

Adenoidal hypertrophy and allergic rhinitis: Is there an inverse relationship?

Franco Ameli, M.D.,¹ Fabio Brocchetti, M.D.,¹ Maria Angela Tosca, M.D.,² Alessio Signori, M.Sc.,³ and Giorgio Ciprandi, M.D.⁴

ABSTRACT

Background: Nasal obstruction is a very common symptom in children. The main causes are allergic rhinitis (AR) and adenoidal hypertrophy (AH); the possible correlation between AR and AH has been investigated by few studies, mainly conducted using radiographic craniometry. This study aimed at investigating this topic by nasal endoscopy.

Methods: There were 205 children (134 boys; mean age, 6.7 years age range, 4–12 years) studied. Clinical visit, nasal endoscopy, and skin-prick test were performed in all patients. Anterior nasal obstruction was graded using the Friedmann's classification. Adenoid size was graded using the Parikh's classification. Perception of symptoms by children was also assessed using the visual analog scale.

Results: Ninety-two children (44.9%) had complete nasal obstruction and 28 children (13.7%) had choanae invasion. There was a negative significant correlation ($r = -0.41$; $p < 0.001$) between nose obstruction severity and volume of adenoids. Decreased probability of greater adenoid volume was associated with increased severity of nose obstruction (odds ratio [OR] = 0.13) and in patients with allergy compared with nonallergic patients (OR = 0.31).

Conclusion: This real-life study shows that large adenoids may be associated with absence of allergy, whereas large turbinates may be associated with small adenoids.

(Am J Rhinol Allergy 27, e5–e10, 2013; doi: 10.2500/ajra.2013.27.3854)

The adenoids are a conglomerate of peripheral lymphatic tissue, mainly constituted by B-cell lymphocytes (50–65% of all adenoidal lymphocytes) and T cells (~40% of adenoidal lymphocytes). Adenoid tissue is situated in the roof of the rhinopharynx. The adenoids are part of the lymphoid tissue that circle the pharynx, collectively defined as the Waldeyer's ring. This ring includes the lingual tonsil (on the base of the tongue), the two palatine tonsils, the lymphoid tissue placed on the posterior wall of the pharynx, and the adenoids. The Waldeyer's ring physiologically serves as a defense against respiratory antigens (microbes, allergens, etc.). Therefore, the adenoid tissue may play a significant role in the adaptive immune response because of its peculiar position at the entry of the upper aerodigestive tract. As a consequence of chronic stimulation, the adenoids may enlarge so that they may almost fill the space between the choana and rhinopharynx, interfering with the passage of the nasal airflow, obstructing the Eustachian tube, and blocking the clearance of the nasal mucus. Adenoidal hypertrophy (AH) is detected in ~1/3 of the general pediatric population and constitutes the most frequent otorhinolaryngological indication for surgical intervention.^{1,2} AH has been associated with nasal obstruction, snoring, sleep apnea, recurrent otitis media, recurrent rhinosinusitis infections, and craniofacial anomalies.³ On the other hand, nasal obstruction is a frequently encountered problem in the pediatric age and it is a nonspecific symptom associated with a variety of disorders, but a proper assessment is mandatory before starting any treatment. In this regard, because of the localization in the posterior wall of the rhinopharynx, the measurement of both the adenoid pad and the airflow obstruction represents a challenging issue. Several modalities to quantify adenoids and the relationships with the upper airways have been proposed: acoustic rhinomanometry, rhinomanometry, endoscopy, intraoperative mirror

rhinopharyngoscopy, and radiographic assessments.⁴ However, the most commonly used preoperative modalities in the clinical practice are lateral neck films and nasal endoscopy.⁵ Several studies compared these two methods to evaluate adenoid size.^{4–11} Nevertheless, the method for evaluating adenoid size involving the direct visualization of the rhinopharynx should be considered the favorite. Thus, nasal endoscopy is believed to be the most accurate method because it provides a direct view of the adenoid pad.⁴

Pathological enlargement of adenoids has been assumed to be the result of prolonged antigenic stimulation associated with chronic inflammation. Therefore, inflammatory changes within the nasal and sinus mucosa could affect the adenoids because they are the most closely situated cluster of organized lymphatic tissue.¹² The physical consequence of adenoidal enlargement is the limitation of airflow. The child may be able to perceive the nasal obstruction symptom with a good reliability, and the subjective perception is constant and frequently worsening, but the child can not obviously recognize the cause of the airflow impairment.¹³ The two most relevant inflammatory conditions in children with nasal obstruction are respiratory infections and allergy. The first mainly mediated by a Th1 immune response, the last by a Th2-polarized response. However, it is well known that children with allergic rhinitis (AR) usually have lymphoid hypertrophy of the upper airways, mainly concerning the adenoids.¹⁴ The possible correlation between allergy and AH has been investigated by few studies.^{12,15–22} Nevertheless, most of these studies were performed using the radiographic craniometry to measure adenoid volume. Therefore, the present study aimed at investigating the possible relationship between adenoid size and allergy in a group of children complaining of nasal obstruction using the nasal endoscopy.

