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Full Length Research Paper

Allergic rhinitis clinics caused by house dust mites (HDM) and pollens

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The aim of this study was to compare allergic rhinitis clinics caused by two most common allergens: house dust mites (HDM) and pollens. Three hundred and fifty patients were evaluated. These patients were pure pollen or HDM allergic according to skin prick test (51 HDM+, 299 Pollen+). Mainly, HDM were allergens for perennial allergic rhinitis, while pollens were for seasonal allergic rhinitis ($p < 0.01$). Both groups were found with similar symptom frequency except palatal, ocular, throat itching and eye redness indicating mostly pollen allergy ($p < 0.05$). Seasonal exacerbations used for the differential diagnosis were found to be very significantly different. Spring and autumn were the seasons where pollen allergy symptom exacerbation was mainly seen ($p < 0.01$) . HDM allergy was uniquely found with symptom exacerbations in winter ($p < 0.05$). Rural area visit was found dominating triggering factor for pollen allergy ($p < 0.05$). The most common triggering factor was house dust exposure in HDM+ group ($p < 0.05$). HDM allergy being mostly mimicking pollen allergy in allergic rhinitis, however, differs from it with some clinical features. This could be detected with detailed history taken from the allergic rhinitis patients. While doing definitive diagnosis, prick test may be helpful with a clear patient history in patients hard to diagnose.

Key words: Mite, pollen, allergy, rhinitis.

INTRODUCTION

Rhinitis consists of allergic and non-allergic subgroups. Allergic rhinitis (AR) is one of the most common airway diseases that results from the inflammatory reaction mediated by specific IgE antibodies and is manifested after the exposure of the nasal mucosa to allergens (Obtułowicz and Składzie , 2005). 10 to 25% of the population suffers from AR (Valero et al., 2009). Patients suffering from specific rhinitis symptoms (nasal obstruction, rhinorrhea, sneezing) with positive allergic findings by either specific IgE or SPT are diagnosed as AR. Nasal provocation test is another technique to confirm the diagnosis (Chusakul et al., 2010). Allergens are divided into two subgroups: indoor and outdoor allergens. The mold, house dust mites (HDM), pets and cockroaches are kinds of indoor and outdoor allergens including pollens especially (Ferguson, 2008; Huss et al., 2001; Rosenstreich et al., 1997). The most common sensitizing allergen in the United States is HDM (Ferguson, 2008). Pollen calendars differ from one geographic area to another while pollens usually increase in spring and decrease in autumn (D'Amato et al., 1998).

HDM have been shown to be important sources of indoor allergens associated with asthma and other allergic conditions, especially AR. HDM are very small creatures that live indoors in warm, moist places. These creatures are not a type of bugs that you can spot and crush, being invisible to the unaided eye. Their translucent bodies further hinder their visibility. HDM have no eyes or antennae, just eight legs, and a mouth-like appendage. The most common dust mite species around the world include *Dermatophagoides pteronyssinus* (Dp), *Dermatophagoides farinae* (Df), *Euroglyphus maynei* and *Blomia tropicalis*. The pyroglyphid mite Dp is distributed from temperate to tropical regions (mainly in Europe). Other important pyroglyphid mite is Df found in drier regions (mainly in North America) (John and Petri, 2006). These microscopic arachnids colonize beds, upholstered furniture and carpets (Arbes et al., 2003). Because mites do not drink and rely on absorption of humidity from the atmosphere, reducing humidity below 50% is recommended as the primary prevention method. HDM do not bite, but live from shed human skin. Because of the medical implications, house dust and the fauna of mites associated with house dust have been tested for the source of the house dust allergen. Mite allergens are mainly present in feces of house dust mites and may become airborne, inhaled by patients, giving rise to asthma, rhinitis or atopic dermatitis (van Bronswijk et al., 1990). HDM allergic patients are also shown to have poorer psychological functioning, indicating the close relationship between moderate-to-severe persistent AR and psychological functioning (Lv et al., 2010).

