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The Relation of Allergy to Adenoid Hypertrophy and Otitis Media with Effusion: A Cross-sectional Study

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ABSTRACT

The exact mechanisms of Adenoid hypertrophy (AHT) pathogenesis and otitis media with effusion (OME) are unclear but there is increasing evidence that allergies may play a role. We aimed to investigate the prevalence of atopy and the effect of anti-allergic drugs in patients with AHT and OME.

In a non-randomized, prospective cross-sectional study, 122 patients younger than 18 years of age with AHT or OME were included. Atopic patients based on clinical symptoms of allergic disorders and/or elevated levels of total serum immunoglobulin E (IgE) were referred to allergists and tested for allergen sensitization by skin prick test (SPT). Atopic patients were treated with nasal corticosteroids and antihistamines. Response to treatment was evaluated by comparing symptoms score before and after the treatment.

In this study 122 patients were evaluated, 116 of them had AHT and 30 patients had OME. The mean age of participants was 6.7±2.4 years old and 68 of them (55.7%) were male. Allergic symptoms were observed in 38 patients with AHT (32.7%) and nine patients with OME (30%). Among the total cases, 34 patients (28%) were considered atopic. SPT was performed on 25 (73%) cases of atopic patients, with 11 (44 %) positive results. The mean symptom score of AHT and OME decreased significantly after treatment respectively, (p=0.001, p=0.007).

According to this study, atopy was relatively common in patients with AHT and OME. Treatment with nasal corticosteroid and antihistamines were effective in these patients.

Keywords: Adenoids; Allergy; Child; Hypertrophy; Otitis media with effusion

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INTRODUCTION

Adenoid hypertrophy (AHT) and otitis media with effusion (OME) are common otolaryngologic disorders

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which affect respectively 19% to 70% and 4.5% to 9.86% of children and adolescent.[1-3] AHT can cause symptoms and complications due to airway obstruction.[1,4] OME can cause symptoms and complications like otalgia and conductive hearing loss.[3,4] By understanding the pathogenesis of AHT and OME, it would be possible to choose alternative nonsurgical treatments and avoid surgical complications. AHT and OME pathogenesis seems to be multifactorial which includes upper respiratory infections, passive smoking, feeding habits, and allergy. The exact mechanism by which allergies cause AHT and OME are not clear. Adenoid hypertrophy and Eustachian tube dysfunction are probably induced by chronic inflammation due to contact with allergens.[5-11] This survey aimed to evaluate the relationship between AHT, OME, and allergy in preoperational patients. The secondary goal of this study was to investigate the response of atopic patients to anti-allergic treatment with nasal corticosteroid and antihistamine.

MATERIALS AND METHODS

In a non-randomized, prospective, cross-sectional study, 122 children less than 18 years old who were waiting for adenoidectomy due to AHT and ventilation tube placement due to OME were enrolled in. The criteria for AHT were chronic nasal obstruction symptoms for at least six months and an obstructed nasopharyngeal airway by adenoid tissue on lateral neck x-ray (more than 0.75 adenoidal-nasopharyngeal ratios).[12] The criteria for OME were: middle ear effusion persisting for at least three months documented by otoscopy examination which was unresponsive to treatment with appropriate antibiotics, conductive hearing loss in audiometry, and middle ear pressure less than -150 mm H2O in tympanometry.[13] Patients were excluded from the study if they had an anatomical deformity in head and neck (i.e., cleft palate), upper airway infections in the last 2 weeks, had taken nasal or systemic steroid in the previous 2 months, had undergone adenoidectomy previously, systemic disease and a known case of immune deficiency and Down syndrome.

The study was undertaken in an academic tertiary care center, otorhinolaryngology department, Shahid Sadoughi hospital, Yazd, Iran between September 2007 and June 2008. The institutional ethics committee approved the study (approved number: 17/1/118115). Informed consent was obtained from the patients and their parents for participation. At study enrollment, patients' demographic, medical history, and symptom characteristics were collected. The symptom questionnaire consisted of a parental assessment of their child's symptoms. Adenoid hypertrophy Symptoms (mouth breathing, nasal obstruction, snoring and apnea) were scored as; 0=absent/none, 1=periodic/sometimes, 2=often/most of the time 3=continuous/all of the time.[14]

The OME symptoms (ear pain, ear fullness or discomfort, vertigo or imbalance, and hearing impairment) were scored as 0=No problem, 1=Hardly a problem, 2=Moderate problem, and 3=Extreme problem.[13] One milliliter of clotted blood was drawn from all patients to evaluate the total immunoglobulin E (IgE) antibody level.

