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Medical Treatment for Rhinosinusitis Associated With Adenoidal Hypertrophy in Children: An Evaluation of Clinical Response and Changes on Magnetic Resonance Imaging

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Objectives: The association between adenoidal hypertrophy and rhinosinusitis with upper airway inflammation is increasingly recognized; however, no study has used magnetic resonance imaging (MRI) to assess the changes in adenoid size after medical treatment of rhinosinusitis.

Methods: Thirteen children referred to a tertiary allergy clinic with symptoms of rhinosinusitis received medical treatment over a 4-month period. All underwent MRI before and after treatment. The medical treatment regimen comprised a short course of oral antibiotics and oral steroids and a longer course of oral antihistamines and intranasal steroids.

Results: The pretreatment MRI demonstrated enlarged adenoids and rhinosinusitis in all 13 children, with evidence of extensive rhinosinusitis in 9 of the 13. The treatment resulted in an improvement in overall symptom score; the most significant improvement was seen in mouth breathing. The posttreatment MRI showed a statistically significant reduction in adenoid size and adenoid/nasopharynx ratio, which was associated with a significant decrease in sinus involvement on MRI.

Conclusions: There is a high association between adenoidal hypertrophy and rhinosinusitis in the context of an allergy clinic. Magnetic resonance imaging can document the changes in adenoid size associated with resolution of rhinosinusitis. Further studies are necessary to validate these pilot data and further assess the effects of medical treatment and the role of MRI in adenoidal hypertrophy.

Key Words: adenoidal hypertrophy, amoxicillin–clavulanate potassium, loratadine, magnetic resonance imaging, sinusitis, steroids.

INTRODUCTION

Adenoidectomy is among the most common operations performed in children worldwide. In 1999 in the United Kingdom, a total of 60,000 patients underwent tonsillectomy with or without adenoidectomy, and another 9,000 underwent adenoidectomy alone.[1] In addition to this surgical workload, physician consultations for the associated symptoms of nasal obstruction, snoring, and sleep-disordered breathing account for a significant part of the total visits to otolaryngology and allergy specialists. These symptoms can impair a child's quality of life and may have unfavorable developmental effects that predispose the child to sleep-related breathing abnormalities later on.[2] Airway obstruction related to adenotonsillar hypertrophy (ATH) can be associated with long-term consequences such as failure to thrive and sleep disturbance leading to inability to concentrate, daytime somnolence, and low results on psychometric tests; in extreme cases, obstructive sleep apnea (OSA) may lead to pulmonary hypertension and right-sided heart failure.[3]

Although the extent of the problem is well understood, the same cannot be said concerning its cause, diagnosis, and optimal treatment. Measurements from lateral cervical radiographs in patients in whom adenoidal hypertrophy (AH) is suspected correlate poorly with clinical symptoms,[4] and posterior indirect (mirror) examination of the postnasal space and flexible nasendoscopy are not always practical in very young children, and as a result are not performed routinely in the United Kingdom. Adenoidal hypertrophy has been shown to be associated with otitis media with effusion,[5] rhinosinusitis,[6] *Haemophilus influenzae* infection,[7] allergy,[8] and irritant exposure.[9] Sinusitis is not routinely evaluated in children with ATH, partly because of radiation concerns regarding computed tomography in the pediatric population.[10]

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Magnetic resonance imaging (MRI) provides an excellent imaging method for the assessment of AH, airway caliber, and paranasal sinus involvement because of its multiplanar capability, excellent soft tissue contrast, and lack of ionizing radiation, which allows sequential scans to be performed to monitor the efficacy of treatment. We have used an MRI protocol to visualize the extent of AH and upper airway changes in outpatients. In this study we demonstrate radiographically the association between AH and rhinosinusitis and evaluate medical treatment of these conditions in terms of both symptomatic improvement and MRI response.

