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Adenoid hypertrophy in children with allergic disease and influential factors

Muhammed Fatih Evcimik [a,*], Mahmut Dogru [b], Ahmet Adnan Cirik [c], Merve Iseri Nepesov [b]

[a] Istanbul Medipol University, Faculty of Medicine, Department of Otorhinolaryngology – Head & Neck Surgery, Istanbul, Turkey
[b] Zeynep Kamil Woman's and Children's Diseases Training and Research Hospital, Department of Pediatrics, Istanbul, Turkey
[c] Umraniye Training and Research Hospital, Department of Otorhinolaryngology – Head & Neck Surgery, Istanbul, Turkey

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ABSTRACT

Objectives: Adenoid hypertrophy (AH) may cause several comorbid conditions including sleep apnea, chronic serous otitis and sinusitis. Such conditions are more common among children with allergic diseases. In our study, we aimed to determine the patient profile associated with higher incidence of adenoid hypertrophy and the related influential factors.

Methods: The study included 1322 children being treated and followed up for allergic conditions. 100 children with no allergic diseases presenting during the same period to the clinic were included as the control group. Skin prick test for the same allergens was performed for all patients. Adenoid tissue was analyzed by an ENT specialist and the diagnosis was confirmed based on the patient history, endoscopic physical examination and radiology.

Results: Of the patients, 765 (57.9%) were males and 557 (42.1%) were females and their mean age was 5.9 ± 3.3 years. In the control group, 56 (56%) children were males and 44 (44%) were females and their mean age was 6.3 ± 4.1 years. Children with allergic disease and control subjects did not differ significantly by age and gender. Adenoid hypertrophy was identified in 164 (12.4%) of the patients with allergic disease and in 3 (3%) of the controls. Allergic children were divided into two groups, as children with and without AH, respectively. The groups did not differ statistically significantly by gender, age or familial history of atopic disease. However, cigarette smoke exposure at home and presence of allergic rhinitis was significantly more frequent in the group of patients with AH. In the logistic model investigating the effect of variables on AH presence (according to age, gender, cigarette smoke exposure, asthma, AR, AD presence, atopy presence, sensitivity to house dust, pollen, epithelium, *Alternaria alternata* and cockroach), AR presence and cigarette smoke exposure were statistically significant.

Conlusions: AH frequency is higher in children with allergic disease compared to controls. The most common sensitivity to allergens among patients with AH was to house dust. Presence of allergic rhinitis and cigarette smoke exposure are risk factors for developing AH. Children with these risk factors should be questioned for AH during their routine examinations.

1. Introduction

Adenoid tissue integrity varies among children but is usually maximal between 2 and 6 years of age, after which the size undergoes regression. As a part of the nasopharyngeal lymphoid tissue, adenoids normally provide resistance against upper respiratory tract infections (URTI) but they may per se become a source of recurring and chronic infection [1]. Adenoid hypertrophy (AH) may cause several comorbid conditions including sleep apnea, chronic serous otitis and sinusitis [2]. Such conditions are more common among children with allergic diseases [3,4].

Allergic diseases in children mostly manifest as asthma, allergic rhinitis (AR) and atopic dermatitis (AD). AH and allergic diseases are the most common cause of morbidity in childhood. They are therefore simultaneously present in a large patient population. There are many studies on AH occurrence with allergic diseases, although the relationship between the etiology of allergy and AH

* Corresponding author at: Istanbul Medipol University, Faculty of Medicine, Department of Otorhinolaryngology, TEM Avrupa Otoyolu Göztepe Çıkışı No: 1, Bağcılar 34214, İstanbul, Turkey. Tel.: +90 533 6253465; fax: +90 212 4523336.
E-mail address: mfevcimik@medipol.edu.tr (M.F. Evcimik).

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has not been investigated sufficiently [5]. In our study, we aimed to determine the patient profile associated with higher incidence of adenoid hypertrophy and the related influential factors.

2. Material and method

The study included 1322 children being treated and followed up for allergic conditions (asthma, AR, AD) in the Pediatric Immunology and Allergy outpatient clinic of Zeynep Kamil Woman's and Children's Diseases Training and Research Hospital. One hundred children with no allergic diseases presenting during the same period to the clinic were included as the control group. Detailed history of allergic conditions was obtained for each patient. Asthma diagnosis was based on the GINA guidelines [6], allergic rhinitis diagnosis was based on the ARIA criteria [7] and AD diagnosis was based on Hanifin Rajka criteria [8]. Skin prick test for the same allergens was performed for all patients. Total IgE and percentage eosinophil were measured. Approval of the local ethics board was received for the study. Consent to participate in the study was received from the parents of all subjects.

2.1. Evaluation for adenoid hypertrophy presence

Adenoid tissue was analyzed by an ENT specialist and the diagnosis was confirmed based on the patient history and endoscopic physical examination. Adenoidal hypertrophy was evaluated to measured choanal occlusion by nasofiberendoscopy (Karl Storz 1101-RPI) by one of the authors. For children who failed to cooperate in endoscopic examination, AH was investigated using lateral nasopharyngeal radiography. The percentages of airway occlusion, adenoid-nasopharynx ratio were assessed by authors. AH diagnosis was based on Saedi criteria [9].

