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An analysis of the incidence of adenoid hypertrophy in allergic children

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Summary

Introduction: The adenoidectomy is the most often made operation in small children but the reasons of adenoid hypertrophy are not completely explained. Some researches show that in part of children, allergy can be a risk factor for adenoid hypertrophy. The main aim of this study was the analysis of adenoid hypertrophy in children with different allergic diseases.

Material and methods: Two separate groups of children were examined. The study group consisted of 436 children between 4 and 9 years old with allergic rhinitis and/or bronchial asthma and/or atopic dermatitis hypersensitive to house dust mites (interview, positive skin-prick test results).

The control group consisted of 229 non-atopic children (negative interview and skin-prick tests) in the similar age. In the both groups we examined the incidence of adenoid hypertrophy and its dependence of the kind of allergic disease, age, sex, infections, results of skin-prick test and other sensitization.

Results: The probability of adenoid hypertrophy was statistically more significant (logistic regression analysis) only in children from the study group with allergic rhinitis. There were no differences in adenoid hypertrophy incidence between children with other allergic diseases and the control group. The children from the study group with adenoid hypertrophy were more often hypersensitive to pollen and moulds allergens than the children without adenoid hypertrophy (chi square test). But there were no differences in the incidence of infection, age, sex and results of skin-prick tests.

Conclusion: The result of our study suggests that the chance of adenoid hypertrophy in allergic children hypersensitive to dust mites is greater only in them with allergic rhinitis.

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1. Introduction

Elements of Waldeyer's ring constitute a component of lymphatic tissue connected with mucosa (mucosa associated lymphoid tissue—MALT) forming the first barrier and area of contact for antigens entering the body, including allergens [1,2]. Advancements in immunology and better understanding of the role of this tissue in the immunological system have led to more cautious assessments of the need for surgery in the area and a tendency to retain the structure [3]. This is undoubtedly a result of better insight into the factors responsible for adenoid hypertrophy. One of the factors most frequently quoted in all ENT handbooks is allergy. Unfortunately, the few publications that discuss this issue do point to the fact that allergy is more frequent in children with adenoid hypertrophy but describe the differences observed as fairly insignificant [4,5]. It may be useful to look at this problem from a different perspective and try to answer the question whether in children with allergy the incidence of adenoid hypertrophy is increased when compared with children without hypersensitivity, and whether organ localization of the allergic disease is significant here. The study presented here is an attempt to answer this question.

2. The aim of the study

The aim of the study was to estimate the incidence of adenoid hypertrophy (AH) in children hypersensitive to dust mites.

3. Material and methods

In a study conducted between 1999 and 2006, 655 children took part. Children were divided into either a study group (SG) or a control group (CG). In all children AH had been diagnosed not only by clinical picture (the symptoms of adenoid hypertrophy and perennial allergic rhinitis are often very similar) but also by additional commonly recognized diagnostic tests. Therefore in every case to confirm the hypertrophy at least two of the following methods were used: mirror examination (posterior rhinoscopy), acoustic rhinometry, lateral radiograph of the nasopharynx and fiberoptic examination of the nasopharynx. The palpation of the adenoid was not used due to little repeatability and reliability.

We consider a case to be adenoid hypertrophy if the adenoid covers at least 50% of the choana in posterior rhinoscopy.

In the radiological assessment we look at the difference between the width of the soft palate

1 cm below the hard palate and the width of the airway between the palate and the adenoidal shadow according to Cohen and Konak method. The adenoid hypertrophy was diagnosed if the distance was narrower than the width of the soft palate [6].

For the endoscopic examination of the nasopharynx we used a flexible fiberoptic nasopharyngoscope, Olypmus ENF P3 (made in Japan). The size of the adenoid was classified into one of the four categories based upon the degree to which the adenoid obstructed the nasopharyngeal airway [7]:

- 1° Adenoid obstructing less than 25% of the nasopharyngeal airway.
- 2° Adenoid obstructing 25–50% of the nasopharyngeal airway.
- 3° Adenoid obstructing 50–75% of the nasopharyngeal airway.
- 4° Adenoid obstructing more than 75% of the nasopharyngeal airway.

