Serum Immunoglobulin and Complement Levels in Atopic Skin Diseases

Mustafa Şenol[1], MD, Elif Özerol[2], PhD, Sezai Şaşmaz[1], MD, Kazım Şahin[3], MD, Fahri Turan[3], MS, Durali Soytürk[4], MD

The human immune system is extremely complex and interacts bidirectionally with environmental, metabolic, and endocrine factors as well as with infectious agents. It is arranged and controlled by genetics. Atopic dermatitis is a hereditary and distinct type inflammatory skin disease which may be associated with other atopic manifestations such as asthma, hay fever, and vernal conjunctivitis. The etiopathogenesis of atopic dermatitis is unknown and it is widely accepted that both intrinsic and extrinsic factors are involved. The aim of this study is to determine antibody responses and changes in complement levels in atopic skin diseases. [Journal of Turgut Özal Medical Center 1997;4(1):47-49]

Key Words: *Atopic skin diseases, immunoglobulins, complement*

Atopik deri hastalıklarında serum immünglobülin ve kompleman düzeyleri

İmmün sistem çok karmaşık bir yapıya sahip olup; çevresel, metabolik, endokrin ve enfeksiyöz faktörlerle karşılıklı etkileşim içindedir. Bu ilişkiler genetik olarak düzenlenmekte ve kontrol edilmektedir. Atopik dermatit, genetik geçişli ve farklı özellikleri olan inflamatuar bir deri hastalığıdır ve astım, allerjik nezle ve allerjik konjonktivit gibi diğer atopik hastalıklarla birlikte bulunabilir. Etyopatogenezi tam olarak bilinmeyen bu hastalıkta intrensek ve ekstrensek faktörlerin rol oynadığı düşünülmektedir. Bu çalışmanın amacı; atopik deri hastalıklarında gelişen antikor cevabını ve kompleman değişikliklerini araştırmaktır. [Turgut Özal Tıp Merkezi Dergisi 1997;4(1):47-49]

Anahtar Kelimeler: *Atopik deri hastalıkları, immünglobülinler, kompleman*

A normally functioning immun system entails all the forces and mechanisms concerned with recognition, specific response and removal of foreign objects after they again access into the body of the host. The body surfaces are the most important factors in case of host defense in the first line. Atopic dermatitis is a chronic inflammatory skin disease associated with elevated serum immunoglobulin -especially IgE- levels and sensitization to a variety of inhalant, food and microbial allergens. Many kinds of allergens can trigger acute IgE-mediated mast-cell dependent exacerbations of eczema in these patients. The complement is one of the major effector system in

[1] İnönü University School of Medicine Departments of Dermatology, Malatya
[2] İnönü University School of Medicine Departments of Microbiology, Malatya
[3] İnönü University School of Medicine Departments of Biochemistry, Malatya
[4] Military Hospital Dermatology Clinic, Malatya

the process of inflammation. Complement activation has been shown to occur in atopic dermatitis. Human IgE recognizes both exogenous allergens and structurally related human proteins has led to the hypothesis that IgE autoreactivity may be a pathogenic factor in atopic diseases (1-5).

In this study, we aimed to determine the serum levels of immunoglobulins G, M, A, and E, and C3 and C4 components of compleman atopic skin diseases and to compare with those of age-matched controls.

MATERALS AND METHODS

A total of 58 patients (26 males and 32 females) with atopic dermatitis and some other atopic skin diseases such as neurodermatitis, lichen simlex, nummular eczema, pompholix, and papular urticaria and 40 healthy controls (19 males and 21 females) were included in this study. The patients had no secondary infection or other kinds of dermatological diseases. Plasma from patients and controls were collected, centrifuged, and seperated sera without lypemia and hemolysis were frozen at -20°C until assayed. IgE was measured using enzyme-linked immunosorbent assay (IgE ELISA Test Kit, Genzyme, CA, USA). IgG, IgM, IgA, C3, and C4 were measured using immunoturbidometric assays (SPQ^{TM} Test System, Atlantic Antibodies, USA) by previously described techniques. The student's t-test in SPSS for Windows program was used for statistical analyses.

RESULTS

Our study group was consisting of 26 men and 32 women aged 1 to 63 (mean 29.7±18.6) years whereas 19 men and 21 women aged 6 to 61 (mean 33.4±11.2) years were chosen as control group. There was no significant difference between study and control groups according to the mean ages ($p>0.05$). In patients with atopic skin diseases, serum IgG values were 1992.49±917.31 mg/dl, IgM levels were 188.91±82.43 mg/dl, IgA values were 264.36±103.61, IgE levels were 177.84±69.07 IU/ml, C3 values were 157.91±51.87 mg/dl, and C4 levels were 29.12±15.03 mg/dl. These values in control group were 2073.35±734.73 mg/dl for IgG, 154.27±81.77 mg/dl for IgM, 341.22±127.82 mg/dl for IgA, 142.35±63.92 IU/ml for IgE, 166.78±71.56 mg/dl for C3, and 37.29±17.59 mg/dl for C4. All of these information is summarized on Table 1.

