Effect of Family History and Parental Smoking on the Development of Asthma in Turkish Adult Asthmatics

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Abstract

Objectives: To determine the distribution of family history and the importance of parental smoking in Turkish people with asthma.

Patients and Methods: 60 (46 female) asthmatics (41±15 years) and 59 (47 female) age and sex matched non-atopic controls (41±15 years) without any respiratory symptom were included in the study. All of the subjects were non-smokers. All of the asthmatics had positive skin prick test.

Results: In this study, 23 of 59 the asthmatics (40%) had severe and 36 of 59 asthmatics (60%) had mild disease. We found that prevalance of asthma in family members of the asthmatics was significantly greater than in controls. 44 of the asthmatics (%75) and 14 of controls (%23) had positive family history for asthma ($p<0.0001$, OR 9.6, CI 4.1-22.3). Positive family history seemed to be comparable between severe asthmatics (17/23, 74%) and mild asthmatics (27/36, 75%). The rate of positive family history was 16/22 (73%) in the patients with bronchial hyperreactivity (BHR), and 11/14 (79%) in the non-BHR asthmatics ($p>0.05$) and tendency of a familial clustering did not show any difference between patients with bronchial hyperreactivity and without BHR. Parental smoking history was seen in 35 (60%) of asthmatics and 42 (70%) of controls, ($p>0.05$).

Conclusions: Our study shows that there is a familial clustering in Turkish asthmatic patients and genetic susceptibility is important in asthma pathogenesis. Parental smoking history has no effect on asthma pathogenesis in the middle-aged asthmatic patient.

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Key words: *asthma, family history, parental smoking*
Abbreviations: *BHR: Bronchial hyperreactivity*

Introduction

It is well known that asthma develops with effects of environmental factors in the patients with genetic succeptibility to asthma. Asthma is a disorder that has a complex genetic inheritence (1). Asthmatic familial clustering has supported the presence of a hereditery component of asthma (2). Environmental and behavioral factors such as western life style facilititates appearance of asthma especially in the patients with genetic succeptibility to asthma. It has been reported that 58.3% of children with asthma had at least one parent who smoked, 38.5% were exposed to maternal smoking (3). Maternal smoking during pregnancy can cause to impairment in airway development and also has negative effects on the development of lung function in early childhood (4). Exposure to environmental tobacco smoke predisposes to respiratory illnesses and

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reduced lung function in infants and school-aged children (5). In addition, relationship between passive exposure to tobacco smoke and high prevalence of asthma and wheeze are observed in early childhood (6) but not in late childhood (7) and teenage years (8). Tobacco smoke is capable of either enhancing responses to antigens that normally stimulate of IgE responses (9) and/or modulating responses to favor production IgE antibodies (10) that are responsible for BHR (11). So, exposure to tobacco is one of the important and common environmental factors that predisposes to asthma. Our aim in this study is to assess the distribution of positive family history and the importance of parental smoking in adult Turkish people with asthma.

Methods

Subjects

Patients were prospectively selected from subjects admitted to outpatient clinic of Department of Pulmonary Medicine in Marmara University Hospital. Age and sex matched non-atopic subjects without any respiratory symptom were selected as controls. 59 (47 female, 12 male) atopic asthmatics and 60 (46 female, 14 male) non-atopic control subjects were enrolled into the study. Before entering the study, all of the subjects were asked to fill a standardized questionnaire (ATS DLD-78A questionnaire) (12) with questions about symptoms, duration of disease, other allergic diseases, family history, smoking habit etc. and were submitted to CXR, Water's graph, lung function test and methacholine challenge test, skin prick test. Diagnosis and determination of severity of asthma was made according to American Thoracic Society criteria (13). All of the subjects were non-smokers. All of the asthmatics had positive skin prick test. Demographics and clinical characteristics of subjects were shown in Table 1.

Table 1. Demographics and clinical characteristics of the subjects

	Asthmatics	Controls
Subjects (n)	59	60
Gender (F/M)	48/11	46/14
Age (year)	41±15	41±15
FEV_1 (% predicted)	75.9±23.5	104.8±11.0
FEV_1/FVC (% predicted)	72.0±13.8	86.4±4.6
Methacholine(PD_{20},mg/mL)	4.3±5.2	-

Each subject gave his/her written informed consent to participiate in the study. The study protocol was approved by local Ethical Committee of Marmara University School of Medicine.