MATERIALS AND METHODS

Patients

Globally, 205 children (71 girls and 134 boys; mean age, 6.7 ± 2.6 years; age range, 4–12 years) affected by persistent upper airway obstruction were consecutively referring to the Ear, Nose, and Throat (ENT) Unit of Villa Montallegro and enrolled into the study. Inclusion criteria consisted of complaints of nasal obstruction (mouth breathing, with or without snoring). Exclusion criteria were (i) a craniofacial syndrome, (ii) recent facial trauma, (iii) significantly deviated septum,

From the ¹Ear, Nose, and Throat Unit, Villa Montallegro Private Clinic, Genoa, Italy, ²Pneumologic and Allergological Paediatric Unit, Istituto G. Gaslini, Genoa, Italy, ³Department of Health Sciences, Section of Biostatistics, Genoa University, Genoa, Italy, and ⁴Allergy and Respiratory Diseases Clinic, Istituto Di Ricovero e Cura a Carattere Scientifico–University Hospital San Martino, Genoa, Italy
The authors have no conflicts of interest to declare pertaining to this article
Address correspondence and reprint requests to Giorgio Ciprandi, M.D., Viale Benedetto XV 6, 16132 Genoa, Italy
E-mail address: gio.cip@libero.it
Copyright © 2013, OceanSide Publications, Inc., U.S.A.

(iv) a concomitant acute rhinosinusitis, and (v) current use of anti-inflammatory and antiallergic drugs.

The local Review Board approved the study design, and the parents of the children gave their informed consent.

Study Design

All children were evaluated by clinical visit, nasal endoscopy, and skin-prick test. The parameters considered were duration of the symptom, perception of nasal obstruction, sensitization, and endoscopic findings (including severity of nose obstruction and adenoid volume).

Perception of Nasal Obstruction

The assessment of the nasal obstruction perception was evaluated in all children by a faces rating scale (FRS) and by a simplified version of the visual analog scale (VAS) at 4 points as previously reported.¹³ FRS ranged on an ordinal scale from 0 (less critical, such as nose completely patent) to 5 (more critical, such as nose completely obstructed), and the simplified version of VAS took on values from 1 to 4: 1, "no nasal obstruction"; 2, "blocked up nose in a light manner"; 3, "blocked up nose"; 4, "completely blocked up nose". FRS is an adaptation of the picture projection technique in which six faces are shown to a child. The first picture is a very happy smiling face and the last is a sad one: they are similar to "emoticon." The pictures in between show varying degrees of sadness.

Endoscopy

Endoscopy was performed with a pediatric rigid endoscope diameter of 2.7 mm with a 30° angle of vision (Karl Storz cod. 7207 ba; Karl Storz, Milan, Italy) with a 300-W cold light source (Storz Xenon Nova cod. 20134001; Karl Storz) and a light cable of 1.8-mm length. Endoscopy was video recorded by a micro camera connected to a digital recorder set (Karl Storz Tele Pack, cod. 20043002-020; Karl Storz). A flexible endoscope (3-mm diameter) was used in restless children and in those with narrow nasal fossa due to anatomic abnormalities. The children laid supine with their heads bent by ~45°. Some cotton wool soaked with anesthetic solution (ossibuprocaine 1%) was placed into the nose for 5 minutes.

The complete description of the procedure was previously described in detail.²³ Briefly, the nasal fossa was evaluated in three steps that allowed investigation of the following anatomic structures according to Lang's description²⁴: (i) the inferior turbinate and its relationship with the inferior meatus, the inferior part of the septum, the aspect of the mucosa and the presence of secretion, and the rhinopharynx; (ii) the maxillary line that begins superiorly at the middle turbinate attachment corresponding at the agger nasi area), the olfactory tract when possible, the middle turbinate, and its contacts with septum or uncinate process; (iii) the uncinate process, the middle meatus and the half posterior of the nasal septum the ethmoidal bulla and its mucosal contacts, and the sphenoethmoidal recess (this step was possible only when the space inside the nasal fossa was adequate, otherwise, sometimes after local decongestion).

Nose Obstruction Assessment by Endoscopy

Inferior turbinates were evaluated during endoscopy and the size was graded from I to III according to the Friedman's classification.²⁵ Grade I was defined as mild enlargement with no obvious obstruction. Grade III was a complete occlusion of the nasal cavity. The turbinates in between were graded as II.

