Original Research

Comparison Of Pathologic Differences In Adenoid Tissues Of Allergic Patients With Non-Allergic Patients

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Abstract

Background: Tonsils are masses of lymphatic tissue that play a significant role in protecting the body against pathogens and allergens due to their location in the upper respiratory and gastrointestinal tracts.
Method: Fifty patients with allergic adenotonsillar hypertrophy and fifty patients with non-allergic adenotonsillar hypertrophy were selected as candidates for surgery by specialist. Obtained samples were sent to the pathology department and then compared in terms of pathology, number of eosinophil, lymphocytes, neutrophils and the presence or absence of lipids by a specialist.
Results: That among all the studied variables, family history of asthma and allergic rhinitis, history of asthma and allergic rhinitis in the patient, positive result of prick test (air allergen, food and mite) and lymphocyte count were significantly higher in patients with allergic adenotonsillar hypertrophy than in patients with non-allergic adenotonsillar hypertrophy. Neutrophil count was significantly higher in patients with non-allergic adenotonsillar hypertrophy than in patients with allergic adenotonsillar hypertrophy (P-value <0.001). In other variables such as eosinophil count, presence of lipids, gender, age, family history of tonsillitis, smoking in the family, largeness of pharyngeal tonsils, diagnosis method, apnea, persistent night snoring and continuous mouth breathing, there was no statistically significant difference between the two groups (P-value> 0.05).
Conclusion: The significant difference between clinical and pathologic characteristics of adenoid tissues of allergic patients and non-allergic patients suggest that special attention should be paid for diagnosis and treatment of these patients. However, more comprehensive study with a larger sample size is needed to evaluate this issue more accurately.

Keywords: Tonsil, Adenotonsillar Hypertrophy, Allergy, Eosinophil, Lymphocyte, Neutrophil
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Introduction

Tonsils are masses of lymphatic tissue that play a significant role in protecting the body against pathogens and allergens due to their location in the upper respiratory and gastrointestinal tracts [1, 2]. Tonsils, are immune system's first line of defense against foreign agents and like other organs in the lymphatic system, play an important role in fighting infections [3,4]. Adenotonsillar hypertrophy is the term usually used to define the atypical enlargement of the pharyngeal and palatine tonsils [5]. Adenotonsillar hypertrophy can occur because of infectious and non-infectious reasons. Among non-infectious reasons, reflux, allergies and exposure to secondhand smoke have been suggested [6]. Furthermore, many infectious agents including Adenovirus, Corona virus, Coxsackie virus, Cytomegalovirus, Epstein-Barr virus, Herpes simplex virus, Parainfluenza virus and Rhinovirus, Haemophilus influenza, Staphylococcus aureus, Neisseria gonorrhoeae, Fusobacterium and Peptostreptococcus can stimulate adenotonsillar hypertrophy [5, 2, 7-9]. Adenoid hypertrophy can also be a sign of a more serious disorders such as lymphoma or nasal sinus malignancy [10,11]. Allergy may play an important role in children with tonsillar hypertrophy. Due to undeveloped immune system and frequent infections and inflammatory disorders related to the respiratory tract, children are more prone to tonsillitis [6]. Adenotonsillar hypertrophy causes nose obstruction, rhinorrhea, nasal breathing problems, cough, snoring, or abnormal breathing in children. In sever obstructions, patient may suffer from sinusitis and also facial pain [12]. Eustachian tube blockage in adenotonsillar hypertrophy may lead to otalgia, hoarseness and recurrent middle ear infections [12]. Adenoid hypertrophy results in behavioral problems, pulmonary hypertension, and psychiatric disorders such as depression in children. Besides other reasons, allergic sensitivity can also cause adenotonsillar hypertrophy [13]. The present study aimed to compare the pathologic differences in adenoid tissues of allergic patients with non-allergic patients for the first time.