Allergy clinics' algorithms for the diagnosis of allergic rhinitis consist of two powerfull tools: patient history and skin prick tests. As the most common allergens are HDM and pollens, the large part of the patients need to be differentiated by these tools. Besides, some patients may be allergic to both groups of allergens. The dominating allergen could only be found according to the clinic of the patient, and then further correct therapy methods could be chosen. The prevention from the allergen and allergen-specific immunotherapy should be applied against the clinically dominating allergen. The aim of this study was to compare allergic rhinitis clinics caused by two most common allergens as HDM and pollens. Also the discrepancies between the demographic findings of HDM and pollen allergic patients were discussed.

MATERIALS AND METHODS

Three hundred and fifty patients were evaluated. These patients were pure pollen or HDM allergic according to skin prick test (SPT). Patients were diagnosed with both detailed allergic history (clinical symptoms, season of the year, triggers, family history) and the SPT. In the HDM allergic group, there were 51 patients (20 male, 31 female) aged 31.8 ± 12.4 years (9 - 59 years); whereas pollen allergic group consisted of 299 patients (127 male, 172 female) aged 28.8 ± 12.5 (6 - 65 years).

Nasal congestion/obstruction, rhinorrhoea, sneezing, headache, postnasal drip, loss of smell (hyposmia or anosmia), nasal, pharyngeal, palatal itching and ocular symptoms (itching, redness, tearing) were questioned at the first visit to the clinic. Patients with rhinorrhoea were also asked about their nasal discharge characteristics (viscosity and color). Nasal examination was done by both anterior rhinoscopy and nasal endoscopy.

Skin prick tests were performed with multi-test applicators (Multi-Test® II device, Lincoln Diagnostics, Inc Illinois, USA) and the standardized allergen extracts solutions with negative (physiologic saline/0.4 % phenol) and positive (histamine 1+999 w/v/0.4 % phenol) controls (Allergopharma, MERCK KGaA, Darmstadt, Germany). The allergy panel, complying with allergy profile in the south of Turkey, consisted of both indoor and outdoor allergens: Grasses and cereals (velvet grass, orchard grass, rye-grass, timothy grass, bluegrass, meadow fescue, barley, oat, wheat, rye), weeds (mugwort, stinging nettle, dandelion, English plantain), trees blossoming both early (alder, hazel, poplar, elm, sallow) and mid (birch, European birch, oak, plane), mites (*D. farinea* and *D. pteronyssinus*).

We defined a reliable SPT in which the median wheal diameter of the positive control is more than 3 mm. We included patients with positive SPT which the median wheal diameter of the skin reaction is 3 mm. Non-allergic and mixed allergic (pollen + HDM) rhinitis patients were excluded. Groups were defined as purely HDM allergic patients (HDM+) and pollen allergic patients (Pollen+).

Normal distribution of the groups and homogeneity of variances were analyzed by Kolmogorof-Simirnov and Levene tests, respectively. Student t-test in independent samples with equal variances, and multiple chi-square tests were used for the comparison of groups. Statistical significance was determined as $p < 0.05$.

RESULTS

There were 17 (33.3%) students, 6 (11.8%) housewives, 1 (2%) retired and 27 (53%) working individuals in HDM+ group. Pollen+ group included 115 (38.5%) students, 40 (13.4%) housewives, 7 (2.3%) retired, 3 (1%) unemployed and 134 (44.8%) employed individuals (Table 1). There was no significant difference between groups according to their way to continue their life. Three (5.9%) patients of HDM+ group were diagnosed as seasonal and 48 (94%) as perennial allergic rhinitis. Seven patients (2.3%) of the Pollen+ group were diagnosed as perennial allergic rhinitis and remaining 292 (97.7%) as seasonal allergic rhinitis. This distribution was significantly different as HDM allergy causes perennial allergic rhinitis; and pollen allergy causes the seasonal allergy ($p < 0.01$) . Symptom distribution of subgroups was given in Table 2. Palatal, ocular and throat itching as well as eye redness were more prominent symptoms in Pollen+ group ($p < 0.05$).

