 years old.

consistent with our univariate findings in the Kaplan-Meier analysis (Fig. 1c). However, BMI percentile was significant in the logistic regression, which showed that for each additional BMI percentile point, the odds of undergoing revision increase by 0.8 %. Rather than a significant increase in risk once a child reaches the point of obesity, our results may reflect a more linear relationship between adiposity and AH. Inflammation may be a confounding variable in this correlation based on its links to both AH and obesity [22,23]. For instance, a 2021 study found that elevated levels of IL-32, a pro-inflammatory cytokine, can precipitate or worsen AH [24]. A 2016 study found that obese patients express increased levels of IL-32 in both visceral and subcutaneous adipose tissue compared to non-obese patients, and weight loss has been shown to decrease these levels [25]. If patients with larger and more numerous adipocytes produce higher quantities of pro-inflammatory cytokines, this may play a role in the increased rates of ATH as a function of BMI percentile and corresponding risk of OSA in the obese pediatric population. A 2018 study by Coutras et al. noted that overweight or obese pediatric patients were more likely to have obstruction at the lingual tonsil, adenoid, or tongue base compared with children of normal or low weight [26]. Obesity-related OSA is multifactorial and cannot be explained by AH alone, although markers of systemic inflammation found in adipose tissue may contribute to hypertrophy of multiple areas of lymphoid tissue in the upper airway.

An additional risk factor for revision adenoidectomy is age at primary surgical intervention. Age at first adenoidectomy has been contested as a risk factor for revision in the literature, with some studies reporting lower risk with older age [18,19], some reporting lower risk with younger age [20], and some reporting no association at all [15].

However, we found that for each additional year of age at the time of initial surgery, the odds of revision decreased by 15.6 %, and hazard decreased by 14.2 %. Schneuer et al. noted similar findings to the current study, reporting that revision rates decrease with older age at the time of the initial surgery [19]. As adenoid growth typically slows around age 6 [27], it follows that if an adenoidectomy is performed while growth factors for the tissue are still present, even minimal residual adenoid tissue could result in clinically significant regrowth.

Additionally, adenotonsillectomy seems to have a protective effect when compared to adenoidectomy, decreasing the odds of a revision procedure by 70 % in our cohort. The 2022 Schneuer study noted revision rates to be consistently lower with adenotonsillectomy compared to adenoidectomy [19]. It is important to note that there may be some selection bias in this finding. If children are already undergoing general anesthesia for tonsillectomy, a surgeon may be more likely to revise the adenoid tissue, even if the tissue is minimally obstructive. In our cohort, 44.5 % of primary interventions were adenoidectomies, and the remaining 55.5 % were adenotonsillectomies (Table 1). In cases of tonsillectomy, adding adenoidectomy adds minimal surgical time, morbidity, and risks to the surgical procedure.

In the logistic regression, the presence of allergic rhinitis as a comorbidity increased the odds of requiring a second adenoidectomy by 72.2 %. This finding agrees with prior literature noting that among children with AH, adenoid size is increased in patients with comorbid allergic rhinitis [28]. This is also consistent with the hypothesis that inflammation may worsen AH, as allergic rhinitis causes chronic atopic inflammation in the region of interest [29]. Some studies have suggested that certain allergens may be more implicated in allergic rhinitis-related AH than others [30], which may explain the borderline p-value seen in the current study ($p = 0.039$), given that we did not stratify our atopic rhinitis cases by specific allergen. In the Cox model, allergic rhinitis was not a significant contributor to the hazard of requiring revision over time ($p = 0.056$), potentially because the relationship is not mediated by time-dependency but rather by allergen-specific factors.

GERD was a significant risk factor in the Cox regression, decreasing the hazard of requiring a revision over time by 40.5 % in this group. In contrast with our findings, prior studies of the general population have shown an association between AH and GERD, although the mechanism of the causal relationship has not been elucidated [31,32]. It has been postulated that the adenoids may be directly inflamed by peptic acid in cases of GERD that are severe enough to cause laryngopharyngeal reflux [33]. Another hypothesis implicates *Helicobacter pylori* in the pathogenesis of AH, supported by a 2014 study by Aydin et al. that was able to demonstrate *H. pylori* colonization of the adenoids in some patients with AH [34]. Because GERD was a common comorbidity in our cohort (34.9 %) and because its association with AH seems to require severe disease, perhaps the prevalence of milder cases unrelated to AH contributed to this significant finding rather than GERD serving as a protective factor against AH.

History of laryngomalacia or tracheomalacia increased the hazard of revision adenoidectomy by 90.9 % in the Cox regression. While a direct link between airway malformations and AH has not been established, histopathologic specimens from children with laryngomalacia have revealed concurrent inflammation in the majority of patients, with repeated hypoxemia as one proposed mechanism [35,36]. Additionally, acquired pediatric tracheomalacia can be precipitated by airway inflammation [37]. Thus, both congenital and acquired airway malformations are associated with increased inflammation, which could increase the incidence of adenoid regrowth in these patients.

Strengths of the current study include the large patient population and rigorous statistical analyses. The current study has several limitations. There is selection bias due to the inclusion criterion of 2 years of post-operative follow-up and the large loss of patients due to lack of such follow-up, potentially enriching the studied cohort with more medically complex patients. For this reason, these results may not be generalized to the general pediatric population but may be considered in the care of patients with comorbidities. Data were collected via retrospective chart review; therefore, there may be inaccurate or incomplete documentation of patient information, comorbidities, or surgical procedures. While all patients were followed for at least 2 years after initial surgery, some revisions occurred 8 years later; therefore, the study may underestimate the true frequency of revision surgery in this cohort. This study included patients below the age of 2. In these young patients, the clinical relevance of BMI calculations and obesity classification is largely unknown. It is also important to note that BMI percentile is not an entirely accurate quantification of adiposity because it does not take body composition into account. As stated above, patients undergoing tonsillectomy may be more likely to have concurrent revision adenoidectomy, regardless of adenoid size. Finally, variations and/or incomplete descriptions of adenoid tissue at the time of surgery prevented further stratification by adenoid size.

5. Conclusions

Increased BMI percentile, younger age at primary intervention, allergic rhinitis, and adenoidectomy without tonsillectomy may increase the risk of requiring a secondary adenoidectomy. Age and initial surgery type may impact the interval between procedures as well as whether a revision becomes necessary in this sub-population. Further understanding of obesity-induced inflammation might inform guidelines for treatment and management of AH and OSA in the obese pediatric population. Future prospective studies that objectively assess AH and/or utilize anti-inflammatory medications may allow improved understanding of the interaction of inflammation, AH, and pediatric OSA.

CRediT authorship contribution statement

Liliana Arida-Moody: Conceptualization, Data curation, Formal analysis, Methodology, Writing – original draft, Writing – review & editing. **Daniel R.S. Habib:** Conceptualization, Formal analysis, Methodology, Writing – review & editing. **Emma H. Neal:** Conceptualization, Data curation, Formal analysis, Methodology, Writing – original draft, Writing – review & editing. **Amy S. Whigham:** Conceptualization, Methodology, Writing – original draft, Writing – review & editing.

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Declaration of competing interest

The authors report no conflicts of interest.

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Data availability

The dataset underlying this study contains protected health information and cannot be shared publicly due to patient privacy concerns and institutional review board (IRB) restrictions. De-identified data may be made available upon reasonable request to the corresponding author, subject to IRB approval and completion of a data use agreement. Statistical analyses were conducted in RStudio (v2024.12.1+563), and

analysis code is available from the corresponding author upon request.

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