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Is there any correlation between allergy and adenotonsillar tissue hypertrophy?

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ABSTRACT

Objectives: Tonsil and adenoid are part of Waldeyer's ring; the basic function of which are antibody formation, which later react against a great variety of antigens. The Adenotonsillectomy is the most common operation in small children but the exact reasons of adenotonsillar hypertrophy remains unknown, some researches have shown that allergy may be at risk factor for adenotonsillar hypertrophy. **Methods:** Thorough one year two separated groups of children at the ENT and allergy ward of children's hospital was enrolled in the study. The study group consisted of 117 children between 1 and 14 years old (with average of 6) who had adenotonsillar hypertrophy. The control group consisted of 100 children in the similar age that had not adenotonsillar hypertrophy. Both groups were examined for the incidence of allergic disease, results of skin prick test, serum IgE levels and close contact to smoke.

Results: In the study group 70.3% of children with adenotonsillar hypertrophy had positive skin prick test. But only 10% of children in control group had positive skin prick test. Increased serum total IgE level was confirmed in 48% of children with positive skin prick test in study group were in close contact with smoker parents.

Conclusion: Allergy and sensitivity to different kinds of allergens are important risk factors for adenotonsillar hypertrophy in children. Allergy control may have role in reducing the rate of adenotonsillectomy in children suffering allergic reactions with adenotonsillar hypertrophy.

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1. Introduction

The adenoids and tonsils are predominantly B cell organs: B cell lymphocytes comprise 50–65% of all adenoid and tonsillar lymphocyte. T cell lymphocytes comprise approximately 40% of adenoid and tonsillar lymphocytes, and 3% are mature plasma cells [1].

During childhood, adenotonsillar tissue has role in resistance of the host against the pathogens; many organisms can induce inflammation of Waldeyer's ring. Because the oropharynx is colonized by many organisms, most infections of Waldeyer's ring are poly microbial. Some of the organisms are part of the normal oral pharyngeal flora; where others are external pathogen. Some studies suggested new hypothesis for chronic adenotonsillar tissue inflammation, such as chronic infection by *Helicobacter pylori* [2,3].

There are also some issues that allergy may cause adenotonsillar inflammation in children. The adenotonsillar tissues are continuously exposed to antigens, including allergens that enter the body. Although the relationship between adenotonsillar

hypertrophy and allergy has been discussed in medical books, in fact there have been no comprehensive studies of the problem.

The reports on this condition have consisted mainly of general statements that have not been based on specific research studies [4].

In this present study we examined any correlation between allergy and adenotonsillar tissue hypertrophy.

2. Methods

The present study was conducted at the Tabriz Medical University Children's hospital where is the tertiary referral center from 2007–2008. The study group included children with adenotonsillar hypertrophy (selected randomly) and control group included of children who had other illnesses except adenotonsillar hypertrophy.

The inclusion criteria was diagnosis of adenotonsillar hypertrophy based on taking medical history and head and neck physical examination and lateral neck radiography in some circumstances to find objective and subjective evidences of adenotonsillar hypertrophy by an otolaryngologist in the ENT clinic. We use Brodsky et al. assessment scale for tonsillar hypertrophy so in this scale 0 indicates that the tonsils do not impinge on the airway, 1+

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indicates less than 25% air way obstruction, 2+ indicates 25–50% airway obstruction, 3+ indicates 50–75% airway obstruction, and 4+ indicates more than 75% obstruction. All of children were referred to allergy clinic excluding those who previously had used long-term intranasal steroids or antihistamines and those who had undergone any surgical intervention on adenotonsillar tissues.

Allergy was confirmed by skin prick test (SPT) and in some cases, by serum IgE level determination using the ELISA test as well.

Reactions to seasonal inhaled allergens including (pollen, grass, weed, and tree) perennial allergens (mould spores, dust mites, animal dander, and feather), food allergens (almond, egg, milk, tomato, wheat, fruit) and chemicals (e.g. latex) were assessed. The tests manufactured by the French company Stallergenes were used.

A questionnaire was completed regarding obstructive and infectious complaints suggesting adenotonsillar hypertrophy and history of allergy in patients and their families. Then the results of complete blood count, serum IgE level and skin prick test were added to each patient questionnaire.

To avoid inaccuracies in estimating the missing values due to discontinuation, only the children who completed the study were included in this analysis. The statistical analysis was performed using chi-square test. The guardians signed their informed consent after receiving information about the study.

The study protocol was approved by the Ethical Committee of Islamic Azad Tabriz University of medical sciences.

