

Risk Factors for Adenoidal Regrowth Among Patients in a Pediatric Allergy Practice

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Hypertrophied adenoid glands are a common finding in pediatric patients with obstructive nasal symptoms. Recurrence of the symptoms following adenoidectomy is reported. One possible etiology for recurrent symptoms is regrowth of the adenoid tissue; however, prevalence of and risk factors for regrowth are not well delineated. The study was undertaken to determine the frequency of regrowth of adenoid tissue in a population of post-adenoidectomy patients presenting to a pediatric allergy office with obstructive nasal complaints, as well as to identify potential contributing risk factors in this population. Patients presenting with obstructive nasal symptoms, status post-adenoidectomy were retrospectively identified. Charts were reviewed for evidence of adenoid regrowth on imaging studies. A retrospective analysis was then performed comparing the presence and absence of adenoid regrowth with aeroallergen sensitivity, sinusitis, gastroesophageal reflux disease (GERD), tobacco exposure, asthma, and immunodeficiency disorders. One hundred and six patients' histories were reviewed for this study. Forty-five percent of patients had evidence of adenoid regrowth on imaging studies, while 55% did not. The presence of GERD was more common in the group with adenoid regrowth ($P = 0.012$). Otherwise, the groups did not differ significantly. Among patients presenting with obstructive nasal symptoms following adenoidectomy, GERD was significantly more prevalent in those with regrowth of adenoid tissue.

Introduction

THE ADENOID GLANDS, LOCATED in the nasopharynx, are part of the Waldeyer ring. This entity, composed of lymphoid tissue encircling the pharynx, also includes the palatine and lingual tonsils. Although the Waldeyer ring serves a protective role, hypertrophied adenoid tissue can lead to significant morbidity including obstructive sleep apnea, cor pulmonale, neurocognitive disturbances, and growth failure.^{1,2} Adenoidal hypertrophy has also been implicated in chronic mouth-breathing, persistent middle ear effusions, and recurrent or chronic rhinosinusitis.³ The underlying cause of adenoidal hypertrophy is unknown but is likely multifactorial.⁴ Previously associated risk factors for hypertrophied adenoids include allergic rhinitis,^{4,5} mold sensitivity,⁴ tobacco smoke exposure,⁶ polymicrobial infection,⁷⁻⁹ and abnormal mucociliary clearance.¹⁰ Association between adenoidal hypertrophy and gastroesophageal reflux disease (GERD) has also been noted in the literature.¹¹⁻¹⁴

Adenoidectomy, one of the most common surgeries performed in the pediatric age group, is successful in alleviating obstructive nasal symptoms in the majority of patients with

hypertrophied adenoid glands.¹⁵ Despite these results, recurrence of obstructive nasal symptoms is reported in 14% to 26% of cases. The possibility exists for residual lymphoid tissue to hypertrophy and thus lead to regrowth of the gland. Other area tissue also has potential for hypertrophy such as in the case of tubal tonsillar hypertrophy. Data is limited regarding the frequency and etiology of regrowth of adenoids, although recent interest is noted in the literature.¹³ This study set out to determine the frequency of regrowth of adenoid tissue in a population of post-adenoidectomy patients presenting to a pediatric allergy office with obstructive nasal complaints, as well as to identify potential risk factors. This information would serve as a clinical tool for practicing allergists to guide the work-up and management of these patients.

Materials and Methods

Study population

Patients selected for this study were those with history of adenoidectomy presenting to an academic pediatric allergy

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practice with recurrent obstructive nasal symptoms (including persistent mouth-breathing or snoring) from January 2001 to January 2005. The Institutional Review Board at our hospital approved the study.