In this method, we diagnose adenoid hypertrophy if the adenoid occupies at least 50% of the nasopharyngeal airway.

For acoustic rhinometry the device used was a RhinoScan with the SRE 2000 module (made in Denmark). To avoid irritation of nasal mucous, this kind of examination was always carried out before the endoscopic inspection. A nasal decongestant was used prior to measurements in order to reduce the effects of nasal mucous edema, which can skew the results of rhinometry, especially on the posterior section of the rhinometric curve. Three measurements were made for each side of the nose. Curves with obvious artefacts were discarded. The mean area-distance curve was calculated for each side of the nose. The results were determined by measuring fitted published data which state that the pharyngeal tonsil is located approximately between 6 and 12 cm from the beginning of the front nostril (nasopharyngeal cross-sectional area) [8].

In the case of a normal and non-constricted nasopharyngeal cavity, it is typical for the rhinometric curve section corresponding to the cavity to diverge from the Y-axis of the diagram. This is due to the fact that the two narrower interior nose parts are joined into one larger that is one cavity of the upper pharynx. The curve approaching the Y-axis indicates that there is a narrowing of nasopharyngeal cavity which is caused by the adenoid enlargement in children in nearly all cases.

The acoustic rhinometry was used only as the second, additional diagnostic method.

In the SG there were 436 children (207 girls and 229 boys) aged between 4 and 9 (mean age 6.46 ± 1.57) hypersensitive to dust mites who,

during the course of the study, suffered from typical allergic diseases entities: chronic (perennial) allergic rhinitis (AR), bronchial asthma (BA) and atopic dermatitis (AD). The occurrence of BA was detected in 345 (79.1%) children, AR in 148 (33.9%) children, AD in 29 (6.7%) children. These conditions occurred individually or in various combinations and the frequency of their occurrence was different for children of different ages (Table 1).

In all the children examined the allergic background of the illness was confirmed by medical history, a clinical findings and positive results of skin prick tests with dust mite allergens (*Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*; tests 708 and 725 manufactured by Allergopharma Co.).

The control group included 229 children (111 girls and 118 boys) between 4 and 9 years of age (mean age 6.51 ± 1.66 years) in whom symptoms of allergic disease had not been detected and skin prick tests with the sensitizing allergens yielded negative results. In this study, apart from assessing the influence of the above-mentioned conditions on the incidence of AH, the following aspects were analyzed:

- age and sex of the children under examination;
- presence of hypersensitivity to allergens other than dust mites;
- occurrence of symptoms of atopic dermatitis in the first year of life;
- results of skin prick test (SPT) to *Dermatophagoides pteronyssinus* allergen;
- recurrent states of inflammation of the upper respiratory tract found in patients' medical history.

Hypersensitivities to other allergens are understood as presence of positive results of skin prick tests with allergens such as: plant pollens (trees, grasses/rye, weeds), mold spore (*Alternaria alternata*, *Cladosporium herbarum*), domestic animal allergens (dog, cat, guinea pig, hamster, rabbit), plumage allergens (hen, duck, goose), milk, flour, eggs, soya beans and peanuts. Tests were conducted with the use of allergen mixtures as well as single allergens manufactured by Allergopharma Co. (Germany). For the purpose of this study, evaluation of

SPT results with mite allergen was carried out using a semi-quantitative scale, by determining the mean value (in millimeters) of perpendicular diameters of the allergic wheal appearing in response to the allergen which is then compared with the value obtained during an analogous survey of the post-histaminic wheal (positive control) and the reaction to negative control (0.9% NaCl).

The coefficient acquired in this way was classified into one of the five degrees [9–14]:

- 0°—an allergenic reaction equal to negative control reaction.
- 1°—mean diameter of the allergen-induced wheal is greater than the reaction to the negative control and smaller than half the diameter of histamine-induced wheal.
- 2°—mean diameter of the allergen-induced wheal is equal to or greater than half the mean diameter of the histamine-induced wheal, but smaller than its full diameter.
- 3°—mean diameter of the allergen-induced wheal is equal to or greater than mean diameter of the histamine-induced wheal, but smaller than its two diameters.
- 4°—mean diameter of the allergen-induced wheal is at least twice as large as mean diameter of the histamine-induced wheal.