3. Results

The study group consisted of 117 patients (6 missed, 44 girls, and 67 boys). The control population consisted of 100 children (40 girls and 60 boys). The ages ranged from 1 to 14 years, with a mean age of 6 years in both groups.

By completing the questionnaire, 70.3% ($n = 78$) of patients and 10% ($n = 10$) of control group had positive skin prick test. The difference of positive SPT in both groups was analyzed and with 95% confidence interval, it was statistically significant ($P = 0.04$). Prick test was negative in 90% of control group.

Comparing the serum IgE level in both the study and the control groups, showed that serum IgE level was increased to 8.3% ($n = 8$) in study group (8 patients were missed) and 1% ($n = 1$) in the control group (Fig. 1). A chi-square analysis was statistically significant ($P = 0.04$).

On the other hand, results showed that 48% ($n = 43$) of patients with positive SPT had smoker guardians at home. A chi-square

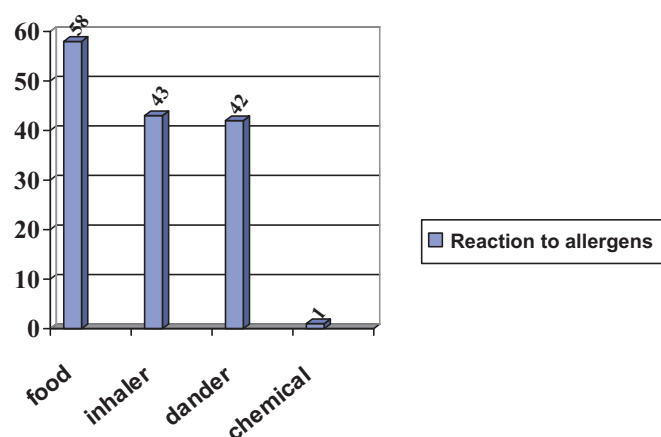


Fig. 2. Frequency of reactions to allergens.

analysis showed statistically significant values ($P = 0.02$). 23.3% of patients with negative SPT and only 5% of control group had smoker at home.

The most common signs and symptoms of adenotonsillar hypertrophy were: snoring 81.1% ($n = 90$), breathing with open mouth 77.5% ($n = 86$), recent pharyngitis 66.7% ($n = 74$), halitosis 50.5% ($n = 56$), sleep problems 45.9% ($n = 51$), chronic pharyngitis 41.8% ($n = 48$), dysphagia 29.7% ($n = 33$), recurrent otitis media 16.2% ($n = 18$), enuresis 10.8% ($n = 12$), failure to thrive 4.5% ($n = 5$), febrile convulsion due to pharyngitis 0.9% ($n = 1$) and peritonsillar abscess 0.9% ($n = 1$).

From 111 patients with adenotonsillar hypertrophy, 25.2% ($n = 17$) had history of cutaneous allergic reactions (eczema, urticaria, and purities), 29.7% ($n = 22$) of them had a history of respiratory allergic reactions (asthma and allergic rhinitis). The history of gastrointestinal allergic reactions has not been studied (Fig. 2).

Positive history of cutaneous allergy in relatives of patients with positive SPT was confirmed in 22.5% of patients and respiratory reactions were confirmed in 45% of them, respectively.

In study group 28.8% ($n = 23$) of patients had positive SPT to one allergen, 61.3% ($n = 49$) of them had positive SPT to 1–6 allergens and 10% ($n = 8$) of patients had positive SPT to more than 6 allergens. Food allergens and in the next steps; pollens, dust mite, animal dander and finally latex were the most common allergens. The difference between cutaneous allergic reactions and living place was not statistically significant (CI: 95%, $P = 0.94$) also, the difference between respiratory allergic reactions and living place was not statistically significant (CI: 95%, $P = 0.75$).

4. Discussion

Adenotonsillar tissues are the main component of Waldeyer's ring and can be hypertrophied by different causes [1]. Allergy is thought to be a risk factor for adenotonsillar hypertrophy. Allergy mainly comprises upper respiratory tract and mostly represents as allergic rhinitis; but due to anatomic nearness of pharynx to nose and the same lymphatic drainage of these regions; it is thought that allergens and especially respiratory allergens may cause adenotonsillar hypertrophy. Some studies have been done to manifest this correlation. All classes of immunoglobulin are synthesized in pharyngeal tonsil. This includes IgE, which is an essential class for allergic reactions [4,5].