Adenoidal Volume Assessment

The patients were evaluated by nasal endoscopy for adenoid hypertrophy. The adenoids were graded according to Parikh's classification that is based on the anatomic relationships between the adenoid tissue and the vomer, soft palate, and torus tubarius.²⁶ The

grading is based on the relationship between the adenoids and the adjacent structures when the patient is at rest (*i.e.*, when the soft palate is not elevated). Specifically, grade 1 adenoids are nonobstructive and are not in contact with any of the previously mentioned anatomic subsites; subsequently, grades 2, 3, and 4 adenoids touch the torus tubarius, vomer, and soft plate (at rest), respectively.

Skin-Prick Test

Allergy was assessed by the presence of sensitization to the most common classes of aeroallergens using a skin-prick test. It was performed as stated by the European Academy of Allergy and Clinical Immunology.²⁷ The allergen panel consisted of the following: house-dust mites (*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*), cats, dogs, grasses mix, Compositae mix, *Parietaria judaica*, birch, hazel trees, olive trees, cypress, *Alternaria tenuis*, *Cladosporium*, and *Aspergilli* mix. The concentration of allergen extracts was 100 immune reactivity/mL (Stallergenes, Milan, Italy). A histamine solution in distilled water (10 mg/mL) was used as positive control and the glycerol-buffer diluent of the allergen preparations was used as negative control. Each patient was skin tested on the volar surface of the forearm using 1-mm prick lancets (Stallergenes). The skin reaction was recorded after 15 minutes by evaluating the skin response in comparison with the wheal given by the positive and the negative control. A wheal diameter of at least 3 mm was considered as a positive reaction. The AR diagnosis was made if nasal symptom history was concordant with sensitization.

Statistical Analysis

Mean and SD or median and interquartile range for continuous variables and counts and percentages for categorical ones were reported. The Spearman's rank correlation coefficient (ρ) was used to assess correlation between ordinal clinical variables, such as adenoids volume and nose obstruction on Friedman scale. The association between adenoids volume and allergy or nose obstruction was assessed by χ^2 -test. Finally, ordinal logistic regression model (adenoid volume was the dependent variable) was used to assess the relation with nose obstruction and allergy, considering the possible confounding effect of age and disease duration.

Odds ratios (OR) of higher levels on scales of categorical variables compared with lower level (reference) were reported together with 95% CI. These ORs represent increase or decrease of probability to obtain higher values of the dependent variable, such as the adenoids volume.

A value of $p < 0.05$ was considered statistically significant. SPSS Version 19 (IBM Corp., New York, NY) was used for computation.

RESULTS

The demographic and clinical characteristics of patients are reported in Table 1. Children had a mean age of 6.7 years (SD, 2.6 years), symptom duration of 12 months (interquartile range, 6–20 months), and most of them were allergic (156 patients; 76.1%). In particular, ~1/3 of allergic children were polysensitized. The assessment of nasal obstruction using the Friedmann scale revealed that 92 children (44.9%) had complete nasal obstruction at endoscopic evaluation and 76 (37.1%) had a partial obstruction. Twenty-eight children (13.7%) had choanae invasion (grade 4 of adenoid volume), and 45 (22%) had grade III. The assessment of nasal obstruction by VAS showed that 16 children (7.8%) reported the value of 4, and 104 (50.7%) reported the value of 3; using FRS, 13 children (6.3%) reported the value of 5, 49 children (23.9%) reported 4, and 94 (45.9%) reported 3. In addition, 57 children had volume 1 of tonsils; 82 had volume 2; 60 had volume 3; and 6 had volume 4.

The distribution of children according to the volume of adenoids and the presence of allergy, including the number of sensitizations, is shown in Fig. 1. Among the patients with a grade 1 (the lowest) or 2 of volume of adenoids, respectively, 60.8 and 63.8% were monosen-

Table 1 Demographic and clinical characteristics of patients

	Mean (SD)/Median (IQR)/n (%)
Age (yr)	6.7 (2.6)
Symptom duration (mo)	12 (6–20)
Nose obstruction (Friedmann)	
1	37 (18)
2	76 (37.1)
3	92 (44.9)
Allergy	
No	49 (23.9)
Monosensitization	103 (50.2)
Polysensitization	53 (25.9)
Adenoid volume	
1	74 (36.1)
2	58 (28.3)
3	45 (22)
4	28 (13.7)
VAS	
1	11 (5.4)
2	74 (36.1)
3	104 (50.7)
4	16 (7.8)
FRS	
0–1	7 (3.5)
2	42 (20.5)
3	94 (45.9)
4	49 (23.9)
5	13 (6.3)

IQR = interquartile range; FRS = faces rating scale; VAS = visual analog scale.

