Correlation between Density of House Dust Mites and Relapse Rate of Atopic Dermatitis: A Cross Sectional Study

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Abstract: Background: Inhaled allergens especially house dust mites (HDM) greatly contribute to the occurrence of atopic dermatitis (AD). It is supported by research conducted by Collof (1992) that measured the density of HDM on mattresses of AD and mattresses of non atopic healthy people and the result of HDM density on mattresses of patients with AD is higher than non atopic healthy controls. Teplitsky et al (Jerusalem, 2008) reported that patients with AD showed a higher prevalence of mites on their skin than did healthy individuals, which could be involved in allergic sensitization and disease exacerbation. The present study aims to investigate the correlation between density of HDM and relapse rate of AD, confirmed with skin prick test (SPT) of HDM. Methods: In present study 30 participants were included. Dust from each bedroom was collected. The density of HDM was calculated while the participants were checked their sensitivity of HDM by SPT. Establishing the diagnosis of AD based on Hanifin-Radja criteria. Each participant was asked about recurrence of AD in the last three months. Results: Seven (23.3%) samples had moderate density of HDM while 23 (76.7%) had low density of HDM. From anamnesis, 10 (33.3%) participants were diagnosed with AD and SPT showed that 50% samples were sensitive against HDM. Out of 10 AD participants, 8 (80%) were positive with HDM allergens. All 10 participants with AD had relapse within three months. Conclusion: Out of 10 participants with atopic dermatitis, two (20%) had moderate density of HDM in bedroom (p>0.05). Also, from seven samples with moderate density of HDM, three (20%) were sensitive against HDM (p>0.05). About 80% of AD showed sensitivity against HDM while all 10 AD participants had relapse within last three months indicated that HDM allergen could induce exacerbation of disease in AD, but the correlation between HDM density and the relapse rate could not be proven yet. This present study suggests further investigation with larger samples size.

1 INTRODUCTION

Atopic dermatitis (AD) is a chronically relapsing skin disease that occurs most commonly during early infancy and childhood (Leung et al., 2012). This disease usually begins in the early life and is often seen in the people with the personal or family background of asthma or swelling of the mucous membranes. Forty five percent of children experience the early onset in the first 6 months of their life, 60% during their first year and 85% before the age of 5. This disease can assist with asthma and allergic rhinitis. This disease can cause sleep, educational, and social disorders in patients (Leung et al., 2012; Norris et al., 1998).

AD is a highly pruritic inflammatory skin disease that results from complex interactions between genetic susceptibility genes resulting in a defective skin barrier, defects in the innate immune system, and heightened immunologic responses to allergens and microbial antigens (Leung et al., 2012). Sensitization to inhalant allergens such as house dust mite (HDM) allergens, detectable with specific IgE tests, is very common in adolescent and adult patients suffering from AD (Werfel et al., 2006; Gavino et al., 2008). A T-cell-mediated reaction is critical in the worsening of eczema, which can be triggered by the epicutaneous application of HDM allergens in sensitized patients. HDM allergens penetrate the skin where they are trapped via specific IgE on high-affinity Fc-receptors on Langerhans cells. Langerhans cells may subsequently present the allergens to T lymphocytes, leading to specific T-cell proliferation and eczema (Werfel et al., 2006; Gavino et al., 2008; Leung et al., 2012).

To cause transdermal sensitization, HDM
Allergens must be absorbed through the skin, processed by dendritic cells, and presented to T-helper lymphocytes. For efficient absorption, close and prolonged contact between the skin and HDM allergens is probably necessary, for example, via HDM-contaminated clothes and bedding (Plattsmills & Chapman, 1993). Accordingly, several studies have reported that the homes of patients with AD contain large amounts of HDM and their antigens, compared with controls, and that clothes and bedding are an important source of HDM (Teplitsky, 2008).

The aim of the present study was to determine the number of HDM and to correlate its density with incidence of atopic dermatitis, also comparing with skin sensitivity towards HDM by conducting skin prick test (SPT).

2 METHODS

The study samples were 30 children and adolescent of Al Falah and Aisyiyah Reformatory in Padang, with aged range 8 to 23 year old. Ethical approval was obtained and all participants were explained about the purpose of the study and also were informed about the procedure of SPT and the informed consent was obtained from each of the participant. The following data were collected: demographic data for age, sex, personal and family history of atopy. Diagnosis of atopic dermatitis was established based on Hanifin-Rajka criteria.

2.1 Dust Collecting and Counting

Dust was sucked with a vacuum cleaner. At the end of the vacuum cleaner was installed a chiffon cloth to catch the inhaled dust. Suction was done for 3 minutes on each mattress in the reformatory. Dust inserted to plastic containers. The container was labeled according to where the dust was taken. The dust samples were then taken to the Parasitology Laboratory of the Faculty of Medicine, Andalas University for examination.