MATERIALS AND METHODS

Patients. This case series retrospective study included 13 pediatric patients (mean age, 8.4 years; range, 3 to 11 years) who were sequentially referred to a specialist-run pediatric allergy clinic over a period of 8 months to determine whether allergy contributed to their symptoms of rhinosinusitis (more than 6 weeks' duration of mouth breathing with or without snoring, mucopurulent rhinorrhea, hyponasal speech, cough, and reduced appetite). All children received medical treatment as described below. We performed MRI examinations on clinical grounds to confirm diagnosis and to monitor response to treatment.

Clinical Signs and Symptoms. All children were initially assessed with history and physical examination; degree of mouth breathing, snoring, rhinorrhea, and cough were recorded on a scale of 0 to 5 at baseline and after 3 months of treatment by the same clinician. A total clinical score (range, 0 to 20) was calculated by adding the individual scores, and any additional symptoms such as reduced appetite were recorded separately. Clinical examination included anterior rhinoscopy, and we also performed skin prick testing to common aeroallergens (*Alternaria*, *Aspergillus*, *Cladosporium*, *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, grass pollen, tree pollen, cat, and dog).

Magnetic Resonance Imaging. All children underwent examinations on an open 0.5-T magnet (GE Medical Systems, London, England). A protocol of sagittal T1 (6-mm slice thickness with 2-mm interslice gap), axial T1 (4-mm slice thickness with 2-mm interslice gap), and coronal T1 (4-mm slice thickness with 2-mm interslice gap) images was used, in which the images could be obtained in a total examination time of 15 to 20 minutes without the need for sedation. On axial images, at the level of maximal adenoid dimensions, we assessed the anteroposterior and transverse adenoid diameters of the adenoid pad, the anteroposterior nasopharynx diameter, and the adenoid/nasopharynx (A/N) ratio (anteroposterior adenoid measurement divided by nasopharyngeal measurement).

On a midsagittal image, measurement of the adenoidal pad was performed in two planes: a short axis from the point of maximal convexity along a line perpendicular to the basopharyngeal fascia, and a long axis measurement along a line at a 90° angle to the short axis. The nasopharyngeal airway was measured from the superior mucosal surface of the posterior-superior margin of the hard palate to the basisphenoid synchondrosis. The A/N ratio was calculated from the adenoid long axis divided by the nasopharyngeal measurement (Fig 1).

Paranasal sinus involvement was graded on a 4-point scale for each of the ethmoid, maxillary, sphenoid, and frontal sinuses (when present) and was scored according to severity (0, clear; 1, less than one-third opacification; 2, one-third to two-thirds opacification; 3, greater than two-thirds opacification or air-fluid level). A summated sinusitis score was produced by multiplying the number of sinuses involved by their degree of involvement and then dividing the product by the number of sinuses present, ranging from 0 (no sinus involvement) to 3 (severe pansinusitis). The scoring system was weighted to record significant sinusitis. Thus, a child with complete bilateral maxillary sinus opacification (and clear ethmoid and sphenoid sinuses) received a score of $(3 \times 2)/6 = 1$ out of a maximum of 3. Nasal turbinate mucosal thickening was also graded on a 4-point scale (normal/minimal, mild, moderate, and severe). The MRI assessment was repeated after medical treatment, at a mean interval of 82 days (range, 42 to 169 days). Sagittal and axial images were obtained in all 13 children before and after treatment. In 10 children, coronal images were also available. The MRI scans were scored by consensus between two radiologists (C.O. and K.T.) and an otolaryngologist (C.G.). The assessors were blind to the symptom scores at the time of MRI assessment and to whether the MRI was performed at baseline or after treatment.

Treatment. A protocol of 15 days of treatment with amoxicillin–trihydrate clavulanate potassium (48 and 7 mg/kg per day, respectively), 5 days of oral prednisolone sodium phosphate (2 mg/kg per day), and 3 months of mometasone furoate nasal spray (50 μg per nostril per day) and oral loratadine (10 mg/d) was used.