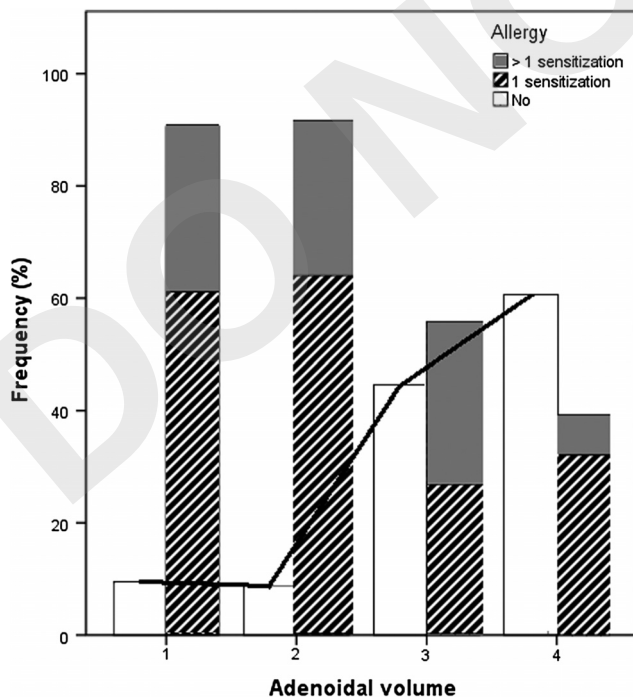


Figure 1. Frequencies of sensitizations in children with adenoidal hypertrophy, according to adenoidal volume.

sitized, and on totality of patients with a grade 4 (the highest) of volume of adenoids 60.7% of children had no allergy (for association between allergy status and volume of adenoids, $p < 0.001$).

Furthermore, among patients with a grade 1 (the lowest) or 2 of volume of adenoids, respectively, 58.1 and 53.4% of patients had grade 3 (the highest) of obstruction on Friedman scale (Fig. 2), and regarding patients with grade 4, 75% showed the lowest grade on the scale of nasal obstruction ($p < 0.001$). Regarding patients with nose obstruction of grade 3, 46.7% had grade 1 of volume of adenoids and for patients with nose obstruction of grade 1, 56.8% had grade 4 of volume of adenoids. This result is also confirmed by a negative significant correlation ($\rho = -0.41$; $p < 0.001$; Table 2) between nose obstruction and volume of adenoids. A positive correlation (Table 2) was also found between volume of adenoids and FRS ($\rho = 0.19$; $p = 0.006$).

The results from multivariate ordinal regression are shown in Table 3. Age and duration of disease were not significant and were not included in the model. A decrease of probability of higher values on adenoid volume scale was associated with an increase in nose obstruction on Friedman scale (OR [2 versus 1] = 0.13 [95% CI, 0.05–0.31]; OR [3 versus 1] = 0.08 [95% CI, 0.03–0.20; $p < 0.001$] and in patients with allergy compared with patients without allergy OR [yes versus no] = 0.31 [95% CI, 0.15–0.65; $p = 0.002$]). On the contrary, an increase of probability of higher values on adenoidal volume scale was associated with an increase in values on FRS scale (OR = 1.51; $p = 0.005$).

DISCUSSION

The nasal symptoms are very common in the pediatric population. Nasal obstruction during childhood is usually attributed to enlarged adenoids, but other causes must be considered. AR is frequent in children, affecting up to 30% of the general population, and may also cause the open-mouth posture and the so-called “adenoidal facies” commonly attributed to AH.²⁸ A clinically significant septal deviation has been reported in 18% of children reporting nasal obstruction.³ Rare causes of nasal obstruction include choanal atresia, polyps, and tumors, any of which may be missed if a thorough examination is not performed.⁶

Children with AH may usually have AR as well as, *vice versa*, children with AR may commonly have AH. Although this topic is