Time period since the beginning of allergic symptoms ranged from 1 to 16 years (mean 6 ± 4.6 years) in HDM+ group and 1 to 25 years (mean 5 ± 4.2 years) in Pollen+ group. There was no statistically significant difference between two groups about the time of the disease history ($p > 0.05$).

Although 48 of 51 (94%) HDM allergic patients gave perennial rhinitis history, 20 (39.2%) had exacerbations in spring, 8 (15.7%) in winter and 5 (10%) in summer. 182 (60.9%) patients in pollen group had exacerbations in spring, 61 (20.4%) in summer, 34 (11.4%) in autumn and 18 (6%) in winter. This was statistically different (Table 3). There was no statistical difference in the exacerbations of the symptoms in the morning between the two groups ($p > 0.05$).

Environmental triggering factors were effective in 115

Table 1. Occupational distribution of subgroups.

	HDM+	Pollen+	P
Student	17 (33.3%)	115 (38.5%)	p > 0,05
Housewife	6 (11.8%)	40(13.4%)	p > 0,05
Retired	1 (2%)	7(2.3%)	p > 0,05
Unemployed	0 (0%)	3 (1%9	p > 0,05
Employed	27 (53%)	134 (44.8%)	p > 0,05

Table 2. The frequency of the symptoms in subgroups.

	HDM+		Pollen+	
	Number	%	Number	%
Nasal congestion	33	64.7	211	70.6
Nasal discharge	43	84	256	85.6
Sneezing	46	90.2	279	93.3
Headache	16	31.3	93	31.1
Postnasal drip	26	50.9	133	44.4
Smelling disorder	9	17.6	81	27
Nasal itching	35	68.6	207	69.2
Palatal itching*	11	21.5	128	42.8
Throat itching*	17	33.3	144	48.1
Ocular itching*	21	41.1	190	63.5
Eye redness*	16	31.3	151	50.5
Eye tearing	30	58.8	184	61.5

*p < 0.05.

Table 3. Seasonal exacerbations.

	HDM+		Pollen+		p
	Number	%	Number	%	
Spring	20	39.2	182	60.9	P < 0.01
Summer	5	9.8	61	20.4	P > 0.05
Autumn	0	0	34	11.4	P = 0.011
Winter	8	15.7	18	6	P < 0.05

(38.5%) patients in pollen group whereas there were only 8 (15.7%) patients in HDM+ group ($p < 0.05$). House dust exposure was found as triggering factor mostly in HDM+ group ($p < 0.05$). There was no statistical difference between two subgroups about other triggering factors like thermal changes, smell and detergents.

There was not a statistical difference between two subgroups about allergic family history ($p > 0.05$). Incidence of concomitant allergic diseases (skin allergy, allergic eye disease, allergic lung disease) was similar in both groups ($p > 0.05$).

Papule diameter of positive controls ranged from 3 to 8 mm (mean: 5.8 ± 1.3) in HDM group and 4 - 19 mm (mean: 6.2 ± 1.8) in pollen group ($p > 0.05$). Erythema diameters ranged from 5 to 30 mm (mean: 15.5 ± 7) in HDM group and 5 to 35 mm (mean: 16.4 ± 6.7) in pollen group ($p > 0.05$).

DISCUSSION

Both pollen and HDM are the most common causes of allergic rhinitis. Pollens as main allergens in allergic rhinitis were widely discussed in the literature. Whereas, HDM are the main allergens of indoor environment. These two groups are worth comparing both clinically and demographically. Using purified groups, we augmented the scientific value of this study to eliminate other contributing factors in the diagnosis. The clinical differences may help to distinguish these patients before the definitive allergy tests. This paper is a summary of the similar and different findings of two groups.