Study design

Patients were identified retrospectively and considered in 2 groups, those with adenoid regrowth and those without. Adenoid regrowth was defined as the presence of enlarged adenoid tissue on lateral neck radiographs done at the time of allergy evaluation. The radiographs, performed with the mouth-closed and brow-up position, were graded as mild, moderate, or severe adenoid enlargement relative to the degree of nasopharyngeal airway obstruction and were read by the same pediatric radiologist. Adenoid tissue bulging into the nasopharynx with no airway occlusion is deemed mild hypertrophy; moderate enlargement narrows the nasopharyngeal airway without occlusion, while severe hypertrophy involves occlusion of the nasopharyngeal airway.

Data analysis of these groups was performed with particular attention paid to history of aeroallergen sensitivity, sinusitis, GERD, tobacco exposure, and concurrent conditions. Surgical method of adenoidectomy was noted when the information was available.

1. *Aeroallergen sensitivity*: As part of their allergy evaluation, percutaneous skin prick testing to aeroallergens was performed. The timing of the prick testing varied among subjects. In some patients, testing was performed prior to adenoidectomy, while in others, testing followed the adenoidectomy. Testing in patients younger than 3 years old included dust mites (*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*), cat, dog, cockroach, and mold mix (total of 10). Patients over 3 years underwent additional skin prick testing to grass mix, Bermuda grass, weed mix, ragweed, alternaria, cladosporium, penicillium mix, and *Aspergillus fumigatus*. A wheal of 3 mm greater than saline control was considered positive.
2. *Sinusitis*: Patients with symptoms suggestive of acute or chronic sinusitis such as offensive nasal discharge,

halitosis, persistent postnasal drip, and facial discomfort, or tenderness underwent sinus imaging with plain films or computed tomography (CT).

3. *GERD*: Historical features suggestive of reflux including: presence of reflux during infancy; chronic cough, poorly controlled asthma, overt emesis, heartburn, oral brash, epigastric abdominal pain, recumbent cough, hoarseness, or frequent burping were identified. Diagnostic evaluations including esophageal pH probe, gastric scintigraphy, barium swallow/upper gastrointestinal series, and upper endoscopy with biopsy were noted.
4. Documentation of passive tobacco smoke exposure, concurrent asthma, and immunodeficiency disorders was reviewed.

Statistical analysis

Chi-square analyses were performed on the demographics; tobacco exposure; and presence of sinusitis, GERD, and asthma. Independent samples *t* tests were carried out on the age and time between adenoidectomy and allergy visit. SPSS (SPSS Inc., Chicago, IL) was used for all analyses.

Results

One hundred and six patients, status post-adenoidectomy, with recurrence of obstructive nasal symptoms, were identified for this study.

Demographics

Patient demographics are shown in Table 1. Mean age ($\pm$ SD) at allergy evaluation was 7 ± 3.2 years; median 6.25 (range: 2.25–16.5 years). Adenoidectomy was performed at 4 ± 2.2 years; median 3.4 (range: 6 months to 12.4 years). Mean time interval from adenoidectomy to allergy evaluation was 3 ± 2.3 years; median 2 (range: 3 months–14 years).

Lateral neck radiographs revealed adenoid regrowth in 48/106 (45%) patients, while 58/106 (55%) had no adenoid tissue noted. The degree of adenoidal hypertrophy was mild in 24/48 (50%), moderate in 21/48 (44%), and severe

TABLE 1. DEMOGRAPHICS OF THE STUDY POPULATION AND TIME ELAPSED BETWEEN SURGERY AND REEVALUATION

	Adenoidal regrowth (n = 48)	Non-regrowth of adenoids (n = 58)	P value
Sex	Male: 32 (67%) Female: 16 (33%)	Male: 33 (57%) Female: 25 (43%)	0.3
Race	Caucasian: 25 (52%) Other: 23 (48%)	Caucasian: 45 (78%) Other: 13 (22%)	0.006
Age at adenoidectomy	1–7.3 y (mean: 3.6 ± 1.6 y) (median: 3.3 y)	6 m–12.4 y (mean: 4.4 ± 2.5 y) (median: 3.8 y)	0.063
Age at reevaluation	2–14 y (mean: 6.25 ± 2.7 y) (median: 5.9 y)	2–16 y (mean: 7.6 ± 3.5 y) (mean: 6.7 y)	0.024
Time between surgery and reevaluation	3 m–7 y (mean: 2.6 ± 1.9 y) (median: 2 y)	4 m–14 y (mean: 3.25 ± 2.5 y) (median: 2 y)	0.155

Abbreviations: m, months; y, years.

in 3/48 patients (6%). Four patients underwent revision adenoidectomy.