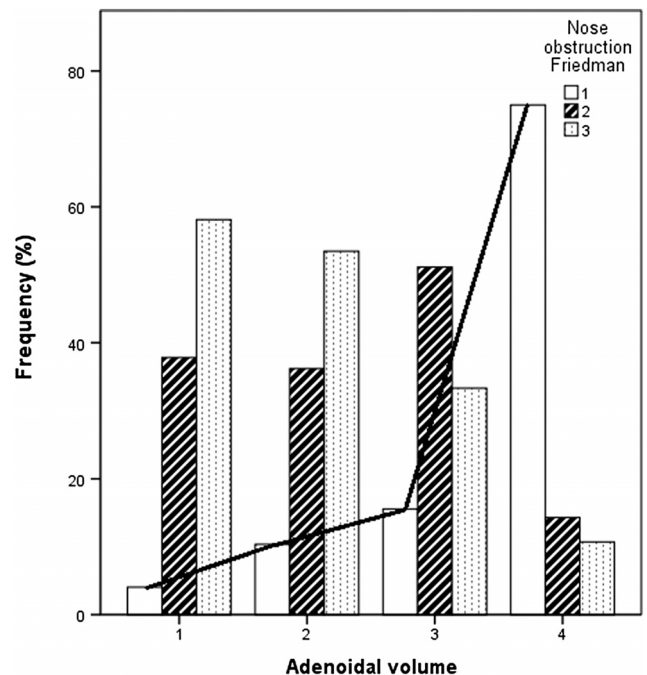


Figure 2. Frequencies of nasal obstruction severity in patients with adenoidal hypertrophy, according to adenoidal volume.

Table 2 Spearman's rank correlation coefficients between ordinal clinical characteristics

		Nasal Obstruction (Friedmann)	VAS
Nasal obstruction (Friedmann)	ρ	—	
	p		
VAS	ρ	0.24	—
	p	0.001	
FRS	ρ	0.12	0.47
	p	0.09	<0.001
Adenoids volume	ρ	-0.41	-0.009
	p	<0.001	0.90

FRS = faces rating scale; VAS = visual analog scale.

Table 3 Results from multivariate ordinal regression

Clinical Characteristics	OR	95% CI	p Value
Nose obstruction (Friedman)			<0.001
1	1.00 (Ref.)	—	
2	0.13	0.05–0.31	
3	0.08	0.03–0.20	
Allergy			0.002
No	1.00 (Ref.)	—	
Yes	0.31	0.15–0.65	

A value of $p < 0.05$ was considered statistically significant.

Ref. = reference category for each variable; OR = odds-ratio.

clinically relevant, few studies investigated the relationships between these two disorders, mainly concerning the possible influence of AR on adenoid enlargement,^{15–22} and a recent review considered the possible relationships between AR and pathogenic factors inducing AH.²⁹ One study from an ENT department found an association between tonsillar hypertrophy and AR.¹⁵ Only 8% of children in 6th grade with tonsillar hypertrophy had AR, whereas AR was apparent in 29.7% of children with tonsillar hypertrophy. One study by McColley and colleagues evaluated 39 children, aged 1–7 years, with habitual snoring; 36% of them were sensitized, a percentage higher than expected for the normal population.¹⁶ These authors suggest an association between snoring and allergy. Huang and Giannoni studied 315 children (aged 1–18 years) with AH and AR and compared them with 315 age-matched controls suffering from AR alone.¹⁷ AH was measured by lateral x-ray craniometry. These authors concluded that sensitivity to mold allergens was recognized to be an important risk factor for AH in children with AR, but the OR was not calculated. Modrzynski and Zawisza conducted a study comparing two separate groups: the study group consisted of 436 children (4–9 years) with AR and/or asthma and/or atopic dermatitis and sensitization to house-dust mites, and the control group consisted of 229 nonatopic coetaneous children.¹⁸ AH was diagnosed by clinical picture and additional diagnostic tests, including at least two of the following methods: posterior rhinoscopy, acoustic rhinometry, lateral radiograph of the rhinopharynx, and fiberoptic examination. The probability of AH was statistically more significant only in children from the study group with AR. Two other studies conducted by Gerber *et al.*¹⁹ and Raphael *et al.*²⁰ reported that allergy was more frequent in children with AH, but the differences observed were fairly insignificant. Sadeghi-Shabestari and colleagues compared 117 children with adenotonsillar hypertrophy, assessed by lateral neck radiography, with 100 children without it.²¹ In children with adenotonsillar hypertrophy, 70.3% of them were sensitized, whereas in the control group only 10% were sensitized. Thus, these authors concluded that allergy

is an important risk factor for adenotonsillar hypertrophy. Nuhoglu and colleagues compared the size of adenoids in 52 children with AR and in 56 children with nonallergic idiopathic rhinitis, calculating the adenoid/rhinopharynx ratio measured on the lateral radiographs.¹² The adenoid/rhinopharynx ratio was very significantly high in the nonallergic patients. These authors suggested that could be a cellular immune defect in allergic children, which causes the enlargement of adenoids. This might be explained with the hypothesis that allergic patients have a Th2 polarization and, consequently, a deficiency in T-helper 1 cell activity and interferon γ production. This issue might be relevant and it will be discussed later.

These studies were performed using radiographic craniometry and nasal endoscopy is considered the most accurate tool to assess the rhinopharynx; therefore, it is the gold standard to evaluate adenoids. The present study was designed to evaluate the relationship between AH and AR in children with nasal obstruction.

