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Role of Allergy Diagnosed by Immunoglobulin E in the Etiology of Pediatric Otitis Media with Effusion at a Tertiary Care Centre in Southern Tamil Nadu

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Abstract
This study aimed to study the role of allergy diagnosed by immunoglobulin E (IgE) in the etiology of pediatric otitis media with effusion (OME) by detection of IgE in middle ear effusion using the enzyme linked immunosorbent assay technique. This study was carried out in the period from June 2023 to May 2024 in the Dept of ENT, Sree Mookambika Institute Medical Sciences, Kulasekharam, Tamil Nadu. This was a prospective study that was carried out on 80 cases., including 60 pediatric patients diagnosed with chronic OME [resistant to medical treatment for >3 months (type B tympanogram)or recurrent after successful medical treatment]. Patients were subjected to a myringotomy with insertion of a tympanostomy tube (grommet tube). Their age ranged from 2-15 years. Twenty children had ENT disease other than OME, mostly adenoidal or tonsillar hypertrophy. The serum level of total IgE was assayed in patients and control groups as well as in the middle ear effusion of patients. The results showed that total IgE was higher in the serum of patients with OME than the serum of the control group; there was a high significant correlation between total IgE in serum and total IgE in effusion in patients with OME. Allergy is a possible risk factor for the development of pediatric OME.

INTRODUCTION

Otitis media with effusion (OME) is defined as the presence of fluid in the middle ear without the signs or symptoms of acute ear infection[1]. The most common complication of OME is hearing loss and it is the most common cause of hearing loss inchildren[2]. Long standing bilateral OME may cause long lasting cognitive and language problems in the affected child[3]. OME is proven to be a symptom of allergic inflammation associated with the releaseo eosinophilic cationic protein into the middle earmucosa and effusion in individuals who are universally allergic with a positive skin test[4].

Allergy is associated with increased middle eareffusion (MEE) volume and the middle ear mucosa can mount an antigen-immunoglobulin E (IgE)interaction[5]. The mechanism by which allergy might cause OME remains hypothetical and controversial, but may involve one or more of the following mechanisms: (a) middle ear mucosa functioning as a'shock (target) organ., (b) inflammatory swelling of the mucosa of Eustachian tube., (c) inflammatory obstruction of the nose and (d) aspiration of bacteria laden allergic nasopharyngeal secretions into the middle earcavity[6]. Hurst[7] described four factors to prove that chronic otitis media with effusion (COME) is an allergic disease. (a) To establish a clear, associated, objective diagnosis of atopy in patients with persistent OME. (b) To detect an association of allergic T helper2 immune mediated histochemical reactivity with in the middle ear. The concept that active immunologic processes may be localized processes in the middle earhas been established in animal models as well as in humans. (c) To prove that the middle ear is a target organf allergy, we need to establish that the inflammation within the middle ear is truly allergic in nature. This is what we did in our study by demonstration of IgEin MEE. Therefore, our study provides a part of the answer not the entire answer. (d) To prove the allergy connection requires direct evidence of a dose-response curve and consistency of results[7].

MATERIALS AND METHODS

This study was carried out in the period from June 2023-May 2024 in the Dept of ENT, Sree Mookambika Institute Medical Sciences, Kulasekharam, Tamil Nadu. This was a prospective study that was carried out on80 cases including 60 pediatric patients diagnosed with COME [resistant to medical treatment for >3 months (type B tympanogram; pneumaticotoscopy was performed for all patients) or recurrent after successful medical treatment]. Patients were subjected to myringotomy with insertion of tympanostomy tube (grommet tube). Their age ranged from 2-15 years (35 females and 25 males) and there were 20 children with ENT disease other than OME, mostly adenoidal or tonsillar hypertrophy., their age ranged from 3-12 years (14 females and six males).The serum level of total IgE was assayed in the patient and control groups as well as in the MEE of patients. Exclusion criteria were as follows: (a) acute otitis media.,(b) sensorineural hearing loss., (c) immunodeficiency diseases., (d) Down's disease., (e) cleft palate., (f) patients with gastric problems., (g) craniofacial abnormalities.,(h) chronic underlying medical disease. Patients and control groups were subjected to the following:

- **Assessment of history:** including history suggesting allergy such as symptoms suggesting allergic rhinitis, asthma, eczema, urticaria, conjunctivitis, food sensitivity and anaphylaxis. We selected children (patients) with a known history of allergic diseases (allergic rhinitis, asthma)
- **General examination**: to detect any underlying chronic diseases. For example, hepatic diseases that affect the total immunoglobulin concentration in blood to detect any allergic diseases
- Clinical otorhinolaryngologic examination
- Audiological evaluation in the form of pure tone audiometry and tympanometry Pure tone audiometry was performed to exclude sensorineural hearing loss and to detect conductive hearing loss from OME. Tympanometry was performed to diagnose OME (type tympanogram)
- **Laboratory investigation:** assay of total IgE by enzyme linked immunosorbent assay (ELISA)in a MEE fluid sample obtained from patients prepared for ventilation tube (grommet tube)insertion under general anesthesia by 5 ml widebore under complete aseptic conditions., MEE was obtained from both ears of the same case.
- A sample from both ears was considered a single sample. MEE samples from both ears were about 1-4 ml
- Assay of total IgE in serum by ELISA in the blood sample obtained by sterile venipuncture from both patients and controls.