With regard to gender, boys outnumbered girls in both groups ($P = 0.3$). Patients without adenoid regrowth were predominantly Caucasian (78% versus 52%, $P = 0.006$). Age at primary adenoidectomy was similar between the 2 groups (3.6 ± 1.6 years; median 3.3 (regrowth) versus 4.4 ± 2.5 years; median 3.8 (non-regrowth), $P = 0.063$). Those with adenoidal regrowth presented with recurrent symptoms at a younger age (6.25 ± 2.7 ; median 5.9 years versus 7.6 ± 3.5 ; median 6.7 years, $P = 0.024$). The mean time elapsed between adenoidectomy and subsequent evaluation was similar in the 2 groups (2.6 ± 1.9 ; median 2 years (regrowth) versus 3.25 ± 2.5 ; median 2 years (non-regrowth), $P = 0.156$).

Risk factors

Risk factors are shown in Table 2.

Aeroallergen sensitivity. Aeroallergen sensitivity was equivalent between the 2 groups (40%, $P = 0.83$); the most common sensitivities were pollens, followed by molds, dust mites, animals, and cockroaches.

Sinusitis. Sinus disease was suspected and imaging available for 82/106 patients (77%): 37/48 (77%) of the adenoid regrowth group and 45/58 (78%) of the non-regrowth group. Among the 37 patients from the adenoid regrowth group, 19/37 (51%) underwent CT scan, and 18/37 (49%) sinus radiographs. Twenty six of the 37 (70%) patients had abnormal imaging findings including mucosal thickening or sinus opacification.

The non-regrowth group had similar findings: 29/45 (64%) had CT scans; 16/45 (36%) had sinus radiograph; abnormalities were noted in 25/45 (56%) studies ($P = 0.17$).

GERD. History included subjective assessment for GERD for all patients. Forty patients (40/106; 38%) had objective GERD evaluation, 23 patients from the adenoid regrowth group (23/48; 48%), and 17 from the non-regrowth group (17/58; 29%). The type of test performed was not uniform (illustrated in Table 3), all were included in the analysis. Of significance, 70% of objective GERD evaluations were positive among the adenoidal regrowth group, compared with 29% among the non-regrowth group ($P = 0.012$). When subjective assessment for GERD was included in the analysis, the difference between the groups became more pronounced (75% versus 31%, respectively, $P = 0.001$).

Second-hand tobacco smoke exposure. This was similar in the 2 groups ($P = 0.5$).

Co-morbidities. Asthma was present in 63% of the adenoidal regrowth group and 72% of the non-regrowth group

($P = 0.28$). One child had partial IgA deficiency in the adenoidal regrowth group. In the non-regrowth group, 1 child had hypogammaglobulinemia and was receiving monthly intravenous immunoglobulin infusions; 3 others had partial IgA deficiency.

Surgical method

Medical records indicated method of surgery in 61/106 patients, 29/48 (60%) in the adenoidal regrowth group and 32/58 (55%) in the non-regrowth group. In the regrowth group, methods included: electrocautery with suction Bovie (21/29), curettage (4/29), and surgical microdebrider (4/29). In the non-regrowth group, methods included: suction Bovie (18/32), curettage (13/32), and surgical microdebrider (1/32). The available numbers were too small for any meaningful statistical analysis.