This study provided some interesting findings. First, about ¾ of children were allergic, whereas relevant AH, such as grades 3 and 4, was detectable only in 1/3 of them. There was a significant relationship between the subjective perception of nasal obstruction (by VAS and FCR) and the macroscopic evaluation of anterior nasal obstruction, whereas perception by VAS was not related to adenoid volume. These data might mean that the perception of anterior obstruction is more reliable than the posterior one. Furthermore, the volume of adenoids was inversely related with the grade of the anterior nasal obstruction so it could seem that if the nose is closed the adenoids do not enlarge. The same consideration could be hypothesized for allergy: children with AH of grade 4 rarely are allergic. In fact, the multivariate ordinal regression underlines these concepts: severe anterior obstruction and allergy may “protect” (OR = 0.08 and 0.31, respectively) from severe AH. Therefore, the present study confirms Nohoglu's report that showed higher adenoid volumes in nonallergic children.¹² The pathological AH could depend on exaggerate antigenic stimulation, mainly mediated by Th1 response.¹² However, the studies concerning the relationship between allergy and AH are conflicting and, rarely, the adenoid volume was accurately measured. We have to note that the Waldeyer's ring represents the first barrier toward the antigens entering the body.³⁰ Adenoid tissue being deputed to immune response is characterized by the presence of active constitutive immune response, *e.g.*, the number of Toll-like receptors is overexpressed.³¹ concerning the adaptive immune response, T cells resident in adenoids are able to produce Th1-dependent cytokines, mainly interferon γ .³² When an increased function is required to the lymphatic tissue in filtering infectious antigens, AH may occur. Also, adenoids may be reservoirs of pathogenic organisms.³³ In this way, a vicious circle persists because main factors are involved in maintaining chronic hyperstimulation of the immune response, including the overexpression of chitinases able to induce and amplify local inflammation by activating pattern recognition receptors and pathways such as nuclear factor κB .³⁴ Thus, recurrent respiratory infections could cause an increased function of pharyngeal lymphatic tissue, thus inducing AH.

On the other hand, airborne allergens may overstimulate the immune system at the adenoidal level. In fact, it was shown that adenoids are involved in IgE-mediated sensitization with local differentiation of IgE-producing plasma cells constituting a probable source of mucosal B cells for the upper airways.³⁵ Moreover, allergic subjects have a different distribution of mast cells, the main effective cell in allergic inflammation, into tonsillar tissue in comparison with normal subjects so mast cells in the interfollicular area might be promptly activated by direct contact with CD4⁺ T cells.³⁶ Children with AH are characterized by impaired immunologic parameters, persisting also after adenoidectomy for a long time.³⁷ However, the correlations among allergy, recurrent infections, and AH remain obscure. Actually, adenoidal tissue represents a fundamental site for the adaptive immune response, both inducing secretory

immunity and regulating the production of antibodies. As two new subsets of T-helper cells have been discovered, such as Th17 (involved in protecting the host against extracellular pathogens) and Treg (fundamental for inducing and maintaining the immunologic tolerance to foreign and self-antigens), they were evaluated both in allergic and in infectious disorders. In this regard, Sade *et al.* investigated the Th17 and Treg expression in hypertrophied adenoids.³⁸ These authors found a significant negative linear correlation between Th17/Treg ratio and the clinical severity in 20 children undergoing adenoidectomy. This result might have great pathophysiological relevance, because allergic inflammation is characterized by Th17 overexpression³⁹ and defective Treg function.⁴⁰ Therefore, the AH severity could be inversely related to impaired Th17 and/or Treg functioning. In this complex pathway, our findings could add an interesting contribution to better understanding the relationship between allergy and AH. The present study was based on a real-life setting, such as the studied cohort was constituted of children complaining nasal obstruction. They were visited at an ENT office undergoing nasal endoscopy and further evaluated at an allergy office. Thus, starting from nasal obstruction symptoms, a diagnosis was performed: AR, AH, or both. The results would seem to show that large turbinates rarely are associated with large adenoids as well as large adenoids rarely are associated with allergy. A possible interpretation might be that severe anterior nasal obstruction, mainly caused by allergy, affects the passage of allergens able to stimulate adenoid tissue to enlarge. Also, infections may play a more important role in the absence of allergy.

The main limitation of the present study is the absence of immunologic parameters useful to better understand the meaning of the data. Therefore, further immunologic studies should be performed to address this issue. Another issue to be considered could be the evaluation of tonsil hypertrophy and the possible impact of AH on tonsil volume. In this regard, we are conducting a study investigating the possible relationship among tonsil volume, allergy, turbinate hypertrophy, and adenoids. Furthermore, mouth breathing (because of anterior nasal obstruction caused by turbinate hypertrophy or AH) may induce a preferential exposure of the mouth and/or tonsils to allergens and infectious agents. This issue deserves further adequate investigation for better understanding the effects of upper airway obstruction on tonsils.