Marek and colleagues studied the prevalence of adenoid hypertrophy in allergic patients and showed that adenoid hypertrophy in patients with allergic rhinitis is more common than other allergic patients. They also found that respiratory

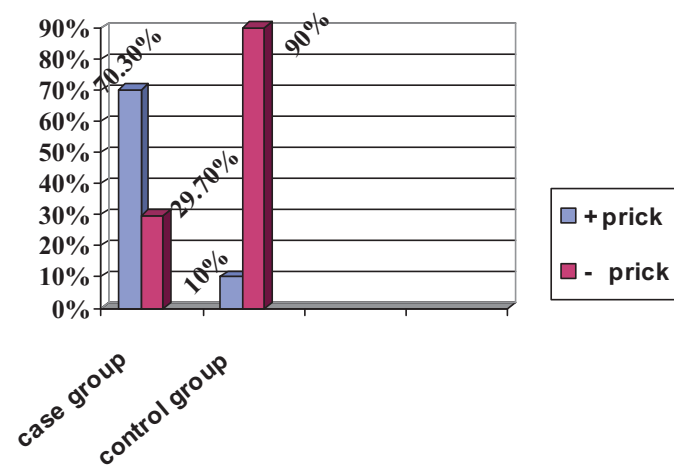


Fig. 1. Comparison of the percentage of positive skin prick test in case and control groups.

allergens are the most common allergens. [6]. In our study, respiratory allergy and allergic rhinitis was the most common form of allergy.

Huang et al. show that adenoid hypertrophy occurred more frequently in children with allergic rhinitis due to allergy to mould spores than in children allergic to other allergens [7]. Also in another study, dust mites were the most common allergens that were found in patients with adenotonsillar hypertrophy but food allergens were not shown to play a significant role in such group. In our study a large percentage of patients were allergic to food allergens and then inhaler allergens. Ganzer and Bachert show that production of sIgE in allergic rhinitis occurs not only in nasal mucosa but also within the lymphatic tissue of Waldeyer's ring [8].

In another study; Becker and colleagues stated that in adenoidal homogenates from atopic children, the same type of sIgE was detected in serum and in skin prick tests [9]. On the other hands Griffin et al. measured serum IgE levels in all patients but the IgE levels were found to lie within normal range and concluded that the prevalence of allergy in children with severe adenotonsillar disease is the same as that of age-matched controls [10].

In our study; total serum IgE level was significantly higher than children with no evidence of adenotonsillar hypertrophy ($P < 0.005$).

Endo and Bellioni have shown in different studies that allergic reactions of the nasal mucosa are also found in pharyngeal tonsils [11,12]. Sydor and Chodynicky showed that mast cells are found in pharyngeal tissue especially in interfollicular region [13]. Zielinski reported that lymphatic follicles are numerous and larger in children with allergic rhinitis [14].

Endo and Karchev state that eosinophilic infiltration and interstitial edema in sub epithelial region is typical finding in allergic reactions and is identified in children with allergic rhinitis [15,16]. In our study, the adenotonsillar tissues were not examined histopathologically, but any correlation was not found between eosinophilia and adenotonsillar hypertrophy.

Modrzynski shows that during nasal endoscopes and acoustic rhinometry in spring season, pharyngeal tissue transiently is hypertrophied [17]. He also examines the influence of atopy on the adenoid hypertrophy and suggests that allergic rhinitis is an important risk factor for adenoid hypertrophy in children and in other study they examined the incidence of adenoid hypertrophy in allergic children and concluded that the risk of adenoid hypertrophy in allergic children hypersensitive to dust mites is greater only in them with allergic rhinitis [18,19]. Hofman et al. studied the influence of IgE-mediated sensitization on adenotonsillar disease in children and showed 22.9% of adenotonsillectomized children had positive Rast results to inhalative allergens [20].

In most of the studies, the correlation of adenotonsillar enlargement in allergic patients and their immunologic and histologic findings have been assessed, but no study has been conducted to assess the correlation of physical and para clinical findings in patients with or without adenotonsillar hypertrophy. Our study showed that, there is a meaningful correlation between positive skin prick test and the high level of serum IgE in patients with adenotonsillar hypertrophy rather than other children.

Also our study stated that smoke as an allergen can be a cause of adenotonsillar hypertrophy in children who has smoker guardians.

In contrast to other studies, in our study, the food allergens and then inhaled allergens were the most common allergens. We think parental awareness of allergic foods and the avoidance of their usage are important factors for prevention of adenotonsillar hypertrophy and also air pollutions may be an additional cause in the industrial cities. However, more investigations are needed to assess this hypothesis.

5. Conclusions

Allergy control may have role in reducing the rate of adenotonsillectomy in children suffering allergic reactions with adenotonsillar hypertrophy.

Smoke cessation and identifying food and respiratory allergens and avoiding contacts with those may have a role in reducing the rate of children candidates for adenotonsillectomy.

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