After data collection from patients, patients who were atopic according to their clinical history (including atopic dermatitis, allergic rhinitis, and asthma) and paraclinical data (total IgE serum level more than normal level proportional to their age) were referred to an allergist. Referred patients were evaluated for allergen sensitization by SPT. SPTs were performed to common food and aeroallergens in the study region with commercial solutions (Stallergenes, Antony, France). All tests were performed with histamine chloride as positive and 0.9% sodium chloride as the negative control. A wheal diameter of 3 mm more than the negative control was considered as a positive answer. Atopic patients undertook nasal corticosteroid (fluticasone propionate, 50 mcg/spray) one spray per nostril once daily every morning, and a second-generation antihistamine (Loratadine or Cetirizine) orally at bedtime. The efficacy of treatment was assessed by comparing the mean symptom score of AHT and OME before and after treatment.

Statistical Analysis

The data were analyzed using SPSS software version 18 (SPSS Inc., Chicago, IL, USA). Baseline characteristic of all patients was analyzed using the t-test. The primary outcome variable was the adenoid hypertrophy and the OME symptoms score. Baseline and after treatment scores of the two groups were compared using the wilcoxon test. The chi-square test was used to evaluate the relationship between qualitative variables. A p-value of less than 0.05 was considered statistically significant for all analysis.

RESULTS

In this study, 122 patients with AHT and OME were included. The mean±SD age of patients was 6.7±2.4 years and 68 (55.7%) of patients were male.

Among the 122 patients under survey, 116 had AHT and 30 had OME. The prevalence of allergic disorders in patients with AHT and OME is shown in Figure1. In total, 38 (32.76%) patients with AHT and 9 (30%) patients with OME had allergic disorders.

In patients under the study, 30 patients (24.6%) had higher IgE levels proportional to their age while in the other 92 patients (75.4%) IgE levels were reported to be normal. The mean± SD IgE Level was 156±94 IU/mL. Among the patients with adenoid hypertrophy and elevated IgE, 78% have clinical allergic symptoms. While in other with normal IgE, 19% were reported to have clinical allergic symptoms. A higher IgE level was found in AHT patients with allergic symptoms (p<0.001).

The frequency of atopy diagnosed by history and elevated IgE level (proportional to their age) was 28% (34/122) in the study group. The patients with atopy (34 patients) were asked to take the SPT. Twenty-five patients agreed to take the SPT. Among the 25 patients, all of them had clinical signs of allergy, and seventeen patients (68%) had higher levels of IgE. Allergen sensitization was reported in 11 of 25 patients (44%) who agreed to take SPT. The most common allergens to which patient's sensitivity was found were categorized as below:

Russian thistle: 8 (32%), Tree mix: 7 (28%), Mugwort: 5 (20%), Mite mix: 5 (20%), Grass mix 5 (20%), Cockroach: 3 (12%) and Egg 2 (8%).

The mean ± SD AHT and OME symptom score decreased significantly from 8.72± 1.31 and 7.22± 1.39 before treatment to 4.22 and 4.11 after treatment (p=0.001, p=0.007); respectively.

Response to treatment (defined as a reduction in symptom score of more than 50%) in patients with allergen sensitization (according to SPT test) was significantly higher than patients without allergen sensitization (100%, and 64%) respectively (p<0.05) (Figure 2).

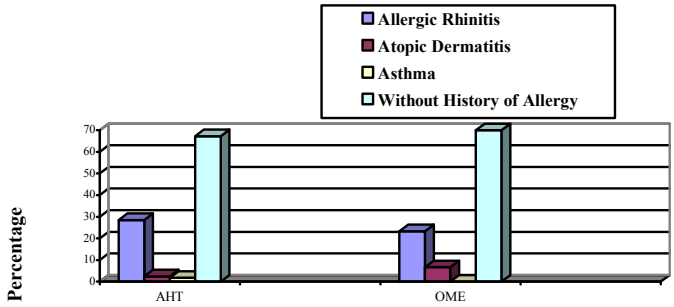

Figure 1. Prevalence of allergic disorders in patients with Adenoid Hypertrophy (AHT) and Otitis Media with Effusion (OME) (In term of percentage from 116 patients with AHT and 30 patients with OME)