Statistical Analysis. All variables for which the Kolmogorov-Smirnov test proved normality (MRI measurements) were expressed as mean ± SD, whereas ordinal, non-normally distributed variables (symp-

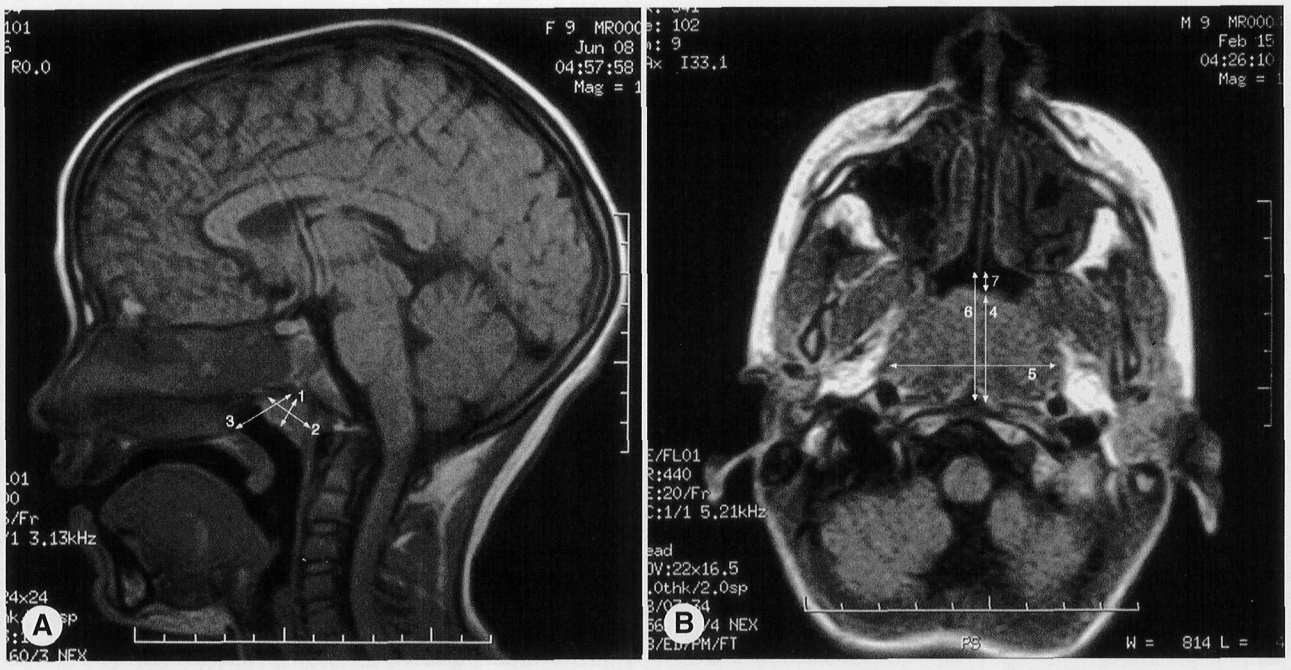

Fig 1. Data acquisition. **A)** Midsagittal T1 image (6-mm slice thickness, 2-mm spacing). 1 — maximum adenoid dimension, from point of maximum convexity along line perpendicular to basipharyngeal fascia; 2 — maximum longitudinal adenoid dimension, perpendicular to 1; 3 — nasopharyngeal airway, from superior mucosal surface of posterior-superior margin of hard palate to basisphenoid synchondrosis. Adenoid/nasopharynx ratio = 1/3. **B)** Axial T1 image (4-mm slice thickness, 2-mm spacing) at point of maximum adenoid dimension. 4 — maximum anteroposterior adenoid dimension; 5 — maximum transverse adenoid dimension; 6 — nasopharynx, from posterior margin of nasal septum to posterior margin of adenoids; 7 — anteroposterior dimension of airway, from posterior margin of nasal septum to anterior margin of adenoids.

tom scores) were described with the use of medians and ranges. Subsequent comparisons were performed with a paired *t*-test for the normally distributed variables, and Wilcoxon signed rank tests were used for the remaining variables. Two-tailed tests with a significance level of .05 were used in all comparisons. SPSS 8.0 statistical software was used for the analysis.