Atopy was regarded as the presence of positive skin prick test (14).

Skin Prick Testing

Skin prick testing (Center Laboratories, Port Washington, NY,USA; MULTI-TEST applicator, Lincoln Diagnostics Inc, Decateur, Illinois, USA) was carried out using house dust mite, tree-mix, grass-mix, mold-mix, animal dander. Skin prick tests were regarded as positive if the maximum weal diameter was at least 3 mm or greater than negative control for any of the allergens tested.

Spirometri and Methacholine Challenge Testing

The subjects performed FVC maneuvers (Sensormedics, S5313, Ca, USA) according to the standardized methods recommended by American Thoracic Society (15). The best one of three acceptable measurements was accepted. Methacholine challenge test (Mediprom dosimeter, FDC 88, France) was performed according to previous studies in only mild asthmatics (16).

Statistical analysis

T-test for independent groups and χ^2 test was used to assess the difference between groups. Differences were considered to be statistically significant when at $p<0.05$.

Results

In this study, 70% of the asthmatics had exercise-induced asthma. Nocturnal asthma and aspirin-induced asthma and allergic rhinitis was found in 73%, 12%, 70% of asthmatics, respectively. 23 of 59 the asthmatics (40%) had severe and the rest of the asthmatics (36/59, 60%) had mild disease.

Skin prick test was positive to 1 allergen in 40% of the patients. Positive results to 2,3,4,5 different allergens were found in 25%, 17%,10%,8% of the asthmatics, respectively.

We found that prevalance of asthma in family members of the asthmatics was significantly greater than in the controls. 44 of the asthmatics (%75) and 14 of the controls (%23) had positive family history for asthma, ($p<0.0001$, OR 9.6, CI 4.1-22.3), Figure 1. Positive family history seemed to be comparable between the severe asthmatics (17/23, 74%) and the mild asthmatics (27/36, 75%).

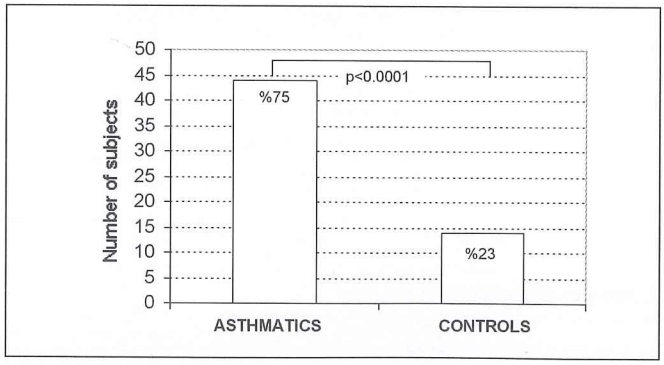

Figure 1. *Family history of asthma in asthmatic subjects and controls.*

The rate of positive family history was 16/22 (73%) in patients with BHR and 11/14 (79%) in the asthmatics without BHR, (p>0.05). Tendency of a familial clustering did not show any difference.

Parental smoking history was seen in 35 (60%) of the asthmatics and 42 (70%) of the controls, (p>0.05), Figure 2.

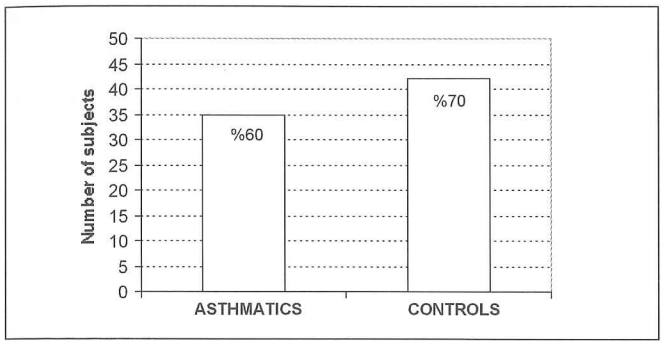

Figure 2. *Parental smoking history in asthmatic subjects and controls, p>0.05.*

Discussion

Our findings support the importance of the family history as a determinant of asthma in Turkish population. There was no significant association between parental smoking and asthma in the adult population.