Discussion

Regrowth of adenoid tissue was diagnosed in 45% of 106 patients presenting post-adenoidectomy to an allergy practice complaining of obstructive nasal symptoms. Half of this group had moderate to severe adenoid enlargement, and 4 patients required repeat surgery. The average age of the patients with regrowth was 6.25 years, 17 months younger than those with no regrowth. Despite this difference, the age at adenoidectomy and the interval from surgery to subsequent reevaluation did not vary significantly between the 2 groups. Caucasians were significantly predominant in the non-regrowth group. Of the risk factors examined, the groups differed significantly only in presence of GERD.

This study's initial goal was to define the frequency of adenoid regrowth. Our findings of 45% may be influenced by a biased population and may not reflect the true prevalence of adenoid regrowth. Buchinsky et al. reported lower prevalence when they conducted telephonic interviews with 175 post-adenoidectomy patients.¹⁵ Forty six of these patients were symptomatic (ie, nasal obstruction) and 35/46 underwent nasopharyngoscopy. The mean age at follow-up was 9.2 years, and time after surgery ranged from 2.1 to 5.1 years (mean 3.3 years). Enlarged adenoid glands, resulting in nasopharyngeal obstruction ranging from 10% to 40%, were documented in 71% of these patients. However, no patients met the criterion for significant adenoid enlargement, defined as 50% or greater obstruction of the nasopharynx. Furthermore, Wong and colleagues prospectively examined 23 children after electrosurgical adenoid ablation. The average age of these patients was 6 years (range 3–12 years).

TABLE 2. RISK FACTORS FOR ADENOIDAL REGROWTH

	Adenoidal regrowth (n = 48)	Non-regrowth of adenoids (n = 58)	P value
Aeroallergen sensitivity	20 (42%)	23 (40%)	0.83
Tobacco exposure	16 (33%)	23 (40%)	0.5
Sinusitis	26/37 (70%)	25/45 (56%)	0.17
GERD	Total: 36	Total: 18	0.001
	Symptoms only: 20	Symptoms only: 13	0.012
	Positive studies: 16	Positive studies: 5	

Abbreviation: GERD, gastroesophageal reflux disease.

TABLE 3. STUDIES PERFORMED FOR GERD

	<i>Adenoidal regrowth (positive/total performed)</i>	<i>Non-regrowth of adenoids (positive/ total performed)</i>
EGD	5/5	1/3
UGI	6/8	1/3
Scintigraphy	5/10	3/9
pH probe	0/0	0/2

Abbreviations: EGD, esophagogastroduodenoscopy; GERD, gastroesophageal reflux disease; UGI, upper gastrointestinal series.

Postoperative evaluation at 6–8 weeks, by nasopharyngoscopy, found 3 children with tiny residual tissue and 1 child with adenoidal hypertrophy obstructing at least two-thirds of the choanae. Six of the other patients followed for 1 year after surgery showed no regrowth.¹⁶ To review and compare, nearly half of our population was diagnosed with some degree of adenoid regrowth, 50% of whom had moderate to severe obstruction of the nasopharynx on radiography. Our population was younger, yet the mean time interval between evaluation and surgery was similar to the Buchinsky group. Perhaps, the discordant findings are due to the use of lateral neck radiography for diagnosis versus nasopharyngoscopy. The limitations of lateral neck radiography are widely discussed in the literature. One may hypothesize that we overestimated the degree of hypertrophy, and therefore the frequency of regrowth, however, in support of our data, lateral neck radiograph has been shown to closely approximate intraoperative findings in 85% of patients; furthermore, radiologists underestimated the size of adenoid tissue and degree of nasopharyngeal obstruction in 75% of cases where there was disparity between techniques.¹⁷ A separate study comparing lateral neck radiograph with nasopharyngoscopy showed 66% sensitivity and 96% specificity of the former.¹⁸ Taking this into account, it would suggest that lateral neck radiograph, while by no means perfect, may have underestimated the true degree of nasopharyngeal obstruction in our patients and that our findings may have had a greater degree of significance with the use of nasopharyngoscopy. To the contrary, in a recent study comparing lateral neck radiography with nasopharyngoscopy and video fluoroscopy, radiography was inconsistent in defining the degree of hypertrophy and both over and underestimated the adenoid size making it generally unreliable. With this information, our data would be significantly limited in that we utilized radiography only to make the diagnosis of adenoid hypertrophy.¹⁹