RESULTS

BEFORE TREATMENT

Clinical Symptoms and Investigations. All 13 children presented with mouth breathing (median score, 4; range, 2 to 5). In 11 of the 13 children, it was associated with mucopurulent rhinorrhea at baseline (median score, 4; range, 0 to 5). Cough and snoring were present in various degrees in 11 and 7 children, respectively (median scores, 3). In 5 children, reduced appetite was a prominent symptom. All 13 children had evidence of rhinitis on anterior rhinoscopy, and 5 of the 13 (38%) had positive skin prick tests to inhalant allergens.

Magnetic Resonance Imaging. The mean adenoid size, as measured on the midsagittal plane, was 12 mm (short axis) by 23 mm (long axis). The airway diameter was reduced at 11 mm, and the A/N ratio was 52%. On the axial plane, the mean adenoid diameter (anteroposterior axis) was 19 mm, whereas on the same axis the airway was only 4 mm, producing an A/N ratio of 77%.

Sinuses. Extensive sinus involvement was documented: all 13 children in our study group had some degree of mucosal thickening affecting the paranasal sinuses. Nine of 13 children (69%) had involvement of all paranasal sinuses (greater than one-third sinus opacification in all sinuses) or at least 2 completely opacified sinuses (overall sinusitis score of 1 or higher). The mean score for all patients was 1.49. Inferior turbinate swelling was also noted in 12 of the 13 scans (mean score, 1.53).

AFTER TREATMENT

All 13 children completed the treatment, and all were included in the analysis. No complications were reported. Compliance with treatment, as assessed by parental reports, was good.

Clinical Symptoms. In 12 of the 13 children, treatment resulted in significant overall clinical improvement as assessed an average of 3 months later (Fig 2). Specifically, mouth breathing improved in 12 children (from a median of 4 to a median of 2; $p = .002$), and nasal symptoms improved in 10 of 11 children (from a median of 4 to a median of 1; $p = .005$). Additionally, cough was reduced in 10 of 11 children (p

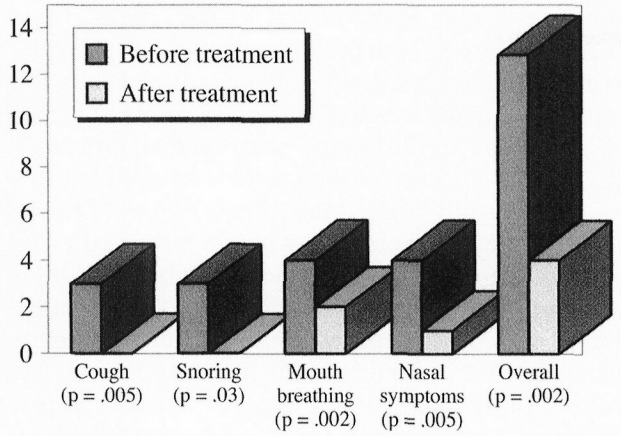

Fig 2. Clinical examination scores (medians) at baseline and after treatment. Individual symptom scores range from 0 to 5; overall symptom scores range from 0 to 20.

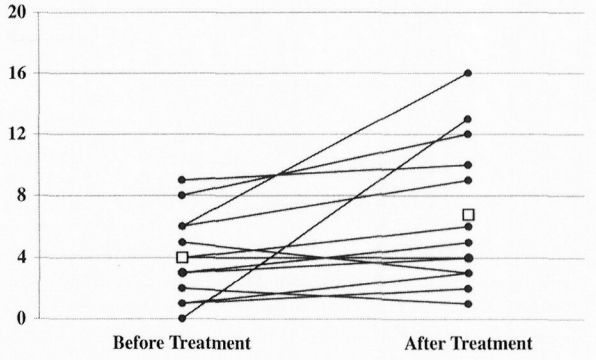

Fig 3. Nasopharyngeal airway. Anteroposterior axial diameter on magnetic resonance imaging before and after treatment. Rectangles represent mean values.