Demographic findings as age, sex distributions and occupational conditions were found similar in two subgroups. Family history and associated allergic disease were not different for two groups. These findings indicated that both allergens work on the same pathogenesis ways.

Mainly, HDM are the allergens for perennial allergic rhinitis, while pollens are for seasonal allergic rhinitis. Both groups were found with similar symptom frequency except palatal, ocular and throat itching and eye redness indicating mostly pollen allergy. Both pollens and HDM were thought to exacerbate rhinitis symptoms in the morning. Patients having close contacts with HDM during the night may wake up in the morning with running nose, sneezing and nasal blockage. Pollens were also at a higher level in the air at early morning possibly causing more symptoms at this interval. Thus, we could not find any difference for morning symptom exacerbations between the groups. However, seasonal exacerbations used for differential diagnosis were found to be very significantly different. Spring and autumn were the seasons where pollen allergy symptom exacerbation was mainly seen. Sahney et al. (2008) reported pollen calendar of their country. In their report, the most frequent pollination season was spring for trees and flowers and autumn for grasses. Atmospheric pollen concentrations are at the highest level in April, May and August in our country (Erkara, 2008; Guvensen and Ozturk, 2003). This may be contributed to air temperature, humidity, and wind changes. HDM allergy was uniquely found with symptom exacerbations in winter. The highest concentrations of HDM are seen in November and December (Sun and Lue, 2000). This can be associated with humidity and climate conditions of winter. Also, people spend time mostly in indoor areas at this season and exposure to HDM increases. There was no statistical difference about allergic exacerbations between subgroups in summer ($p > 0.05$).

Rural area visit was found dominating triggering factor for pollen allergy. The most common triggering factor was house dust exposure in HDM+ group. There was no statistical difference between two subgroups about other triggering factors like thermal changes, smell and detergents indicating that both groups have similar associated nasal hyperreactivity.

Mites shed an abundance of allergenic proteins. Particularly abundant in allergens are the extracts of mite faeces as well as the extracts of their purified bodies or culture substrate (Thomas et al., 2004) . Some of the gut enzymes (notably proteases) produced by the house mite persist in their fecal matter, and can be strongly allergenic. Using different means of assessing exposure to house dust mites (Acarex -test, major allergen (Der p 1 + Der f 1, Der p 2 + Der f 2), content measured by ELISA) has allowed better identification of mite reservoirs and allergens in patients' homes (Pauli et al., 2001). In this study, HDM allergy was detected with purified allergens using skin prick test.

There is convincing evidence that avoidance of mite allergen can effectively reduce allergic symptoms. Patients can be moved to a mite allergen-free environment, or mite and mite allergen abatement can be performed to reduce exposure in existing residences. House dust mite control measures are based on the cognition of factors contributing to mite development, especially indoor relative humidity. Mite allergen avoidance strategies include 3 different methods: avoidance of mite producing allergens, elimination of mite reservoirs, especially textile reservoirs, dwellings designed so as to inhibit mite proliferation (Pauli et al., 2001; Sheikh and Hurwitz, 2003).

In recent years, greater attention has been given to the role of indoor allergens as a cause of sensitization and allergic respiratory diseases. Although indoor allergen control measures to reduce symptoms in individuals allergic to mites have produced controversial results. Exposure to high indoor aeroallergen levels, especially to house dust mite allergens, is an important environmental risk factor for allergic sensitization. Effective house dust mite allergen avoidance will never be achieved using a single control measure; various methods are required to affect the multiple factors that facilitate high indoor allergen levels. Education of the patients and their families is also an important component of environmental control strategies.

As a conclusion, HDM allergy being mostly mimicking pollen allergy in allergic rhinitis, however, differs from it, with some clinical features. This could be detected with detailed history taken from the AR patients. Recommendation of specific preventive methods to patients could be possible with correct differential diagnosis methods. Definitive diagnosis with prick test may be helped by a clear patient history in hard to diagnose patients.