It is known that smoking has negative effects on asthma. Maternal smoking during pregnancy increases the occurence of physician-diagnosed asthma during childhood (17). Children with positive history of parental asthma have greater incidence of asthma if there is maternal smoking during pregnancy according to results of Italian SIDRIA study (18). It has been reported that current environmental tobacco smoke is only associated with wheezing but not physician-diagnosed asthma (17). But it has also been reported that exposure to current smoking increases the risk of current asthma in children and in adolesence despite being much less than in children (18,19). It is generally regarded that environmental tobacco smoke operates as a cofactor not as a factor that induces asthma (17). Smoking is one of the trigerring factors in patients with asthma but the role of exposure to smoking decreases with increasing age.

In our study, we could not observe a significant relationship between family smoking and increased asthma incidence in the adult group. Parental smoking history was not significantly different between astmatics and controls. This finding is consistent with the observation that environmental tobacco smoke is cofactor. On the other hand, this result may also be related to the high rate of smoking in Turkish population.

Asthma is a complex genetic disorder. The familial aggregation (1,2,20), high asthma incidence in twin studies (21,22), and high asthma prevalence in isolated, inbreed population have shown evidence for genetic influences in asthma. Familial aggregation was observed in asthmatics in our study group when compared to controls. This finding shows importance of genetic influence on asthma pathogenesis like other reported studies. Futhermore, severity of asthma was not effected by familial history of asthma in our study population. Atopic asthmatic patients were included in the study, because genetic features of these asthmatics were also studied in another study.

Positive family history was not significantly different in patients with or without nonspesific BHR. It has been accepted that nonspesific BHR is a seperately inherited phenotypic trait due to the discrepancy between nonspesific BHR and symptomatic asthma (1). So, this observation can be accepted as an expected finding.

In conclusion, the familial clustering in Turkish asthmatic patients showed that genetic susceptibility is important in asthma pathogenesis. However, paternal smoking history could not be considered as a factor on asthma pathogenesis in the middle-aged asthmatic patient.

References

1. Sanford A., Weir T., Pare P. The genetics of asthma. Am J Crit Care Med 1996; 153: 1749-1765.
2. Panhuysen CIM., Bleecker ER., Koeter GH., Meyers DA., Postma DS. Characterizations of Obstructive airway disease in family members of probants with asthma. Am J Crit Care Med 1998; 157: 1734-1742.
3. Belousova EG., Toelle BG., Xuan W., Peat JK. The effect of parental smoking on presence of wheez or airway hyper-responsiveness in New South Wales school children. Aust N Z J Med 1999; 29(6): 794-800.
4. Tager IB., Ngo L., Hanrahan JP. Maternal smoking during pregnancy, Effects of Lung function during the first 18 months of life. Am J Crit Care Med 1995; 152: 977-983.
5. Stick SM., Burton PM., Gurrin L., LeSouef PN. Effects of maternal smoking during pregnancy and a family history of asthma on respiratory function in newborn infants. The Lancet 1996; 348: 1060-1064.
6. Jenkins MA., Hopper JL., Bowes G., carlin JB., Flander LB., Giles GG. Factors in childhood as predictor of asthma in adult life. B M J 1994; 309: 90-93.
7. Henderson FW., Henry MM., Ivins SS., Morris R., Meebe EC., Leu SY., Stewart PW. Correlates of recurrent wheezing in school-aged children. Am J Crit Care Med 1995; 151: 1786-1793.
8. Lewis S., Richards D.,Bynner J., Butler N., Britton J. Prospective study of risk factors for early and persistant wheezing in childhood. Eur Respir J 1995; 8: 349-356.
9. Zetterstrom O., Osterman K., Machado L., S.G.O.J. An other smoking hazard: raised serum IgE concentration nd increased risk of occupational allergy. B M J 1981; 283: 1215-1217.
10. Kjellman NI., Effect of parenteral smoking on IgE levels in children. Lancet 1981; 1: 993-994.
11. Sears MR., Burrows M., Flannery EM., Herbison GP., Hewitt CJ., Holaway MD. Releation between airway responsiveness and serum Ige in children with asthma and in apparently normal children. N Eng J Med 1991; 325: 1067-1071.
12. Ferris BG. ATS statement: epidemiology stantadization project. Am Rev Respir Dis 1978; 118: 1-120.