= .005), and snoring improved in 6 of 7 children (p = .034). Appetite improved in all 5 affected children (p = .030). The overall median clinical score was reduced from 13 of 20 to 4 of 20 after treatment (p = .002).

Magnetic Resonance Imaging. After medical treatment, there was a statistically significant reduction in adenoid size, both in absolute terms and in the A/N ratio, with a consequent increase in the airway (Fig 3). On the midsagittal plane, the mean adenoid short-axis diameter was reduced by 2 mm (95% confidence interval [CI], 0.7 to 3.2 mm; p = .005), with a concomitant increase in airway diameter of 1.9 mm (95% CI, 0.02 to 3.82 mm; p = .048). The mean long-axis adenoid diameter was reduced by 3.2 mm (95% CI, 0.8 to 5.5 mm; p = .012), and the A/N ratio was reduced by 8% (95% CI, 3% to 12%; p = .002). Similar results were observed on axial images, with significant reduction of the anteroposterior adenoid diameter by 3.2 mm (95% CI, 0.2 to 8.7 mm; p = .009) and a corresponding increase in airway diameter of 2.76 mm (95% CI, 0.21 to 5.32 mm; p = .036; see Table).

Sinuses. Eight of the 13 children showed an improvement of more than 0.5 in their sinusitis scores after the end of the treatment regimen. There was a statistically significant improvement in the mean sinus score of our study group, from 1.48 before treatment to 0.69 after therapy (p = .006; Fig 4). This improvement in sinus disease was accompanied by a reduction in inferior turbinate nasal mucosal thickening, although the change was not statistically significant (p = .055).

DISCUSSION

There is increasing evidence that nasal inflammation is part of a continuum of upper and lower airway inflammation.[11,12] The nasal mucosa, the lining of the sinuses, and the nasopharyngeal mucosa are contiguous, and their local lymphatic drainage is to the lymphoid tissue of Waldeyer's ring. Thus, chronic inflammation caused by infection, irritation (indoor and outdoor pollutants), or allergy leads to enlarged adenoids. The enlargement of adenoids is mainly the result of lymphoid proliferation.[13] Lymphoid hyperplasia of the adenoids and tonsils has been shown to be directly proportional to the aerobic bacterial load and the absolute number of B and T cells,[13] with *H*

ADENOID AND AIRWAY MEASUREMENTS ON MAGNETIC RESONANCE IMAGING BEFORE AND AFTER TREATMENT

	Before Treatment	*After Treatment*	*p*
Midsagittal plane			
Adenoid (short axis)	12.30 ± 2.59	10.30 ± 2.49	.005*
Adenoid (long axis)	22.93 ± 5.69	19.69 ± 5.54	.012*
Airway diameter	11.23 ± 3.23	13.15 ± 3.16	.048*
Adenoid/nasopharynx ratio	0.52 ± 1.12	0.44 ± 1.11	.002*
Axial plane			
Adenoid (anteroposterior axis)	18.76 ± 5.00	15.5 ± 6.63	.041*
Adenoid (transverse axis)	37.30 ± 6.85	33.00 ± 5.40	.091
Airway diameter	4.00 ± 2.73	6.76 ± 4.74	.036*
Adenoid/nasopharynx ratio	0.79 ± 0.12	0.65 ± 0.26	.130

Data are mean ± SD in millimeters.
*p < .05.