Method

This cross sectional study conducted between March and August 2020 in Tabriz Children's Hospital. Fifty patients with allergic adenotonsillar hypertrophy were selected as candidates for surgery according to standard criteria by an allergist. Based on prick test results, the patients divided in aeroallergen, food allergy and mite allergy. Fifty patients with non-allergic adenotonsillar hypertrophy were selected according to an ENT specialist as candidates for surgery by the nasopharynx. The inclusion criteria were proved allergic and non-allergic adenotonsillar hypertrophy, age 14 or younger and patients desire to participate in the study. The exclusion criteria were age more than 14 and the patient's unwillingness to participate in the study. A questionnaire was prepared for patients and their demographic information was completed and then they underwent tonsillectomy by a specialist. After surgery, the samples were obtained, placed in a formalin fixative solution, and sent to the pathology department. The samples underwent tonsillectomy by a specialist. The sections of sampled were prepared and stained with hematoxylin and eosin. Tonsil samples were compared in patients with allergic and non-allergic adenotonsillar hypertrophy in terms of pathology, number of eosinophil, lymphocytes, neutrophils and the presence or absence of lipids. The number of neutrophils in the inflammatory part of the tonsils was counted in several fields with 1000x magnification and the average number in each field was estimated. The mean eosinophil count was reported in 10 fields with 1000x magnification. The presence or absence of adipose tissue around the tonsil tissues was also reported. Ethical Committee of Tabriz University of Medical Sciences approved the study. The study was started after obtaining the consent of the children's parents. Treatment was provided to

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patients free of charge, and patients and their parents could leave the study at any time. All patient information was kept confidential.

Statistical analysis was performed using SPSS v22. The data normality was assessed using Kolmogorov-Smirnov normality test. Frequency (percentage) was used to describe qualitative data and mean ± standard deviation was used for quantitative data. Where the data was not normal, the median (25th and 75th percentiles) was used. Chi-square test was used to analyze the qualitative data in both groups. Independent t-test was used to analyze quantitative normal data in both groups. The Mann-Whitney test was used if the data was not normal. A P value< 0.05 was considered as statistically significant.

Results

In this study, 100 patients with allergic and non-allergic adenotonsillar hypertrophy who were candidates for surgery were evaluated. The mean age of the patients with allergic and non-allergic adenotonsillar hypertrophy was 7.48 (±2.3) and 6.80 (±2.6) years, respectively. In allergic and non-allergic adenotonsillar hypertrophy patients, 30 cases (60%) and 36 cases (72%) were male, respectively. Family history of asthma and allergic rhinitis, history of asthma and allergic rhinitis in the patient, positive result of prick test (air allergen, food and mite) and lymphocyte count were significantly higher in patients with allergic adenotonsillar hypertrophy than in patients with non-allergic adenotonsillar hypertrophy ($p<0.001$ for all). The mean of eosinophil count was 16.44 (±6.0) and 18.02 (±6.3), lymphocyte counts was 7.48 (±1.4) and 5.84 (±2.4), neutrophil counts was 10.72 (±2.9) and 15.93 (±2.8), and the presence of lipids was 18 cases (48.6%) and 19 cases (51.4%), in the allergic and non-allergic group respectively. The neutrophil count was significantly higher in patients with non-allergic adenotonsillar hypertrophy than in patients with allergic adenotonsillar hypertrophy (P-value <0.001). While in other variables such as eosinophil count, presence of lipids, gender, age, family history of tonsillitis, smoking in the family, largeness of pharyngeal tonsils, diagnosis method, apnea, persistent night snoring and continuous mouth breathing, there was no statistically significant difference between the two groups (P-value> 0.05).

Discussion

In the present study, the pathologic differences in adenoid tissues of allergic and non-allergic adenotonsillar hypertrophy patients were investigated for the first time. Among all the studied variables, family history of asthma and allergic rhinitis, history of asthma and allergic rhinitis in the patient, positive result of prick test and lymphocyte count were significantly higher in patients with allergic adenotonsillar hypertrophy than in patients with non-allergic adenotonsillar hypertrophy. Neutrophil count was significantly higher in patients with non-allergic adenotonsillar hypertrophy than in patients with allergic adenotonsillar hypertrophy.

