MIGRAINE HEADACHE IN A CHILD WITH HOUSE DUST AND MITE ALLERGY. 
A CASE REPORT

Anang Endaryanto, Ariyanto Harsono

ABSTRACT

Background: House dust mite is common aeroallergen. It can cause type 1 allergy (IgE-mediated) symptoms from dust inhalation with different spectrums of the disease. The relationship between migraine headache and aeroallergen has been questioned for many years. Objective: To report a case of migraine headaches in a child due to house dust and mite allergy and the course of this allergy after specific immunotherapy. Methods: Case report; routine skin testing and laboratory tests. Results: A 9-year-old boy with a 2-years history of migraine headaches every morning especially in the rainy season. Skin tests revealed positive reactions to commercial house dust extract, mite extract and Der p 1 extracts. An elevated total IgE level and specific house dust and mite IgE RAST found in the serum of patient. After 3 months of initial immunotherapy the symptoms decreased significantly. Conclusions: This case reported the rare case of childhood migraine headache due to house dust and mite allergy. This report demonstrates that without immunotherapy, a migraine headache did not yield symptoms improvement.

Keywords: migraine headache, house dust, mite, aeroallergen, type 1 allergy, immunotherapy, child

INTRODUCTION

Allergy to house dust mite is manifested predominantly as a Type 1 (IgE-mediated) reaction with different spectrums of the disease and mostly often result from its inhalation. Mites make up a large part of house dust allergens (Gore, 2004). Asthma and perennial allergic rhinitis therefore dominate the clinical picture. Mites of the species of Dermatophagoides and Euroglyphus feed on human skin danders which are particularly abundant in mattresses, bed bases, pillows, carpets, upholstered furniture and fluffy toys. Their growth is maximal under hot (above 20°C) and humid conditions (80% relative humidity). In fact, even though mites are present in the household all year round, there are usually have peak season (September/October to April/May) in many countries. Patients allergic to mites therefore have symptoms all year round but with a recrudescence during this peak period. Moreover, the symptoms of patients allergic to mites are aggravated when it is humid or in the rainy season. The prevalence of sensitization to mites in the general population is higher in humid regions (20-35%) than in dry ones (15%) (Arian, 2001)

Migraine is a complex neurological disorder that affects about 15% of the population and which can be disabling. In the last 10 years, much progress has been made in the understanding of the molecular basis of migraine, dictating new approaches to pharmacological treatment. Successful management of migraine headache involves identifying and avoiding headache triggers and using appropriate abortive or prophylactic treatment once migraine is recognized, but the pathophysiology of migraine is still largely unknown. The relationship between migraine headache and aeroallergen has been questioned for many years. There is some evidence shows that avoiding some allergens, such as inhalant or environmental allergen, can eliminate or abolish headache periods, without the need for continuous medication especially in pediatrics. (Ku, 2005).

The purpose of this paper is to report a case of migraine headaches in a 9 year old child due to house dust and mite allergy and the course of this allergy after specific immunotherapy.

CASE REPORT

First Visit

This 9-year-old boy was brought to the private practice for evaluation in December 2003. The mother stated that he had had migraine headache every morning.
especially in rainy season. In the last 5 years he also had had a tendency to recurrent cough, allergic rhinitis, urticaria, and eyelid angioedema. Headache symptom had always worsened for 5 to 6 months during the rainy season in the last 2 years. There was no history of asthma, known food allergy, or frequent infections.

The 3 episodes of headache symptoms had occurred 1 week before this visit. Originally, it was thought that food allergy was implicated because all of these episodes occurred around the breakfast time. The mother, however, had been unable to precipitate symptoms by repeated trials of the same foods consumed in the breakfasts. With investigating other possible causes, it had become apparent that these episodes had always occurred every morning, especially in the rainy season.

On questioning the mother regarding history of allergy, it was found that the patient had a tendency to urticaria, recurrent cough and allergic rhinitis since the infant period. If the allergy symptoms had appeared, the patient always visit to his pediatrician who informed his mother that the child had had allergy, and for which pharmacotherapy and allergen avoidance had been given regularly. Within a few days pharmacotherapy, the symptoms had always resolved. But after 2-3 days pharmacotherapy had finished, the symptoms occurred again. Since 2 years before this visit, different from the other symptoms which had been declined, the headache had become more severe and because of that, he was often absent from school.