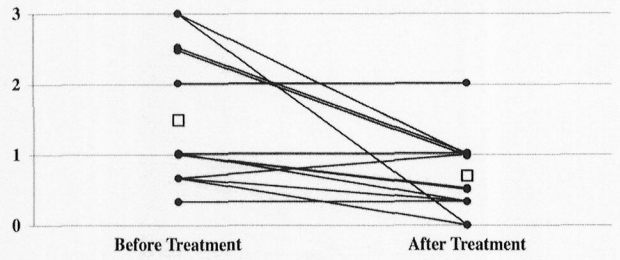

Fig 4. Sinusitis scores on magnetic resonance imaging before and after treatment. Mild — <1; moderate — 1 to 2; severe — >2. Rectangles represent mean values.

influenzae and other β-lactamase–producing bacteria playing a key role.[7,14] The prevalence of AH is increased in children with allergy,[15,16] and conversely, as many as 36% of patients with ATH and OSA have been found to have positive radioallergosorbent test results, with the presence of atopy being an important independent predictor of the severity of OSA.[17] In our study, 38% of children with AH had positive skin tests for inhalant allergens.

Enlarged and/or infected adenoids have been associated with chronic rhinitis and sinusitis in children,[6] and it has been suggested that they may perpetuate upper airway inflammation. Adenoids can serve as a reservoir for bacteria and thus cause recurrent sinus infections,[18] and mucociliary clearance appears to be impaired in children with adenoiditis.[19] Conversely, chronic rhinosinusitis, with the resultant upper airway inflammation and postnasal drip, can cause lymphoid hyperplasia and AH. In this context, removal of the adenoids has been advocated as a treatment option for chronic sinusitis.[20]

The recognition that upper airway inflammation underpins AH raises the question of whether anti-inflammatory treatment has a role in its management. Surgical adenoidectomy relieves the mechanical obstruction caused by AH, but ignores the predisposing pathophysiologic condition. Medical anti-inflammatory and antimicrobial treatment could concurrently treat both AH and associated upper airway inflammation. Various treatment regimens based on this approach have been attempted.

A double-blind, placebo-controlled, randomized trial in children with ATH demonstrated that a 30-day course with amoxicillin-clavulanate can reduce the need for surgery. This beneficial effect persisted for 24 months after the end of treatment; 16% of patients avoided surgery.[21]

Steroids have a known lympholytic and anti-inflammatory effect, and they have been used for many years for tonsillar hypertrophy causing airway obstruction in infectious mononucleosis.[22] Three previous studies assessed their efficacy in AH. In a randomized, double-blind study,[23] a 5-day course of oral prednisolone was used in children with OSA and ATH. Although steroids were not effective in reducing the apnea-hypopnea index (AHI) as assessed at the end of treatment, the A/N ratio decreased significantly in 7 of 9 subjects. Another double-blind, placebo-controlled study of a 6-week course of nasal fluticasone propionate in children with OSA showed a significant improvement in almost all children and a substantial decrease in AHI.[24] In another double-blind, placebo-controlled crossover study[25] in children with AH without OSA, an 8-week course of nasal beclomethazone dipropionate resulted in an improvement in symptoms by 82%, accompanied by a persistent (24 weeks) and highly significant reduction in adenoid size.

In our clinic, a 5-day course of oral prednisolone was used because we believe it represents the optimal benefit/risk ratio. The effects of the short-term systemic administration of prednisolone were consolidated with a 3-month course of nasal mometasone, a nasal steroid that has been shown to be relatively safe for long-term management of allergic rhinitis in children.[26] The 3-month course of loratadine was used to address the presence of allergy because of the known high incidence of atopy in children with adenoiditis. Amoxicillin–clavulanate potassium was chosen because it has been shown to be effective for the treatment of ATH and because its wide coverage that includes β-lactamase–producing *H influenzae* and anaerobes corresponds to the microbial profile of the bacteria associated with lymphoid hypertrophy in chronic adenoiditis. All of these treatments additionally have a role in the management of chronic rhinosinusitis,[27] which was present in all 13 children.