The total IgE quantitative test kit is based on the principle of a solid phase ELISA. The assay system utilizes a monoclonal anti IgE antibody for solid phase (micro titer wells) immobilization and a mouse monoclonal anti IgE antibody in the antibody-enzyme (horseradish peroxidase)conjugate solution. The test sample is allowed to react with the solid phase antibodies., after incubation and washing, the enzyme

conjugate will be added, resulting in the sandwiched formation of IgE between the solid phase and the conjugated antibodies. After a second wash, a solution of tetra methyl benzidine is added and incubated for 15 min, resulting in the development of a blue color. The color development is stopped with the addition of stop solution and the color changes to yellow and is measured spectrophotometrically at 450 nm. The concentration of IgE is directly proportional to the color intensity of the test sample.

RESULTS AND DISCUSSIONS

The majority of patients in the patient group were females (58.3%), only 41.7% were males. The majority of patients in the control group were females (70%) and only 30% were males. There was no significant difference. The current research showed that the IgE in the serum of the ptients in the patient group was 26.4-712.4 IU/ml, with a mean value of 262.6±175.4 IU/ml, whereas IgE in the serum of the control group was 29.6-147.2 IU/ml, with amean value of 68.5±37.3 IU/ml. This study found a difference between patients and control in the mean total IgE in serum., there was a highly significant difference as P<0.0001.There is a very strong uphill linear pattern and a positive correlation. There is a highly significant correlation between total IgE in serum and total IgEin middle eareffusion.

The study was carried out to determine the role of allergy in the etiology of OME. OME is the presence of fluid in the middle ear without signs or symptoms of acute middle ear infection. The persistence of MEE for at least 3 months is defined as COME. It is a multi factorial disease and still remains the most common cause of deafness in children[7].Although OME continues to be one of the most prevalent childhood diseases, considerable controversy remains in terms of its pathogenesis. OME is primarilya chronic inflammatory condition and the causes of inflammation are multi factorial. Because conventional treatment modalities have failed to eliminate the complication of OME, further research must target the cause and prevention of OME[9].Recent guidelines from otologists, pediatricians and allergists on the basis of clinical evidence support the role of atopy in the development of OME. The involvement of IgE mediated allergic reactions in the pathogenesis of OME has been suggested by clinical observations of a high prevalence of OME among patients with allergies[10]. The important role of allergy in the genesis and recurrence of OME is also supported by the data in the literature that indicate statistically significant differences in audiological characteristics among atopic and nonatopic patients with OME. In fact, in atopic children, a predominance of bilateral OME and greater hearing impairment are found[11].The serum IgE concentration of normal individuals is10-100 IU/ml[12]. The IgE serum concentration in apatient is dependent on both the extent of the allergic reaction and the number of different allergens to which he/she is sensitized. Nonallergic normal individual shave IgE concentrations that vary widely and increase steadily during childhood, reaching their highest level at age of 15-20 years, there after remaining constant until about the age of 60 years, when they slowly decline[13].

The current research is in agreement with Passali[14], who found a relation between allergic rhinitis and the development of COME., their study was carried out on 100 atopic patients with COME followed for 6 months and in whom the allergen was detected. Hurst[7] postulated that COME is considered frequently to be an IgE mediated, late phase allergic disease. The significant incidence of atopy associated with OME has suggested a role of allergy in the pathogenesis of OME. Analysis of inflammatory mediators indicates that the mucosa of the middle ear can respond to antigen in the same way as does the mucosa of the lower respiratory tract. Recent characterization of the mucosa and effusion from atopic patients with OME shows T helper 2 cytokine and cellular profiles consistent with an allergic response, supporting the role of allergy in OME. In addition, animal studies indicate that inhibition of characteristic allergy cytokines can prevent the production of MEE. As the understanding of allergy and its role in the inflammation of OME continues to deepen, focused treatments of OME in the atopic population can be introduced[15].The results of Keles[16] showed that total IgE levels were significantly higher in the serum samples of the patient group compared with those of the control group. These results are in agreement with the current research. This study is in agreement with the findings of Alles[17] as they concluded that allergy was more common in a group of children with chronic or recurrent otitis media compared with the general population. This study is in agreement with McCoul[18], who reported that analyses of the effusion content have consistently shown significantly elevated level off allergy related mediators [interleukins (IL) 4,IL 5, IL 6, RANTES, ECP, tryptase, IgE] as well as differences between atopic and nonatopic patients with COME.

Nasal symptoms were more common in the OME group, which may reflect a higher prevalence of adenoidal hyperplasia. The study of Souter[19] was carried out on 89 children aged 6 or 7 years with OME confirmed intraoperatively during ventilationtube insertion between 2001 and 2005. The prevalence of allergic symptoms and nasal symptoms in children Otit is media with effusion Mohammed El Sharnoby[15] 5with OME was compared with an age

matchedreference group.The present study is also not in agreement with Cassano[20] as they reported that there is no correlation between allergic rhinitis and adenoid hypertrophy and OME., we found an increased risk of resistant andrecurrent OME with atopy, and increased serum IgEis usually associated with increased effusion IgE as itsupports allergy as one of the major risk factors in thepathogenesis of OME.This study used acute otitis media with impendingperforation to compare between the concentration of IgE as an indicator of allergy in the effusion of Chronic Otits Media with Effusionin the case of group 1 (OME) and effusion of acuteotitis media in group 2.

CONCLUSION

Allergy diagnosed by IgE plays a role in the developmentof Chronic Otits Media with Effusion. Total IgE is higher in the serum of patients with Chronic Otits Media with Effusion than in the serum of the control group.There is a significant positive correlation between total IgE in the serum and that in the middle eareffusion of patients with Chronic Otits Media with Effusion. Chronic Otits Media with Effusion may get benefit from treating andcontrol allergic disorders.

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