REFERENCES

Arbes SJ Jr, Cohn RD, Yin M, Muilenberg ML, Burge HA, Friedman W, Zeldin DC (2003). House dust mite allergen in US beds: results from the First National Survey of Lead and Allergens in Housing. J. Allergy Clin. Immunol., 111(2) : 408-414.

Chusakul S, Phannaso C, Sangsarsri S, Aeumjaturapat S, Snidvongs K (2010). House-dust mite nasal provocation: a diagnostic tool in perennial rhinitis. Am. J. Rhinol. Allergy, 24: 133-136.

D'Amato G, Spieksma FT, Liccardi G, Jäger S, Russo M, Kontou-Fili K, Nikkels H, Wüthrich B, Bonini S (1998). Pollen-related allergy in Europe. Allergy, 53(6): 567-578.

Erkara IP (2008). Concentrations of airborne pollen grains in Sivrihisar (Eskisehir), Turkey. Environ. Monit. Assess., 138(1-3): 81-91.

Ferguson BJ (2008). Environmental controls of allergies. Otolaryngol. Clin. North Am., 41(2): 411-417.

Guvensen A, Ozturk M (2003). Airborne pollen calendar of Izmir - Turkey. Ann. Agric. Environ. Med., 10(1): 37-44.

Huss K, Adkinson NF Jr, Eggleston PA, Dawson C, Van Natta ML, Hamilton RG (2001). House dust mite and cockroach exposure are strong risk factors for positive allergy skin test responses in the Childhood Asthma Management Program. J. Allergy Clin. Immunol., 107(1): 48-54.

John DT, Petri WA (2006). Arthropods and human disease. In: Markell and Voge’s Medical Parasitology. Ninth Edition. Saunders, Elsevier. pp. 322-351.

Lv X, Han D, Xi L, Zhang L (2010). Psychological Aspects of Female Patients with Moderate-to-Severe Persistent Allergic Rhinitis. ORL J. Otorhinolaryngol. Relat. Spec., 72: 235-241.

Obtułowicz K, Składzie J (2005). Allergia rhinosinusitis. Diagnosis, programming and treatment monitoring. Przegl. Lek., 62(12): 1475-1479.

Pauli G, de Blay F, Ott M, Bessot JC (2001). Mites and their allergens: identification and extermination methods Allerg. Immunol. (Paris), 33(8): 333-335.

Rosenstreich DL, Eggleston P, Kattan M, Baker D, Slavin RG, Gergen P, Mitchell H, McNiff-Mortimer K, Lynn H, Ownby D, Malveaux F (1997). The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. N. Engl. J. Med., 336(19): 1356-1363.

Sahney M, Chaurasia S (2008). Seasonal variations of airborne pollen in Allahabad, India. Ann. Agric. Environ. Med., 15(2): 287-293.

Sheikh A, Hurwitz B (2003). House dust mite avoidance measures for perennial allergic rhinitis: a systematic review of efficacy. Br. J. Gen. Pract., 53(489): 318-322.

Sun HL, Lue KH (2000). Household distribution of house dust mite in central Taiwan. J. Microbiol. Immunol. Infect., 33(4): 233-236.

Thomas WR, Smith WA, Hales BJ (2004). The allergenic specificities of the house dust mite. Chang Gung Med. J., 27(8): 563-569.

Valero A, Pereira C, Loureiro C, Martínez-Cócera C, Murio C, Rico P, Palomino R, Dávila I (2009). Interrelationship between skin sensitization, rhinitis, and asthma in patients with allergic rhinitis: a study of Spain and Portugal. J. Investig. Allergol. Clin. Immunol., 19(3): 167-172.

van Bronswijk JE, Schober G, Kniest FM (1990). The management of house dust mite allergies. Clin. Ther., 12(3): 221-226.