In accordance with a commonly accepted principle, reactions smaller than 3 mm were disregarded. By recurrent states of inflammation of the upper respiratory tract, it is understood that there occurred at least four infections within the last 6 months. Because of the type of data analyzed and the fact that their distribution did not fit the normal pattern (Shapiro–Wilk test), several nonparametric tests were used for statistical analysis (described within the text). Cases in which p was less than or equal to 0.05 were considered as significant differences. Calculations were made with the use of the polish version of STATISTICA 5 suite (StatSoft; USA). The study presented here was done as part of a larger study concerning the relations between atopic allergy with pathologies of the pharyngeal lymphatic system. Consent for the study was granted by the local Bioethics Committee.

Table 1 Percentage of children with a given allergic disease in SG in relation to age

The kind of allergic disease	Age (years)					
	4	5	6	7	8	9
Allergic rhinitis	23.4%	27.5%	30.2%	36.1%	37.3%	44.7%
Bronchial asthma	83.0%	79.7%	82.6%	77.1%	77.3%	76.3%
Atopic dermatitis	14.9%	7.2%	5.8%	6.0%	4.0%	5.3%

4. Results

In the SG adenoid hypertrophy was identified in 176 cases (40.4%), whereas in the CG—in 51 cases (22.3%). Therefore, the differences observed were statistically highly significant (chi square test; $p < 0.00000$).

At the same time, closer analysis revealed that the frequency of occurrence of AH in the SG varied and depended on the type of the coexisting allergic disease, which is illustrated by Table 2 below. As can be observed, AH occurred more frequently in children who suffered from AR as a separate disease entity (statistically significant differences: chi square test; $p < 0.00000$) or along with BA (statistically significant differences: chi square test; $p < 0.00000$) while there were no such differences in children with AD, and BA alone was more frequent in children without AH. The graph presented below represents the frequency of AH occurrence in children at different ages who suffer from AR as well as in the remaining part of the SG and in the CG. As Table 3 shows, mean frequency of occurrence of AH in children from SG not suffering from AR (20.6%) was even lower than in the CG, although these differences were so small, that they were statistically insignificant (chi square test; $p = 0.8$).

Children from the SG with AH compared with children without AH were more frequently hypersensitive to certain other air allergens, especially plant pollens ($p < 0.0000$) or mold spore ($p = 0.003$). Such differences (chi square test), however, were not found in relation to plumage allergens ($p = 0.6$), animal allergens ($p = 0.1$), milk ($p = 0.9$), eggs ($p = 0.6$), flour ($p = 0.2$), soya ($p = 0.4$) and peanuts ($p = 0.8$).

The subgroups under comparison did not differ in the frequency of occurrence of AD in the first year of life ($p = 0.3$) and frequent infections of the upper respiratory tract ($p = 0.1$). In the latter parameter, there were no significant differences between

children with AH and without AH in CG ($p = 0.7$). In children with coexistent AR, infections occurred a little more often than in children without allergies (25.8 and 30.1%, respectively; no statistically significant differences).

In the SG, the mean values of SPT results were 2.35 ± 0.75 for children with AH and 2.39 ± 0.75 for children without AH (statistically insignificant differences, Mann–Whitney test; $p = 0.99$). These mean values did not vary when children were additionally subgrouped into those suffering from AR or AD and AS (Kruskal–Wallis ANOVA test; $p = 0.96$).

For evaluation of direction and strength of connection between the existence of AH and other variables evaluated in the SG, nonlinear regression analysis was used with logistic model. The following variables were taken into account:

- AH—adenoid hypertrophy: qualitative dependent variable (1, present; 0, not present).
- AR, BA, AD: qualitative independent variables describing possible presence of a given condition (1, present; 0, not present).
- SPT: qualitative variable describing the result of the skin prick test with dust mite allergen (scale of 1–4).
- INF, I, F: independent variables describing the existence of frequent infections (INF) and possible additional hypersensitivity to any other inhalatory (I) or food allergens (F) tested (1, present; 0, not present).
- AGE—dependent variable describing the age of the children examined (scale of 4–9).