Neutrophil count was significantly higher in patients with non-allergic adenotonsillar hypertrophy than in patients with allergic adenotonsillar hypertrophy. Some studies have investigated the effects of allergic conditions on neutrophil count of adenotonsillar hypertrophy patients. In agreement with our results, Quaranta et al. [14] study on the role of different types of chronic rhinitis in the development of otitis media with effusion in children with adenoid hypertrophy reported that neutrophil count was significantly lower in patients with allergic rhinitis than in patients with non-allergic rhinitis.

The present study showed that lymphocyte count was significantly higher in patients with allergic adenotonsillar hypertrophy than in patients with non-allergic adenotonsillar hypertrophy. Sadeghi et al. [15] declared that sensitivity to allergens and allergy are risk factors of children tonsillar hypertrophy. Some other studies have reported that children with allergies are more susceptible to develop tonsillar hypertrophy [16, 17]. The lifespan of T lymphocytes is reported to prolonged

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in allergic inflammation [18]. This may explain the higher lymphocyte count in patients with allergic adenotonsillar hypertrophy.

The results of this study indicated that family history of asthma and allergic rhinitis, history of asthma and allergic rhinitis in the patient and positive result of prick test were significantly higher in patients with allergic adenotonsillar hypertrophy than in patients with non-allergic adenotonsillar hypertrophy. In fact, these results were expected because family or patient history of asthma and allergic rhinitis have direct relationship with allergic conditions, furthermore, positive result of prick test shows a type of allergy. There was no statistically significant difference in eosinophil count between the patients with allergic and non-allergic adenotonsillar hypertrophy. In agreement with our results, the study by Sadeghi-Shabestari et al., found that no association in present between eosinophilia and adenotonsillar hypertrophy [15]. In another study by Khazraei et al., [19] there was no significant relationship between the number of eosinophils and the presence of allergic rhinitis. It is concluded from their study that the number of eosinophils could not be the sole determining factor in the diagnosis of allergic rhinitis. However, some other studies have reported inconsistent results with the present study findings. Berjis et al. [20] reported that the percentage of eosinophils in nasal secretions has a statistically significant relationship with the result of prick test as the gold standard. They concluded that eosinophil count in nasal secretions is helpful for the diagnosis of allergic rhinitis, however, it has not any significant correlation with the disease severity. Endo et al. [21] and Karchev et al. [22] suggested that eosinophilic infiltration to the sub-epithelial region is a common phenomenon in allergic reactions in children with allergic rhinitis. Differences in the sample size and inclusion and exclusion criteria such as disease severity and underlying factors may explain the discrepancy of the results.

Although our study did not find any statistically significant difference between allergic and non-allergic adenotonsillar hypertrophy patients regarding the smoking in the family, there are several studies that reported apposite results. Evcimik et al., [23] Rout et al., [24] Finkelstein et al., [25] and Virkkula et al. [26] reported that tobacco smoke exposure is a predisposing factors for adenotonsillar hypertrophy. In the study of Hashemian et al., [27] 27% of patients had a history of exposure to cigarette smoke and they suggested it as a predisposing factor in the recurrence of adenoid hypertrophy. Like eosinophil count, differences in the sample size and inclusion and exclusion criteria such as disease severity and underlying factors may explain the discrepancy of this results.

Conclusion

It is concluded from this study that family history of asthma and allergic rhinitis, history of asthma and allergic rhinitis in the patient, positive result of prick test, lymphocyte count and neutrophil count should be considered more in differentiating and treating the allergic and non-allergic adenotonsillar hypertrophy patients.

Conflict of interest

There is no conflict of interest in the implementation of this study.