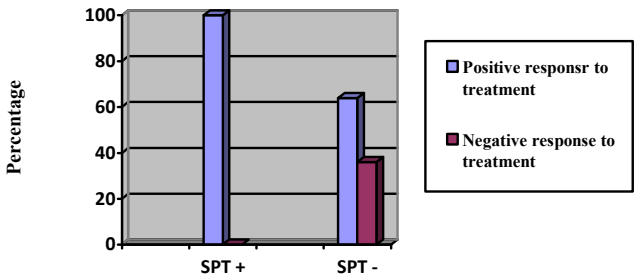

Figure 2. Relationship between skin prick test results (SPT) and response to treatment (Positive response to treatment defined as a reduction in symptom score of more than 50%)

DISCUSSION

The role of allergy in OME has been evaluated in several studies.[7,11] In a study by Tomonaga et al, 50% (out of 259) of the patients with OME were reported to have allergic rhinitis while 21 % of 650 patients, subjected to allergic rhinitis were found with OME.[11] In comparison, the reported rates in our study were 23.3% and 18% respectively. Among different studies the incidence of allergy, according to clinical symptoms in children with OME varied from less than 5% to more than 80%.[7,11] In our study, the association of OME with allergic disorders was 30%.

The implication of allergy in adenoid hypertrophy has also been examined in several studies.[5,8] In a survey conducted by Moderzynski et al, 32.95% of 134 patients with allergic rhinitis were subjected to AHT.[8] In another study conducted by Sadeghi Shabestari et al, from 111 patients with AHT, 25.2% presented a history of cutaneous allergy and 29.7% a history of respiratory allergy.[9] In a study by Mohamed Yonis et al, from the 50 patients with AHT, 74% had allergic disorders. Interestingly, there was a significant negative correlation between allergic disorders and adenoid volume.[5]

In our study, the prevalence of allergic rhinitis was 28.5% in the patients with AHT and OME, while the prevalence of atopic dermatitis and asthma was much lower (equivalent to 2.5% and 1.7%), respectively. the prevalence of allergic diseases in patients with AHT and OME, in our study, were similar to the study of Sadeghi Shabestari et al and was less than Mohamed Yonis's et al study.[5,9] Different results can be due to differences in race, sample size, defined criteria for allergic disorders, inclusion and exclusion criteria of the study, and other unknown factors.

In a study performed by Karaca et al, 82 patients with symptoms of upper airway obstruction were assessed for allergen sensitization by SPT. All patients were sensitized to at least one of the tested allergens.[10] In the Sadeghi Shabestari's et al study, a positive skin prick test was reported in 70.3% of children with AHT, compared with 10% in the control group.[9] In the study by Mohamed Yonis et al, 78% of 50 patients under study were sensitized to at least one allergen. Unexpectedly, sensitization to house dust mite and cotton dust showed a significant negative association with the volume of adenoid.[5] A positive skin prick test in our study was recorded in 11 of the total 25 (44 %) patients with atopy who accepted to take a prick test. The prevalence of allergen sensitization in our study was lower than in similar studies. We think that one of the main reasons for this discrepancy may be the local climate in which the study was conducted. Yazd's climate is hot and dry. Mite (as a common allergen in other areas) is less common in areas with dry climates. Different results can also be due to differences in sample size and study design. There are several pieces of evidence in favor of medical treatment in AHT and OME.[13-15] In a study by S. Cengel et al, 122 children with AHT and/or OME received intranasal mometasone or without any treatment in the control group. The improvement in OME symptoms was significantly higher in the case group (42.2%) than in the control group (14.5%).[13] Berlucchi et al evaluated the efficacy of intranasal Mometasone Furoate for AHT in 57 children. The adenoid size and severity of symptoms decreased in 21 patients (77.7%) of the treatment group. No improvement was found in the placebo group.[14] In another survey conducted by Solmaz et al, they evaluated the effect of topical mometasone on the adenoid volume in 75 patients with AHT. The adenoid volume decreased significantly after treatment with mometasone (p<0.0001), compared to the control group (p=0.3125).[15] In our study, similar to other studies, the severity of symptoms decreased in most patients (80%) who were treated with intranasal corticosteroids. In patients with allergen sensitization response to treatment (100%) was higher than in patients without allergen sensitization (64%).

The lack of a control group in our study did not allow us to make a comparison between treatment groups. We confirmed AHT based on findings of lateral cervical X-rays. Recent studies recommend the use of nasal endoscopy for this purpose. Larger sample sizes are recommended in future studies

The accompaniment of AHT and OME with allergic disorders was common, with a higher rate of allergic rhinitis. Treatment with nasal corticosteroid and antihistamine in patients with AHT and OME who had atopic disorders was effective and this effect was greater in atopic patients who were sensitized to an allergen. A positive skin prick test may be a useful para clinical test to recognize AHT and OME patients with an atopic background whose response to intranasal corticosteroids and antihistamine would be better.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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