Estimation of the data presented was conducted using Rosenbrock and Quasi Newton method. The model was constructed adopting a hierarchical framework and statistically insignificant variables were gradually removed (stepwise regression; the backward removal method).

The results of the analysis show that for the problem under discussion only two variables proved

Table 2 The incidence of adenoid hypertrophy in children from the study group

	Adenoid hypertrophy present	Adenoid hypertrophy not present	Total
Allergic rhinitis	58 (32.95%)	24 (9.23%)	82 (18.81%)
Bronchial asthma	66 (37.50%)	204 (78.46%)	268 (61.47%)
Atopic dermatitis	1 (0.57%)	2 (0.77%)	3 (0.69%)
Allergic rhinitis and atopic dermatitis	5 (2.84%)	1 (0.38%)	6 (1.38%)
Allergic rhinitis and bronchial asthma	39 (22.16%)	16 (6.15%)	55 (12.61%)
Bronchial asthma and atopic dermatitis	2 (1.14%)	12 (4.62%)	14 (3.21%)
Allergic rhinitis and bronchial asthma and atopic dermatitis	5 (2.84%)	1 (0.38%)	6 (1.38%)
Total	176 (100%)	260 (100%)	436 (100%)

Table 3 Percentage of children with a given allergic disease in SG in relation to age

The examined group	Age (years)					
	4	5	6	7	8	9
Allergic rhinitis present	22.2%	28.0%	25.0%	28.3%	21.3%	17.1%
Allergic rhinitis not present	18.8%	23.3%	32.5%	25.6%	17.5%	14.3%
The whole study group	31.9%	43.5%	45.3%	47.0%	41.3%	28.9%
The control group	63.6%	84.2%	92.3%	80.0%	75.0%	42.9%

to be statistically significant: AGE and AR. The values calculated make it possible to present the model of logistic regression as:

$$P(x) = \frac{e^{-0.008-0.18AGE+2.21AR}}{1 + e^{-0.008-0.18AGE+2.21AR}}$$

A positive estimate of the parameter corresponding to the AR variable indicates that an increase in its value increases the likelihood of adenoid hypertrophy (inversely to age). A calculated individual quotient value (odds ratio) for the AR variable equal to 9.13 confirms the previous observation that in children hypersensitive to dust mites who suffered from AR, the likelihood of AH occurrence was over nine times greater than in children suffering from other allergic diseases (BA, AD).

5. Discussion

Although there are diverse causes of AH and they are not fully understood, it is commonly assumed that chronic, severe and recurrent inflammatory processes occurring around the adenoid may be of particular significance. Allergic reaction is, as is widely known, only one manifestation of such inflammation. The fact that in the age group in which AH occurs most often, AR still appears relatively rarely (allergic march) makes this factor relevant for some children, but at the same time does not allow us to treat it as the essential cause responsible for AH occurrence in the general population. In all likelihood, this is the reason why in children with AH allergy is found only slightly more often than in children without AH. On the other hand, the fact that the clinical symptoms of AR and AH are similar can lead to only one of these disease entities being diagnosed. The fact that most allergists are not laryngologists at the same time may contribute to the situation in which only AR is detected. Some laryngologists who are not allergists may more rarely take account of the allergic background of a child's condition, focusing on AH only. It is known that adenoidal tissue, because of its location on the roof of the nasopharyngeal cavity is constantly exposed to allergens entering the body with the air inhaled through the nose. The proportion of allergen that settles onto the mucous membrane is, due to

ciliary movement, transported to the nasopharyngeal cavity and later to lower parts of the pharynx where it is then swallowed. Mucus is transported from the nasal cavity and paranasal sinuses directly towards the adenoid which is also covered in ciliated epithelium. It is difficult to imagine that contact does not take place between an allergen and immunocompetent cells that constitute one of the main components of adenoidal tissue [15].

