Original Article

The role of allergy in recurrent otitis media with effusion

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Summary. *Objective:* To compare the OME groups that improved when treated with adenoidectomy, VT and medication, and which of them recurred.
Methods: Twenty-two consecutive children with recurrent OME (11%) were selected among 200 children who had adenoidectomies and had been inserted a ventilation tube (VT) for OME. The patients were selected for allergy evaluation because of recurrent OME not responsive to medical therapy and adenoidectomy, requiring more than one myringotomy with tubes. The control group consisted of 24 healthy children that were treated with adenoidectomy and VT, and had no recurrence.
Results: Eight (36.4%) patients had positive skin tests for inhalant and food allergens. Two subjects of the control group (8.3%) had positive skin tests for inhalant and food allergens. This was statistically significant (p:0. 032). A pattern to multiple positive nasal and food reactions was shown in the study group rather than in the control subjects.
Conclusions: The possibility of allergy should be considered in OME cases that did not improve with drug treatment and adenoidectomy and VT insertion but recurred, and a thorough search for allergen was made for proper diagnostic and therapeutic management.

Key words: Allergy, otitis media with effusion.

Introduction

The mechanism for the development of recurrent otitis media with effusion (OME) should be known because this condition may cause hearing loss especially at a critical time for language and speech development in children. The insertion of ventilation tube (VT) and adenoidectomy for OME are the most common operations performed in the pediatric age group [1]. The knowledge of the factors which influence the outcome of OME in children in the long-term [2] is only limited. Despite adequate treatment, the recurrence and chronicity of OME turns it into a more difficult problem than uncomplicated OME.

Although the clinical importance of OME is well recognized, the precise pathogenesis of the disease process is unknown and the condition has provoked considerable controversy [3]. OME is a multifactorial disease. It has long been recognized that Eustachian tube (ET) dysfunction or obstruction predisposes to OME [4]. Adenoids play an important role in the etiology of OME [5]. Cleft palate, Down's syndrome, Kartagener's syndrome and OME associated with nasopharyngeal neoplasm, all attest to the role of ET dysfunction in OME. Also, immunologic and infectious factors that have been associated with OME include multiple upper respiratory infections, previous episodes of acute otitis media, and public nursery environments. Serious scientific data to support an allergy etiology in pathogenesis of OME, however, are somewhat lacking [1,4].

Although there are many studies about the role of allergy in OME [1-5], the role of adenoid tissue in the pathogenesis of OME is not clear still [1, 4, 6]. Only food allergy or nasal allergy have been studied in detail [3, 5]. Neither of these factors, however, is neutrally exclusive and both could contribute to or be caused by viral or bacterial infections [3].

Comparing the OME groups that improved when treated with adenoidectomy and VT and medication, and which of them recurred constituted the purpose of the present study.

Patients and methods

Twenty-two consecutive patients with recurrent OME (11%) were selected among 200 patients who had adenoidectomies and had been inserted VT for OME, during the period between 1995 and 2001 at the department of Otorhinolaryngology, University of Suleyman Demirel. The patients were selected for allergy evaluation because of recurrent OME not responsive to medical therapy and adenoidectomy, requiring more than one myringotomy with tubes. The same ventilation tubes (Paparella type, 1.27 mm ID, silicone) had been inserted at previous operations in all patients. VTs were Kept in place at least one year unless extruded spontaneously from tympanic membrane. In our study patients had complaints of OME for at least three months after following medical and surgical treatment of OME (as described above). The control group consisted of 24 healthy children who were treated with adenoidectomy and VT, and had no recurrence. These control children had no history of upper respiratory infection in the last 3 months.

Criteria used for diagnosis of OME are tympanometry as a major criterion, as well as audiometry, color and position of the tympanic membrane, and air fluid/air bubbles as minor criteria. Recurrent OME was defined as persistent evidence of effusion by otoscopy, and/or tympanogram with a persistent conductive hearing loss, 3 months after adequate medical therapy. Adequate medical therapy was defined as therapy with appropriate antibiotics (amoxicillin clavulanate 40 mg/kg, or lorakarbef 30 mg/kg) and systemic decongestant and nonsteroid anti-inflammatory agents.

Patients with the following conditions were excluded from the study: Perforated tympanic membrane due to VT inserting, recurrent adenoid vegetations, severely deviated nasal septum, nasal polyposis, and cleft palate. Information regarding whether the patients were otitis-prone (having episodes of acute otitis media within 1 year) was not available.