The patient was a product of a normal pregnancy and delivery. Growth and developmental landmarks were normal. He was breast fed until the age of 3 months when a facial rash presumed to be “eczema” occurred. Soy milk feeding was begun; a salve was prescribed for the face and within 3 weeks the rash disappeared. Thereafter, there was no recurrence of this rash, nor were there ever any skin lesions suggestive of atopic dermatitis. Soy milk was continued for approximately 6 months then cow's milk was given to replace it. He tolerated this well. Processed baby foods were introduced at about 6 months of age, and eventually table foods were added with no adverse effects.

Family history revealed that the father had had an allergic rhinitis and his sister, age 4 years, had had pruritic rashes with dog contact. Environmental review was unremarkable except for the presence of feather pillows and numerous stuffed animals on the patient's bed.

On physical examination he was alert, pulse rate was equal and regular at 110 beats/minute, respiratory rate was 28 times/minute, axillary temperature was 37oC, the blood pressure was 120/80 mmHg, and well nourished. There were no sign of anemia and jaundice on eyes examination. There was enlarged nasal turbinate. On inspection of the chest, there was no precordial bulging, ictus cordis was normal, palpable at fifth intercostals space and left mid-clavicle line. On auscultation, the first and second heart sound was normal and no murmur was heard. The breath sound was vesicular, without rales, friction rub and wheezing. Bowel sound was normal. Liver and spleen were not palpable. Examination of extremities showed hypopigmented areas of skin on the dorsum of the hands, arms, and maxillae (subsequent dermatology consultation established the diagnosis of vitiligo). BCG scar was positive.

The laboratory investigation revealed normal complete blood count with significant eosinophilia; erythrocyte sedimentation rate (ESR) and quantitative total and specific immunoglobulin (Ig) E, were high for his age. The chest radiograph was normal; and the spirometry was normal.

**In Vitro Tests**

The numbers of eosinophils circulating in the peripheral blood was 372 eosinophils/uL of blood. The patient's serum was also tested with the full strength extract by the polystyrene tube radioimmunoassay. An elevated total IgE level (1785.2 IU/ml) and specific house dust and mite IgE RAST (1.05 IU/ml and 0.85 IU/ml) found in the serum of patient.

**In Vivo Tests**

Skin testing in this patient was performed using the scratch technique. A positive and negative control test using histamine and saline, respectively, was performed. Skin test reactivity of the house dust, mite and Der p 1 extracts was considered positive if the following criteria were met: (1) erythematic of at least 5 to 10 mm, or (2) a wheal and erythematic reaction of at least 2 to 3-mm wheal and a 5 to 10-mm erythematic, and (3) control testing with diluents, negative and skin testing with the extracts in control individuals, negative. Scratch skin testing to inhalants revealed very strong positive reaction to house dust, mixture of mite and Der p 1 allergen extracts (erythema.wheal: 30mm.8mm, 28mm.7mm and 23mm.7mm respectively). Scratch skin testing to commercially prepared food extracts
resulted weak positive reaction to cow's milk erythema wheal: 5 mm. 2 mm).

Treatment consisted of as needed antihistamine; house dust control and removal of feather pillows were suggested, as was the avoidance of the ingestion of known milk-containing foods. The patient was followed for approximately 3 weeks.

**Return Visit**

The patient returned in three weeks after first visit. Since his first visit, he had had no significant recurrent cough, allergic rhinitis urticaria, and eyelid angioedema, however the complain of headache had not decrease and he had still frequently absent from school. He had no known allergic reactions to any food. Because there was no clinical improvement achieved, the specific immunotherapy for house dust allergy was given.