2.2. Application and evaluation of skin prick test

Skin prick tests were applied on the anterior surface of the forearm when they were appropriate for test such as they were not taking antihistamines. Histamine (10 mg/ml) and physiological saline were used as positive and negative references, respectively. Skin reactions were evaluated 20 min after the application of the skin test, and indurations of $\geq$3 mm was considered indicative of a positive reaction. Skin prick tests to common aeroallergens (*Dermatophagoides pteronyssinus*, *Dermatophagoides farinea*, mixture of grass pollens (*Lollium perenne*, *Dactylis glomerata*, *Phleum pratense*, *Anthoxanthum odaratum*, *Poa pratensis*, *Festuca eliator*, *Agrostis vulgaris*, *Holcus lanatus*, *Cynodon dactylon*, *Avena sativa*, *Avena fatua*, *Lotus corniculatus*), a mixture of grain pollens (oats, wheat, barley, corn), a mixture of tree pollens (*Acer pseudoplanatus*, *Aesculus hippocastanum*, *Robinia pseudoacacia*, *Tilia platyphyllos*, *Platanus vulgaris*), weed-mix pollens (*Medicago sativa*, *Trifolium pratense*, *Brassica nigra*, *Urtica dioica*, *Rumex acetosa*), *Alternaria alternaria*, cockroaches (*Blatella germanica*), cat dander and dog dander (Stallergenes SA, 92160 Antony, France) were performed using stallerpoint. A positive reaction was characterized as 3 mm or greater than that of the negative control.

2.3. Laboratory analysis

Complete Blood Count analysis was measured with an automated blood analyzer Coulter® LH 780 (BeckmanCoulter Inc., Miami, FL) with original method and reagents. The percentage of eosinophils in complete blood count analysis was assessed. The determination of the total IgE was accomplished by nephelometry in an Array Autoanalyser 360 (Beckman).

2.4. Statistical analysis

Data were analyzed by using the program "Statistical Package for Social Sciences (SPSS for Windows 15.0 Chicago, USA). Values for continuous variables were given as either mean $\pm$ standard deviation or as median, based on the normality of distribution. Student's t test was used in the comparison of normal and homogeneous distribution of the parametric values. Chi-square and Mann Whitney U test were used to compare nonparametric values. $p < 0.05$ was considered the significant value.

3. Results

Of the patients, 765 (57.9%) were males and 557 (42.1%) were females and their mean age was 5.9 $\pm$ 3.3 years (range 2 months to 18 years). In the control group, 56 (56%) children were males and 44 (44%) were females and their mean age was 6.3 $\pm$ 4.1 years (range 6 months to 15 years). Children with allergic disease and control subjects did not differ significantly by age and gender. Adenoid hypertrophy was identified in 164 (12.4%) of the patients with allergic disease and in 3 (3%) of the controls ($p = 0.003$) (Table 1).

Of the patients, 692 (52.3%) had asthma, 777 (58.8%) had AR and 164 (12.4%) had AD. One hundred and twenty of the patients with AR (%15.4), 74 (%10.7) of the patients with asthma and 16 (9.8%) of the patients with AD had AH. Compared to the control group, children with AR, asthma and AD had statistically significantly higher frequency of AH ($p = 0.000$, $p = 0.011$ and $p = 0.030$, respectively).

Of the 164 patients with AH, 120 (73.1%) had AR, 74 (45.1%) had asthma and 16 (9.8%) had AD. Allergic children were divided into two groups, as children with and without AH, respectively. The groups did not differ statistically significantly by gender, age or familial history of atopic disease. However, cigarette smoke exposure at home and presence of allergic rhinitis was significantly more frequent in the group of patients with AH ($p = 0.004$, $p = 0.000$, respectively). There were no significant differences between the groups with respect to total IgE levels and percentage eosinophil ($p < 0.05$) (Table 2).

The groups did not differ statistically significantly with respect to presence of atopy, pollen sensitivity, epithelium sensitivity, *Alternaria alternata* sensitivity and cockroach sensitivity; house dust sensitivity was less common in patients with AH ($p < 0.05$) (Table 3).

In the logistic model investigating the effect of variables on AH presence (according to age, gender, cigarette smoke exposure, asthma, AR, AD presence, atopy presence, sensitivity to house dust,

Table 1
Demographic data of the patients and controls and comparative AH frequencies.

	Patient group ($n = 1322$)	Control group ($n = 100$)	p
Gender			
Male/female (n)	765/557	56/44	0.753*
Age (mean $\pm$ standard deviation) years	5.9 $\pm$ 3.3	6.3 $\pm$ 4.1	0.540**
AH frequency n (%)	164 (12.4)	3 (3)	0.003**

* Chi-square.
** Mann Whitney U test.