Diagnosis of inhalant and food allergy include positive epicutaneous prick test (Multitest®, Center Laboratories, Port Washington, NY USA) as a major criterion. History of patients, total IgE, nasal smears as minor criteria. Statistical analysis of data was performed by Fisher chi-square exact, and Mann Whitney U tests.

Results

The study group consisted of 22 patients ranging in age from 5 to 17 years (the mean age was 13). Twelve patients (54.5%) were boys and ten patients were girls (45.5%). One of them had a history of allergy. The control group consisted of 24 children ranging in age from 5 to 16 years (the mean age was 11). Sixteen (66.6%) controls were boys, and eight (33.3%) controls were girls. The entire control group was not allergic by history.

In the study group, all patients had previous adenoidectomies, and had been inserted VT. Two of them (9.1%) had additional tonsillectomies. Eight (36.4%) patients had positive skin tests for inhalant and food allergens. Four patients (18.2%) had a positive test for both inhalant and food allergies; three patients (13.6%) had a positive test only for food allergy; one patient (4.5%) had an allergy only against inhalant allergens. Two subjects of the control group (8.3%) had positive skin tests for inhalant and food allergens. One of them (4.2%) had positive tests for inhalant and food allergies. The other one had a positive test only for food allergy (Figures 1, 2). As a whole, 36.4 per cent of the study group had positive test, as compared with 8.3 per cent of the control population. This was statistically significant (p: 0.032). A pattern to multiple positive nasal and food reactions was shown in the study group rather than the control subjects (Table 1).

On an average, total IgE levels were 23.6±13.9 IU/ml in the study group and 21.4±10.3 IU/ml in the control

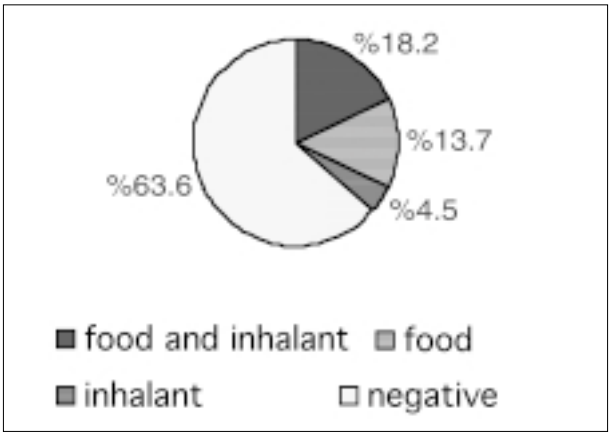

Figure 1. The results of epicutaneous prick test in the study group.

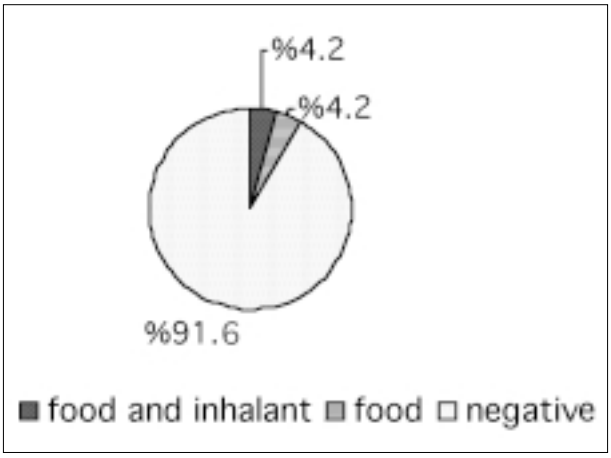

Figure 2. The results of epicutaneous prick test in the control group.

group. There was no significance between the two groups (p:0. 83).

Discussion

Causative factors of OME, such as dysfunction of the ET, infectious inflammation, and allergy, have been considered for many years. Allergy has been considered to be the most important factor of OME based on clinical observations [7-9]. Although allergy had been generally accepted to play a part in the pathogenesis of recurrent OME, the role of allergy in patients who had previous adenoidectomies and had been placed VT tube has been less investigated.

In the pathogenesis of recurrent OME, the role of adenoid tissue has not been studied according to its presence or absence. However, the adenoids play an important role in the etiology of OME. The efficacy of adenoidectomy in the surgical treatment of children with OME has been well established. Kiroglu et al [10], reported that adenoid tissue of patients with OME seems to be an infectious focus, aggravating immune reaction which might attack the middle ear through an ascending route. In children with OME, the main pathological finding associated with tubal dysfunction involved inflammation in the nasopharynx [11]. The pathogenous microorganisms were decreased in the microflora of the nasopharynx after adenoidectomy [5]. Gates, who has performed considerable work on OME and adenoidectomy, reported in a series of articles [12-14] that adenoidectomy has been considered in the initial surgical management of children with OME that is refractory to medical management. Those studies stated that the size of the adenoid cannot be used as a criterion for adenoidectomy in these patients. They suggested that

Table 1. The inhalant and food allergens in the study and control groups.