Dust in a plastic container that had been labeled was filtered using a sieve. Strained dust was put in a petri dish that has been previously weighed to determine the weight of an empty petri dish. Dust was weighted and noted each weight. Weighted dust was put into a container containing 100 ml of 5% sodium chloride solution. The solution was put into a reaction tube. This solution was centrifuged for 4 min at 600 rpm for separating mites from debris. A saturated NaCl solution is added to the reaction tube until it was full and the surface of the convex solution appears. Then covered with cover glass (deck glass) and left for 30 minutes. Cover glass was taken and placed on the object glass. Samples were examined using a 40x light magnification microscope. The number of HDM found was calculated.

2.2 Skin Prick Testing

The medial aspect of the forearms and the upper arms were cleaned and test sites for placing the allergens were marked using a marker 2-3 cm away from the wrist and ante cubital fossae. Distance between two allergens was kept at 2 cm to avoid false positives either due to direct contamination or due to axon reflex. A drop of each allergen was placed on the skin and was pricked with a lancet to introduce the allergen. Equal pressure was applied for all the allergens. Histamine dichloride (10 ng/ml or 0.1%) was used as a positive control and saline as negative control. Results were read after 20 minutes. Wheals at the test site were compared with the wheal produced at the positive control site. Largest diameter of the wheal was measured using a plastic scale provided along with the test kit. A wheal of 50% of diameter positive control or bigger was considered as positive. Negative control was used to rule out any dermographism.

2.3 Statistical Analysis

Data obtained was analyzed using SPSS vr.15.0.

3 RESULTS

We conducted 30 participants which 10 of them have met the Hanifin-Rajka criteria. Seven dust samples had moderate density of HDM and other 23 samples with low density of HDM. From seven samples of moderate density of HDM, only one that have a history of urticarial (p>0.05). 15 participants were sensitive to HDM (positive SPT), and three of them had moderate density of HDM (p>0.05).
From anamnesis, we found that all 10 AD participants had relapse in the last three months. And out of 10 AD participants, eight samples (80%) were sensitive against HDM allergen.

4 DISCUSSION

The house dust mites are the most common environmental allergens. Mites sensitize and induce allergic disorders such as perennial rhinitis and asthma in predisposed individuals. In addition, house dust mites are important deteriorating factors in patients with atopic dermatitis (Tupker et al., 1998).

Although definite causality has yet to be determined, it has been alleged that HDMs play a role in the immunopathogenesis of AD. Not only do AD patients have elevated levels of serum IgE antibodies specific to HDM allergens, biopsy specimens of AD lesional skin have also been shown to be infiltrated with T lymphocytes that recognize HDM. It has been shown that HDM may facilitate its entry into AD skin by enzymatically breaking down the epidermal barrier. Mite allergens are able to activate keratinocytes and induce them to produce and secrete proinflammatory cytokines (Gavino et al., 2008).

Briefly, the prevalence of allergic diseases due to household arthropods have significantly increased in the recent last decades, because people spend most of their time in their home environment and according to the modern lifestyle, houses are warmer and filled with a lot of furniture and not enough air-regulation is provided (Ziyaei et al., 2017). In this study, we determined the correlation between density of HDM with the incidence of atopic dermatitis.

Out of 10 participants with atopic dermatitis, two (20%) had moderate density of HDM in bedroom (p>0.05). Also, from seven samples with moderate density of HDM, three (20%) were sensitive against HDM (p>0.05). There was no evidence to support the first assumption, as no difference in incidence of atopic dermatitis with HDM density that was counted from patients’ clothes and bedding, although an earlier study had reported there were larger numbers of HDMs in the mattresses of patients with AD when compared with controls. However, all AD

<table>
<thead>
<tr>
<th>HDM DENSITY</th>
<th>INCIDENCE OF ATOPIC DERMATITIS</th>
<th>P value</th>
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<tbody>
<tr>
<td></td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>HIGH</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>MODERATE</td>
<td>2 (20.0%)</td>
<td>5 (25.0%)</td>
</tr>
<tr>
<td>LOW</td>
<td>8 (80.0%)</td>
<td>15 (75.0%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>12 (100%)</td>
<td>18 (100%)</td>
</tr>
</tbody>
</table>

Table 1. Correlation between HDM density with incidence of atopic dermatitis

<table>
<thead>
<tr>
<th>HDM DENSITY</th>
<th>SKIN PRICK TEST</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>POSITIVE</td>
<td>NEGATIVE</td>
</tr>
<tr>
<td>HIGH</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>MODERATE</td>
<td>3 (20%)</td>
<td>4 (26.7)</td>
</tr>
<tr>
<td>LOW</td>
<td>12 (80%)</td>
<td>11 (73.3%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>15 (100%)</td>
<td>15 (100%)</td>
</tr>
</tbody>
</table>

Table 2. Correlation between HDM density with sensitivity of HDM
participant had relapse within three months while 80% of them positive to SPT indicates that HDM allergen might induce the recurrence of disease.

5 CONCLUSION

Even though recent evidence supports a role for HDM in atopic dermatitis, the correlation of its density with prevalence of occurrence of atopic dermatitis still needs further research with bigger sample size.

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REFERENCES