In conclusion, this real-life study shows that large adenoids may be associated with absence of allergy, whereas large turbinates may be associated with small adenoids. This finding may be helpful in the clinical management of a child with nasal obstruction because it shows that a detailed evaluation of the nose and the rhinopharynx is mandatory in each child with this complaint and it should be performed by nasal endoscopy. Consequently, the treatment should be geared toward the specific findings in that individual. The present study may have an impact on clinical decision making and care as the ENT specialist should always consider nasal endoscopy in children with nasal obstruction. In addition, the specialist should not be surprised to find a discrepancy between large adenoids and small turbinates or *vice versa*. Another important issue is that nasal obstruction is frequently considered caused by AH in the toddler, even if allergic. This aspect is clinically relevant because the allergic child does not need antibiotic treatment and adenoidectomy is obviously useless. Therefore, this study may suggest that nasal obstruction could not depend on adenoidal obstruction, as believed by some physicians, mainly concerning pediatricians, who do not have the possibility of investigating the nasal cavity.

REFERENCES

1. Rob MI, Westbrook JI, Taylor R, and Rushworth R. Increased rates of ENT surgery among young children: Have clinical guidelines made a difference? *J Pediatr Child Health* 40:627–632, 2004.
2. Van Den Akker EH, Hoes AW, Burton MJ, and Schilder AG. Large international differences in (adenoid)tonsillectomy rates. *Clin Otolaryngol Allied Sci* 29:161–164, 2004.
3. Wang DY, Bernheim N, Kaufman L, and Clement P. Assessment of adenoidal size in children by fiberoptic examination. *Clin Otol* 22:172–177, 1999.
4. Lertsburapa K, Schroeder JW, and Sullivan C. Assessment of adenoidal size: A comparison of lateral radiographic measurements, radiologist assessment, and nasal endoscopy. *Int J Pediatr Otolaryngol* 74:1281–1285, 2010.
5. Caylakli F, Hizal E, Yilmaz I, and Yilmazer C. Correlation between adenoid-nasopharynx ratio and endoscopic examination of adenoidal hypertrophy: A blind prospective clinical study. *Int J Pediatr Otorhinolaryngol* 73:1532–1535, 2009.
6. Kubba H, and Bingham BJ. Endoscopy in the assessment of children with nasal obstruction. *J Laryngol* 115:380–384, 2001.
7. Kubba H, and Bingham BJ. Can nasal endoscopy be used to predict residual symptoms after adenoidectomy for nasal obstruction? *Int J Pediatr Otorhinolaryngol* 58:223–228, 2001.
8. Major MP, Flores-Mir C, and Major PW. Assessment of lateral cephalometric diagnosis of adenoidal hypertrophy and posterior upper airway obstruction: A systematic review. *Am J Orthod Dentofacial Orthop* 130:700–708, 2006.
9. Kindermann CA, Roithmann R, and Lubianca Neto JF. Sensitivity and specificity of nasal flexible fiberoptic endoscopy in the diagnosis of adenoid hypertrophy in children. *Int J Pediatr Otorhinolaryngol* 72:63–67, 2008.
10. Bitar MA, Birjawi G, Youssef M, and Fuleihan N. How frequent is adenoid obstruction? Impact on the diagnostic approach. *Pediatr Int* 51:478–483, 2009.
11. Yilmaz I, Caylakli F, Yilmazer C, et al. Correlation of diagnostic systems with adenoidal tissue volume: a blind prospective study. *Int J Pediatr Otorhinolaryngol* 72:1235–1240, 2008.
12. Nuhoglu C, Nuhoglu Y, Bankaoglu M, and Ceran O. A retrospective analysis of adenoidal size in children with allergic rhinitis and non-allergic idiopathic rhinitis. *Asian Pac J Allergy Immunol* 28:136–140, 2010.
13. Ciprandi G, Tosca MA, Signori A, and Ameli F. Comparison between symptoms and endoscopy in children with nasal obstruction. *Int J Pediatr Otorhinolaryngol* 74:1405–1408, 2010.
14. Lack G. Pediatric allergic rhinitis and comorbid disorders. *J Allergy Clin Immunol* 108:S9–S15, 2001.
15. Yumoto E, Kozawa T, and Yanagihara N. Influence of tonsillar hypertrophy to physical growth and diseases of the nose and ear in school-age children. *Nippon Jiblinkoka Gakkai Kaiho* 94:534–540, 1991.
16. McColley SA, Carroll JL, Curtis S, et al. High prevalence of allergic sensitization in children with habitual snoring and obstructive sleep apnea. *Chest* 111:170–173, 1997.
17. Huang SW, and Giannoni C. The risk of adenoid hypertrophy in children with allergic rhinitis. *Ann Allergy Asthma Immunol* 87:350–355, 2001.
18. Modrzynski M, and Zawisza E. An analysis of the incidence of adenoid hypertrophy in allergic children. *Int J Pediatr Otorhinolaryngol* 71:713–719, 2007.
19. Gerber VK. The importance of allergy in hypertrophy of the nasopharyngeal tonsil. *Vestn Otolaryngol* 28:52–56, 1966.
20. Raphael G, and Kaliner M. Allergy and the pharyngeal lymphoid tissues. *Otolaryngol Clin North Am* 20:295–304, 1987.
21. Sadeghi-Shabestari M, Jabbari Moghaddam Y, and Ghaharri H. Is there any correlation between allergy and adenotonsillar tissue hypertrophy? *Int J Pediatr Otorhinolaryngol* 75:589–591, 2011.
22. Zicari AM, Magliulo G, Rugiano A, et al. The role of rhinomanometry after nasal decongestant test in the assessment of adenoid hypertrophy in children. *Int J Pediatr Otorhinolaryngol* 76:352–356, 2012.
23. Ameli F, Castelnovo P, Pagella F, et al. Nasal endoscopy in asthmatic children: Clinical role in the diagnosis of rhinosinusitis. *Rhinology* 42:15–18, 2004.
24. Lang J. *Clinical Anatomy of the Nose, Nasal Cavity and Paranasal Sinuses*. New York, NY: Thieme, Verlag Stuttgart, The Adenoids, J Lang, 125–143, 1989.
25. Friedman M, Tanyeri H, Lim J, et al. A safe, alternative technique for inferior turbinate reduction. *Laryngoscope* 109:1834–1837, 1999.
26. Parikh SR, Coronel M, Lee JJ, and Brown SM. Validation of a new grading system for endoscopic examination of adenoid hypertrophy. *Otolaryngol Head Neck Surg* 135:684–687, 2006.