	The study group	**The control group**
Inhalant		
Mite 1	1	
Sp. Gras Mix	5	1
Mixed Epidermals	2	
Sp. Mold Mix	2	
Food		
Soybean	1	
Cinnamon	1	
Almond	1	
Onion	1	1
Grapefruit	1	1
Orange	1	1
Tea sirensis	1	
Sweet corn	1	
Peanut	1	
Lemon	3	
Tomato	3	
Lettuce	3	
Tuna	2	
Egg white	6	1
Back wheat	5	1
Plum	6	
Beef	3	1
Vanilla	3	
Whole egg	3	
Carrot	3	
Sweet potato	2	
Goat meat	3	
Watermelon	4	
Wallnut	4	
Celery	2	
Mushroom	4	

reduction of the adenoidal bacterial reservoir might be the mechanism whereby adenoidectomy is effective. In this study patients having adenoidectomy with no adenoid tissue recurrence were included. Thus the possible role of adenoid tissue in the pathogenesis of OME was excluded. We think that it is more suitable to form the control group in such a manner, to understand the effects of allergy on recurrent OME.

Bernstein et al [4] suggested that about two thirds of patients with recurrent OME are not allergic, and specifically have no upper respiratory tract allergies, and approximately one third of patients with recurrent OME (35%) do have allergic rhinitis. Becker et al [6] suggested an allergic genesis of recurrent OME and adenoids in about 20 to 30% of patients. Hurst [9] detected an allergic basis in all 20 patients studied on the basis of RAST and skin testing. In his study, following immunotherapy to inhalants, 65% of patients had normal hearing and the remaining 35% resolved following food elimination. Nsouli et al [3] reported that a high prevalence of food hypersensitivity was detected in 78% of patients with recurrent OME. Some investigators reported that food allergies do not play an important role in the pathogenesis of OME [15,16]. The results of the present study reveal that a high prevalence of inhalant and food hypersensitivity was detected in 36.4% of patients with recurrent OME and previous adenoidectomies, who had been placed a ventilation tube when compared with the control group (8.3%). Eight (36.3%) patients had inhalant allergies and 7 (31.8 %) had food allergies in our series. The prevalence of food and inhalant allergy in our series was similar to the reports of Bernstein et al [4] and Becker et al [6]. However, Nsouli et al [3] and Hurst [9] detected a higher prevalence of food hypersensitivity than in our series. This difference could be explained by differences in habitual food.

It has been proposed that the role of allergy in the etiology and pathogenesis of acute and chronic OME may be one or more of the following mechanisms: 1-middle ear acting as a shock organ', 2-inflammatory swelling of the Eustachian tube or ET as a shock organ' 3-inflammatory obstruction of the nose, or 4-aspiration of bacteria-laden allergic nasopharyngeal secretion into the middle ear cavity [3,4,17]. Nsouli et al [3] suggested that allergic food hypersensitivity may trigger OME, and OME may be due to all four mechanisms mentioned. Also, Bernstein et al [4] reported that inhalant and food allergy may act as a cause of recurrent OME; and that the middle ear and ET are the target organs in these patients.

Allergic inflammation, particularly of the posterior nasopharynx at the orifice of the ET and in the ET, is probably the most common cause of ET obstruction [3]. Corey et al [1] suggested that nasal allergy definitely interferes with ET function. They also stated that the site of ET dysfunction due to allergy is located at the nasopharyngeal end of the ET. Hall and Lucat [18] suggest that allergy may contribute to OME by diluting Eustachian tube surfactant, thereby interfering with the Eustachian tubes clearance function. Another explanation made by Mogi et al [19] suggests that IgE-mediated allergic reactions of the mucous membranes lining the nose, nasopharynx, and Eustachian tube are factors indicating of a chronic state of disease, rather than a cause of OME. In our opinion the ET dysfunction among the above four mechanisms may play a most important role in patients with recurrent OME. In the remaining patients without atopy perhaps a different causative factor may play a significant role in the pathogenesis of middle ear effusion.

Our opinion is that allergy probably plays a more important role in recurrent OME than in uncomplicated OME. Thus, the possibility of allergy should be considered in OME cases that did not improve with drug treatment and adenoidectomy and VT insertion, and had recurrence, as well as a through search for allergen made for proper diagnostic and therapeutic management.

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