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Table & Figure:

Table 1. Demographic characteristics of the study population

Variables	Subsets	Groups	Values
Gender	Male	Allergic	30 (60%)
		None-allergic	36 (72%)
	Female	Allergic	20 (40%)
		None-allergic	14 (28%)
Age		Allergic	7.48±0.4
		None-allergic	6.80±0.6

Table 2. Risk factors and diagnosis in patients with allergic and non-allergic adenotonsillar hypertrophy

Variables	Subsets	Groups	Values
Family history of tonsillitis		Allergic	12 (24%)
		None-allergic	11 (22%)
Family history of allergies	Asthma	Allergic	18 (36%)
		None-allergic	0
	Allergic rhinitis	Allergic	26 (52%)
		None-allergic	0
	Eczema	Allergic	0
		None-allergic	0
	Rash	Allergic	2 (4%)
		None-allergic	0
Smoking		Allergic	10 (20%)
		None-allergic	18 (36%)
History of allergies	Asthma	Allergic	18 (36%)
		None-allergic	0
	Allergic rhinitis	Allergic	26 (52%)
		None-allergic	0
	Eczema	Allergic	4 (8%)

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		None-allergic	0
	Rash	Allergic	2 (4%)
		None-allergic	0
prick test result	Negative	Allergic	4 (8%)
		None-allergic	8 (16%)
	Aeroallergen	Allergic	48 (96%)
		None-allergic	0
	Food	Allergic	40 (80%)
		None-allergic	0
	Mite	Allergic	24 (48%)
		None-allergic	0
Largeness of pharyngeal tonsils	Degree 2	Allergic	50 (100%)
		None-allergic	47 (94%)
Adenoid largeness	Degree 2	Allergic	50 (100%)
		None-allergic	46 (92%)
	Degree 3	Allergic	0
		None-allergic	1 (2%)
Diagnosis	Patient history	Allergic	0
		None-allergic	0
	Throat Exudate	Allergic	40 (80%)
		None-allergic	37 (74%)
	Fever	Allergic	26 (52%)
		None-allergic	24 (48%)
	Cervical lymphadenopathy	Allergic	2 (4%)
		None-allergic	0
	Without coryza symptoms	Allergic	0
		None-allergic	0
Sleep disorders		Allergic	2 (4%)
		None-allergic	0
Snoring at night		Allergic	50 (100%)
		None-allergic	45 (90%)
Breathing with open mouth		Allergic	50 (100%)
		None-allergic	46 (92%)

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Table 3. Demographics, risk factors and diagnosis results in patients with allergic and non-allergic adenotonsillar hypertrophy

Variables	Groups	P-value
Age	Allergic None-allergic	0.165*
Gender	Allergic None-allergic	0.205**
Family history of tonsillitis	Allergic None-allergic	0.812**
Family history of asthma	Allergic None-allergic	0.000**
Family history of allergic rhinitis	Allergic None-allergic	0.000**
Family history of smoking	Allergic None-allergic	0.705**
History of asthma	Allergic None-allergic	0.000**
history of allergic rhinitis	Allergic None-allergic	0.000**
History of eczema	Allergic None-allergic	0.117***
History of rash	Allergic None-allergic	0.495***
prick test result	Allergic None-allergic	0.000**
Adenoid largeness	Allergic None-allergic	0.485***
Throat exudate	Allergic None-allergic	0.476**

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Fever	Allergic / None-allergic	0.689**
Cervical lymphadenopathy	Allergic / None-allergic	0.495***
Sleep disorders	Allergic / None-allergic	0.495***
Constant snoring at night	Allergic / None-allergic	0.056***
Breathing with open mouth	Allergic / None-allergic	0.117***

* P-value by independent samples t-test.

** P-value by Chi-Square test.

*** P-value by Fisher's exact test.

Table 4. Eosinophil, lymphocyte, neutrophil and lipid levels in patients with allergic and non-allergic adenotonsillar hypertrophy

Variables	Groups	Values	P-value
Eosinophil count (10HFP)	Allergic	16.44±6	0.202*
	None-allergic	18.02±6.3	
Lymphocyte count (LFP)	Allergic	7.48±1.4	0.000*
	None-allergic	5.84±2.4	
Neutrophil count (HFP)	Allergic	10.72±2.9	0.000*
	None-allergic	15.39± 2.8	
Presence of lipids	Allergic	48.6±18	0.715**
	None-allergic	51.4±19	

* P-value by independent samples t-test.

** P-value by Chi-Square test.

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