We used MRI on the basis of its excellent soft tissue contrast resolution, its ability for multiplanar imaging, and the lack of radiation, which enables multiple scans to be done on the same patient. A previous study[28] showed that MRI measurements of adenoids correlate with the AHI in children with OSA. Two previous studies of healthy children have shown that adenoids can be clearly identified and measured with MRI and that their size correlates with AH symptoms.[29,30] Interestingly, in both of these studies the maximal adenoid size appeared between the ages of 7 and 10 years,[29,30] but in one study no correlation was noted between adenoid size and previous adenoidectomy.[29]

In our study, 13 children with AH and rhinosinusitis were assessed. The MRI scans confirmed the coexistence of rhinosinusitis with AH; they demonstrated evidence of mucosal disease in all 13 children and significant sinusitis in 9 of the 13 (69%). Adenoidal

hypertrophy with a concomitant decrease in airway caliber was confirmed in all children. After medical treatment, the total symptom scores improved in 12 of the 13 children; the most consistent improvement was seen in mouth breathing, cough, and nasal symptoms, resulting in an overall improvement of 69%. Radiologically, in 11 of the 13 children a significant decrease in adenoid size was documented with a corresponding increase in nasopharyngeal airway caliber. This was accompanied by a significant improvement in sinusitis scores and a decrease in inferior turbinate swelling. Although the absolute decrease in adenoid diameter may appear small — on the order of a few millimeters — it must be noted that these measurements were 1-dimensional, whereas adenoids are 3-dimensional structures; thus, an equal decrease in each dimension would diminish their volume by a power of 3. According to Poiseuille's law, airflow resistance is inversely proportional to the fourth power of the radius; thus, a mean increase in airway diameter by 69% in 1 dimension (from 4 mm to 6.79 mm, as recorded on the axial plane) could be expected to decrease resistance by a factor of 8. The clinical effect of this may be even greater if the flow pattern changes from turbulent to laminar.

There are several limitations to this study, the most obvious of which is the retrospective nature of the assessments and the lack of a control population. However, we believe that serial MRI measurements provide some objective data and render unlikely the possibility of a placebo effect. Furthermore, it is unlikely that the children would have shown spontaneous resolution over such a short period of time, given the previous severity and chronic nature of their symptoms. Second, compliance was not formally assessed in any way other than the parents' responses. Third, follow-up was only done for 3 months, and the long-term outcome of such an approach remains to be determined. Fourth, the association with atopy may be skewed by the referral pattern to a pediatric allergy clinic, although previous studies have shown similar associations with atopy.

CONCLUSIONS

We have shown a high degree of association between AH and chronic rhinosinusitis in a tertiary allergy clinic; objective evidence of allergic disease was present in 38% of subjects. Prolonged treatment with a combined anti-inflammatory and antimicrobial medical regimen resulted in marked clinical improvement, accompanied by improvement in MRI findings, notably, a reduction in adenoid size, an increase in airway caliber, and a reduction in paranasal sinus involvement. Keeping in mind the limitations of our study as described above, we understand that these are only pilot data and that further studies are necessary in order to validate our results before they can be used clinically. Nevertheless, MRI was shown to be a useful method of assessing children with symptoms suggestive of AH and can also document associated evidence of upper airway inflammation (sinusitis and nasal mucosal thickening). Although it is not proposed that medical therapy should replace surgery, it provides a promising alternative in children for whom surgery is unsuitable or in those who are reluctant to undergo surgery. A larger prospective, randomized, controlled trial will be needed to further investigate the effectiveness of medical treatment. It may be possible to identify clinical or radiologic characteristics that could predict a favorable response to medical treatment to assist in the selection of the most appropriate patients for this therapeutic approach. Finally, there is some evidence that the cost of MRI of the paranasal sinuses can compare favorably with that of computed tomographic scanning,[31] but this is dependent on local conditions, and further work regarding the cost-effectiveness of MRI in this clinical context will be required.

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