Another potential confounding variable affecting frequency of regrowth is tubal tonsillar hypertrophy. A recent study examining 24 patients scheduled for revision adenoidectomy found 13 with recurrent adenoid tissue during nasopharyngeal examination under anesthesia and 10 patients with tubal tonsillar hypertrophy—4 members of the latter group had lateral neck radiograph interpreted as adenoidal hypertrophy.²⁰ Therefore, tubal tonsillar hypertrophy may be misinterpreted as enlarged adenoid glands on lateral neck radiograph leading to inaccurate results when radiographs are relied upon for diagnosis.

The follow-up question for how frequently adenoids regrow is why does it happen at all? Inadequate removal at adenoidectomy is postulated but difficult to show in retrospect. This could stem from variable surgical technique versus inherent differences among surgical modalities. We are unable to comment from our data given the numbers of identified surgical methods were too small to compare and analyze statistically.

With regard to other risk factors, we chose to focus on issues previously associated with primary adenoid hypertrophy. Surprisingly, there was no significant difference between our groups when compared for aeroallergen sensitivity, tobacco smoke exposure, and concurrent sinusitis. GERD, however, was significantly more prevalent in the adenoid regrowth group.

Previous studies have addressed associations among GERD, sinusitis, and other upper and lower airway pathologies.^{21–23} Several groups cited association of GERD specifically with adenoid hypertrophy. Contencin et al. found a correlation between the need for early adenoidectomy and history of prolonged regurgitation after the first months of life.¹¹ More recently, Carr et al. retrospectively examined 95 adenoidectomy patients and found 42% to have GERD, compared with 7% in a control group.¹² Furthermore, Monroy et al. found extraesophageal reflux in a proportion of 72 patients who underwent revision adenoidectomy (40% of their population was studied for reflux, 96% had positive studies).¹³ Finally, a recent case report discussed a 3-year-old child with adenoid hypertrophy found to have extraesophageal reflux who objectively responded to reflux therapy on repeat visualization.¹⁴

Reasons for GERD favoring regrowth of adenoid tissue are not well defined; however, reflux-related injury of the nasopharynx may result in edema and inflammation in the upper airways, serving as stimulus for adenoid hypertrophy.¹² Additional explanations include recurrent sinusitis in association with GERD as a source of bacterial infection and subsequent inflammation of the adenoids. Furthermore, persistent nasal obstruction may alter endothoracic pressures and ultimately perpetuate GERD.¹¹ More credible data would require a prospective study with a control group comprised of asymptomatic, post-adenoidectomy patients. However, our findings are suggestive of GERD as a contributor to adenoid regrowth and are supported in the literature by other groups as discussed above.

Limitations in our data include all those inherent to a retrospective study. Although we found a significant proportion of our post-adenoidectomy patients have adenoid regrowth, the true prevalence is unknown. There is a selection bias given the study only included patients with persistent nasal symptoms. Furthermore, objective GERD studies were performed in 48% of patients with adenoid regrowth compared with 29% in the non-regrowth group. Because of the inequity of formal evaluations between the 2 groups, there may be selection bias and the true prevalence of GERD in the study population may be misrepresented.

In conclusion, our study confirms that adenoid regrowth exists and may contribute to persistent nasal obstructive symptoms. Non-Caucasian and younger patients are more likely to have adenoid regrowth. Aeroallergen sensitivity and tobacco smoke exposure were not significant contributing factors. Prevalence of sinus disease was similar in the

2 groups. GERD, as assessed by symptoms and diagnostic investigation, was significantly more prevalent in the group with adenoid regrowth. These findings provide allergists with practical information, helpful in the evaluation of patients with recurrent obstructive nasal symptoms and regrowth of adenoid tissue. Further studies with a prospective design are needed to confirm this association.

Author Disclosure Statement

No competing financial interests exist.

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