27. Dreborg S (Ed). EAACI Subcommittee on Skin Tests. Skin tests used in type I allergy testing. *Allergy* 44(suppl 10):22–31, 1989.
28. Jones NS. Current concepts in the management of paediatric rhinosinusitis. *J Laryngol Otol* 113:1–9, 1999.
29. Marseglia GL, Poddighe D, Caimmi D, and Ciprandi G. Role of adenoids and adenoiditis in children with allergy and otitis media. *Curr Allergy Asthma Rep* 9:460–464, 2009.
30. Helling P, Jorissen M, and Ceuppens JL. The Waldeyer's ring. *Acta Otolaryngol Belg* 54:237–241, 2000.
31. Claeys S, de Belder T, Holtappels G, et al. Human β -defensin and toll-like receptors in the upper airway. *Allergy* 58:748–753, 2003.
32. Ivarsson M, and Lundin BS. Cytokines produced by T cells in adenoid surface secretion are mainly downregulatory or of Th1 type. *Acta Otolaryngol* 126:186–190, 2006.
33. Gates GA. Adenoidectomy for otitis media with effusion. *Ann Otol Rhinol Laryngol Suppl* 163:54–58, 1994.
34. Heo KW, Hur DY, Park SK, et al. Expression of chitinases in hypertrophied adenoids in children. *Otolaryngol Head-Neck Surg* 145:660–665, 2011.
35. Papatziamos G, Van Hage-Hamsten M, Lundahl J, and Hemlin C. IgE-positive plasma cells are present in adenoids of atopic children. *Acta Otolaryngol* 126:180–185, 2006.
36. Yokoi H, Okayama Y, Niyonsaba F, et al. Comparison of human tonsillar mast cell localization and ultrastructural observations between IgE-mediated allergic and nonallergic donors. *Allergy Asthma Proc* 27:415–421, 2006.
37. Zielnik-Jurkiewicz B, and Jurkiewicz D. Implication of immunological abnormalities after adenotonsillectomy. *Int J Pediatr Otolaryngol* 64:127–132, 2002.
38. Sade K, Fishman G, Kivity S, et al. Expression of Th17 and Treg lymphocyte subsets in hypertrophied adenoids of children and its clinical significance. *Immunol Invest* 40:657–666, 2011.
39. Ciprandi G, Fenoglio D, De Amici M, et al. Serum IL-17 in allergic rhinitis. *JACI* 122:650–651.e2, 2008.
40. Ciprandi G, Fenoglio D, Cirillo I, et al. Sublingual HDM-specific immunotherapy induces IL-10 production: Preliminary report. *Ann Allergy Asthma Immunol* 95:38–44, 2005. □

Copyright of American Journal of Rhinology & Allergy is the property of OceanSide Publications Inc. and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.