13. American Thoracic Society. Medical Section of the American Lung Association. Standarts for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. Am Rev Respir Dis 1987; 136:225-245.
14. Cookson WOCM., Faux JA., Sharp PA., Hopkin JM. Linkage between immünoglobulin E responses underlying asthma and rhinitis chromosome 11q. Lancet 1989; 1:1292-1295.
15. American Thoracic Society. Medical Section of the American Lung Association. Standardization of spirometry-1987 update. Am Rev Respir Dis 1987; 136:1285-1298.
16. Berrin Ceyhan, Turgay Çelikel. Effect of inhaled heparin on methacholine-induced bronchial hyperreactivity. Chest 1995; 107: 1009-1012.
17. Guilland FD., Li YF., Peters JM. Effects of maternal smoking during pregnancy and environmental tobacco smoke on asthma and wheezing in children. Am J Crit care Med 2001; 163(2): 429-436.
18. Apabiti N., Mallone S., Forestiere F., Carbo GB., Ferro S., Renzoni E., Sestni P., Ruscani F., Ciccone G., Viegi G., Chellini E., Piffer S. The impact of parental smoking on asthma and wheezing. SIDRIA Collaborative Group. Studi Italiani Sui Disturbi Respiratori nell'infunzia e l'ambiante. Epidemiology 1999; 10(6): 692-698.
19. Cunningham S., O'Connor GT., Dockery DN, Spezier FE. Environmental tobacco smoke, wheezing, and asthma in children in 24 communities. Am Respir Crit Care Med 1996; 153(1): 218-224.
20. Dold S., Wjst M., von Muitus E., Reitmer P., Stiepel E. Genetic risk for asthma, allergic rhinitis and atopic dermatitis. Arch Dis Child 1992; 67: 1018-1022.
21. Nieminen MM., Kaprio J., Koskenvuo M. A population based study of bronchial asthma in twin pairs. Chest 1991; 100: 71-75.
22. Duffy DL., Martin NG., Battistutta D., Hopper JL., Mathews JD. Genetics of asthma and hay fever in Australian twins. Am Rew Respir Dis 1990; 142: 1351-1358.

Kadıköy Boat Station; Photography by Kamil Levent Arslan, MD

Is There a Seasonal Clustering of Onset of Acute Sarcoidosis in Ankara?

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Abstract

The etiology of sarcoidosis is not yet known and there are various surveys reported on this issue. Some of this surveys are on the seasonal clustering of the onset, which may be related to the etiology.

Files of 50 patients who were diagnosed sarcoidosis between January 1980 and September 1998 were retrospectively investigated. The ones having erythema nodosum and arthralgia were thought to have acute sarcoidosis and accepted as group I (n=14) and the other as group II (n=36). The onset season of sarcoidosis was spring and summer in 10 (71.4%) patients in group I and in 22 (61.1%) patients in group II showing no statistically significant difference when the two groups are compared ($p>0.05$).

As a result, determination of the onset or the diagnosis time in sarcoidosis whether acute or not, is significantly more common in spring and summer which may show that the etiology of sarcoidosis may be related to exposure to some environmental factors.

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Key words: *etiology; sarcoidosis, seasons*

Introduction

Seasonal clustering of acute sarcoidosis has been reported in various countries previously (1-7). To find out whether this was valid for the patients in Ankara, a retrospective survey was performed between January 1980 and September 1998. Here the clinical features of the patients with sarcoidosis presenting to Hacettepe University Hospital Department of Chest Diseases are reported and the seasonality of the symptoms of the patients referring with erythema nodosum (EN) and arthralgia is investigated.

Materials and Methods

Files of 76 patients who were diagnosed sarcoidosis in Hacettepe University Hospital Department of Chest Diseases between January 1980 and September 1998 were investigated retrospectively and only 50 were enrolled in the study due to some missing data of the remaining patients. The patients presenting with EN and/or arthralgia were accepted as group I (acute sarcoidosis) and the others as group II. It may not be easy and reliable to determine the onset of the sarcoidosis symptoms other than EN and arthralgia since the disease usually has an insidious beginning, but it is more reliable with

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