After 3 months of specific house dust immunotherapy, there has been improvement of symptoms. During this period of treatment, the headaches complain dramatically decreased and his absence from school became rarer. The numbers of eosinophils circulating in the peripheral blood, total IgE level, and specific house dust-mite IgE RAST level were done after 12 month immunotherapy. The symptoms and laboratory parameter at 0 and 12 month treatment can be seen in Figure 1 and Figure 2 below:

![Graph](image-url)

Figure 1. The numbers of eosinophils of the peripheral blood, the serum concentration of total IgE and the serum concentration of specific house dust IgE RAST at 0 and 12 months therapy.
Migraine Headache in A Child with House Dust and Mite Allergy

DISCUSSION

Sensitization to environmental allergens from indoor and outdoor sources requires more time and is generally observed between the first and tenth years of life. The annual incidence of early sensitization depends on the amount of exposure. In a longitudinal birth cohort study in Germany (MAS) a dose-response relationship could be shown between early exposure to cat and mite allergens and the risk of sensitization during the first years of life (Wan, 2004). It has been demonstrated recently that strong infantile IgE antibody responses to food proteins have to be considered as markers for atopic reactivity in general and are predictors of subsequent sensitization to aeroallergens. As far as clinical symptoms are concerned, atopic dermatitis in general is the first manifestation, with the highest incidence during the first 3 months of life and the highest period prevalence during the first 3 years of life. Atopic manifestations are known to differ from patient to patient with regard to end organ involvement (Wan, 2004). Our patient's history exemplifies essentially all the variations mentioned above. At the age of 3 months, he probably had mild eczema, which was short lived. At age 4 years he had rhinitis; at age 7 years he developed allergic reactivity to the house dust with the main complain of migraine headache.

This report demonstrates that pharmacotherapy and allergen avoidance in a case of migraine headaches caused by house dust and mite allergy was not yield a symptoms improvement without specific immunotherapy. As we know, the response of sensitive host to the house dust and mite allergen is very unique. Wan et al, 1999 reported that epithelial permeability is changed non selectively by house dust mite and pure Der p 1, and make the disruption of epithelial Tight Junction (TJ). Furthermore, and of significance for allergen presentation, the increased permeability following TJ cleavage facilitated the Trans epithelial permeation of Der p 1. The non selectivity of the increase implies that the Trans epithelial permeation of all proteins would be enhanced. This would lead to an increased probability of any inhaled allergen being able to encounter antigen-presenting cells of the airway's immune system at sites where epithelial permeability was increased (Wan, 1999). From other literature, we know that allergy to Der p 1 is frequently associated with reactivity to unrelated allergens (Pollard 1989; Gelber, 1993; Sear, 1989). Several observations suggest that allergic sensitization may be promoted by any environmental proteinase that attacks, directly or indirectly, the integrity of the epithelial barrier. Therefore the prevention of TJ disruption (by inhibiting the environmental proteinase or even promoting TJ reassembly/assembly) could provide an entirely new
approach to the treatment allergy by limiting exposure of the immune system to allergens (6-8). The improvement of symptoms by specific immunotherapy may be due to its capacity in the prevention of TJ disruption by increase of the allergen-specific IgG4 and the regulatory T cells secreting IL-10 which produces IgA (Nelson, 2005).

The patient had had migraine headache every morning, especially in the rainy season. This condition may be caused by the higher humidity in the rainy season. The growth of Dermatophagoides and Euroglyphus is maximal under hot (above 20°C) and humid conditions (80% relative humidity). In fact, even though mites are present in the home all year round, there are usually have peak in rainy season (September/October to April/May) in tropical countries. (2) Moreover, the symptoms of patients allergic to mites are aggravated when it is humid. Ku et al, 2005 from their study reported that there was a high prevalence of migraine headaches in patients with allergic rhinitis due to Aeroallergen compared to those without. They propose that histamine and other allergic mediators could play a key role in the pathogenesis of migraine headaches by way of vasodilatation and inflammation (Trotsky, 1994; Gazerani, 2003).

After 3 months specific immunotherapy was given, there had been significantly improvement of symptoms. The numbers of eosinophils circulating in the peripheral blood, ESR, IgE RAST level and diameter of scratch skin testing were decreased significantly after 12 months immunotherapy. A mechanism underlying allergen immunotherapy was suggested as a result of increases in allergen-specific IgG4, which blocks not only IgE-dependent histamine release from basophile but also IgE-mediated antigen presentation to T cells. Immunotherapy also acts on T cells to modify peripheral and mucosal TH2 response to allergen in favor of TH1 responses. Recent studies have identified regulatory T cells secreting IL-10, which produces suppression of masts cell, eosinophil, and T-cell responses and acts on B cells to favor heavy-chain class switching to IgG4.

REFERENCES