From the study presented here it follows, however, that although in children with an allergy AH occurred more often than in the SG, it was not the presence of atopy that was the main causative factor. For the issue under discussion, one factor was more essential and that was allergic inflammation occurring in the nasal mucosa, which is a mucous membrane for which the adenoid is the most closely located mass of lymphatic tissue. Statistical analyses which were carried out revealed that in children with AR the probability of AH occurrence was significantly greater than in the remaining hypersensitive children and reached its peak values of 90% in the group of 6 year olds. On the other hand, the fact that at ages between 8 and 9 even in children with AR there was a sharp drop in the frequency of AH seems to prove that this factor is significant only to a certain age. Most likely, it is caused by the changes taking place in the entire lymphatic system while the maturation period lasts. One of these changes is the decreasing participation of the adenoid in inflammatory processes taking place in the upper parts of the respiratory tract. Not without reason this study was conducted in a group of children hypersensitive to dust mites. Dust mites are one of the most frequent perennial sensitizing allergens in our conditions, and therefore they have clinical significance. Unfortunately, SPT results with dust mite allergen being identical in the group with AH and in the group without AH disqualifies this study as a predictor of the likelihood of AH occurrence. The analyses showed, however, that in children with AH a coexisting hypersensitivity to plant pollen allergens and mold allergens was more frequent than in children without AH. It is highly likely that in areas where allergens other than dust mites dominate, AR caused by these allergens is the main factor leading to allergic inflammation around the adenoid. Work of

Huang and Giannoni [16] may serve to confirm it because they recognize mold allergens as causes of AH. As was correctly pointed out in a commentary to the above-mentioned studies, the children who participated in them lived in characteristic climatic conditions, in areas of very high humidity where mold spore was the main perennial allergen [17]. Although in several other studies the relevance of food allergy in etiopathogenesis of AH was also stressed [18], among the children who took part in the study presented here this factor proved to be negligible. Of course this does not exclude possible participation of food allergy in triggering other laryngological diseases [19]. It must be mentioned that the literature also reports that in children suffering from allergies affecting the upper respiratory tract, infection of adenoidal tissue by rhinoviruses is far easier. It is presumably connected with a higher expression of ICAM-1 [20]. Although in the study presented here this phenomenon was not assessed directly, it is worth stressing that the frequency of infection in children with AH and AR was slightly higher than in children with AH but without any allergy. The findings we have reported can be better interpreted when compared with other reports showing that the lymphatic tissue of the adenoid is involved in allergic inflammatory processes in the nose. Already in the 1980s, it was demonstrated that in patients with AR IgE synthesis was detected around Waldeyer's lymphatic ring and other local lymph nodes rather than in nasal mucosa itself, in which sensitized mast cells occur already covered with antibodies [21]. More recent studies provided new evidence for increased receptor expression demonstrating high affinity with IgE (FcεRI) as well as mRNA for IL-4. This means that in the adenoid there is immunoglobulin class switching to IgE [22–25]. Literature points to analogous links between AR and AH existing in many other dimensions and it is corroborated by many other studies (biochemical, histopathological, cytological, immunohistochemical, etc.) [26,27] and the effectiveness of antiallergic drugs [28]. We can suppose that the eosinophilic cationic proteins and other inflammatory agents can protractedly stimulate the lymphatic tissue, finally causing the hypertrophy of the adenoid.

To summarize, we can say that the presence of AR increases the likelihood of AH occurrence in children, but at the same time it does not mean that it is its main cause. Similar effects can be induced by any other inflammatory process in the vicinity of nasal mucosa. Yet, it is likely that the increasing number of atopic diseases over the past decades, which also affect children [29], may contribute to a gradual increase in the frequency of allergy-induced AH occurrence. Therefore, it is justified to perform

basic allergy diagnostics in all children with AH, at least by means of skin prick tests. It is also advisable to examine the adenoids in children with AR. It is important for the reasons discussed above, i.e. similar symptoms of AR and AH which can lead to a misdiagnosis or an incomplete initial diagnosis and consequently wrong treatment.

6. Conclusions

In children with allergic rhinitis related to hypersensitivity to dust mites, adenoid hypertrophy occurred significantly more frequently than in children with other allergic diseases (asthma/atopic dermatitis) or with no allergies.

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