DISCUSSION

To determine the etiological role of immunoglobulins and complement fractions in atopic dermatitis and some other atopic skin diseases such as neurodermatitis, lichen simlex chronicus, nummular dermatitis, dyshidrotic eczema (pompholix), and papular urticaria, the circulating antibody and complement profiles were investigated in fifty-eight patients and compared with healthy age-matched controls. Patients with atopic dermatitis response to various exogenous and endogenous antigens mainly by generating an IgE-mediated humoral immune response. Recent investigations have indicated that not only IgE but also IgG and IgA may play some etiological role in the pathogenesis of atopic dermatitis (6,7). Other immunologic mechanisms, especially cell-mediated immune reactions and some neurological events can also be involved.

We found no significant difference between IgG levels in atopic and healthy groups. In the literature, there are controversial results on this issue. In one study, IgG and IgA values were within normal limits in patients, contrary to the statistically significant increase in IgM and IgE values (1). In another study, IgG levels were significantly higher in the atopic subjects than those of controls. IgA levels were significantly lower in atopic group compared with controls. There was no significant difference in IgE concentrations between these groups (8). Morsy et al have found that all the immunoglobulins (IgG, IgM, IgA, and IgE) were higher in atopics (9).

IgM levels were significantly higher in patients with atopic skin disesases than in controls in parallel to some previous studies (1). On the other hand, a

Table 1. Epidemiologic and immunologic properties of the atopic patients and controls

	Patients	Controls	p
Age (years)	1-63 (29.7±18.7)	6-61 (33.4±11.2)	>0.05
Number	58 (26 M, 32 F)	40 (19 M, 21 F)	
IgG (mg/dl)	1992.49±917.31	2073.35±734.73	>0.05
IgM (mg/dl)	188.91±82.43	154.27±81.77	<0.05
IgA (mg/dl)	264.36±103.61	341.22±127.82	<0.05
IgE (IU/ml)	177.84±69.07	142.35±63.92	<0.05
C3 (mg/dl)	157.91±51.87	166.78±71.56	>0.05
C4 (mg/dl)	29.12±15.03	37.29±17.59	>0.05

non significant increase in IgM of patients was observed in one study (9).

We observed a significant decrease in IgA values in patients than those of controls. Hill et al. have found similar results in their investigations (8). In some studies, IgA values were within normal limits in patients (1). On the other hand, in some studies, IgA showed non significant increases in patients compared to controls (9).

IgE levels were significantly higher in our study group than in the control group in parallel to some others (1,3,5,6,9). The role of elevated serum IgE levels in patients with atopic dermatitis has been unclear. It has recently been shown that house dust mite antigens may penetrate the skin, bind to IgE on Langerhans' cells, which in turn mediate the activation of antigen-specific TH2 cells that are the predominant T cells found in early atopic skin lesions. TH2 cells synthesize IL-4, which stimulates the IgE production by B lymphocytes, and are chemotactic for eosinophils. The discovery of microbial superantigens that activate T cells more easily and less specifically than the traditional antigens, helps us to understand how local microbial agents can provoke the outburst of new atopic skin lesions (10). The same contradiction is present is also here, some studies have indicated that measurement of total serum IgE would be of no benefit in the preliminary clinical investigation of a suspected host (8).

We found no significant differences in C3 and C4 values between patients and controls. Most of the previous studies have indicated similar results (4) whereas in one study, elevated levels of complement C3 were observed (3).

The changes in the IgG, IgM, IgA, and in particularly IgE may be specific response to a series of exogenous and endogenous antigens or a non specific reaction. It is concluded that immunity to atopic skin diseases involved mainly a humoral, especially IgE-mediated, immune response but this syndrom is also associated with a cell-mediated immunological response against many kinds of exogenous and endogenous factors. Studies will be performed on large population are needed to solve some controversial results on this subject.

REFERENCES

1. Valdes Sanchez AF, Gomez Echevarria AH, Lastra Alfonso G. Atopic dermatitis: serum immunoglobulins and T-lymphocyte subpopulations. J Investig Allergol Clin Immunol 1991; 1: 154-8.
2. Somos Z, Schneider I. Serum and secretory immunoglobulins in atopic dermatitis. Orv Hetil 1993 Jun 20; 134: 1359-61.
3. Sergeev Iu V, Zimin Iu I, Reznikov Iu P, Malyshev VS, Pimenova NS. Atopic dermatitis. I. Characteristics of the clinical course and state of the immune status in relation to the initial level of serum IgE. Vestn Dermatol Venerol 1989; 3: 8-12.
4. Sergeev Iu V, Reznikov Iu P, Lobanova EV, Pimenova NS. Atopic dermatitis. II. The status of complement proteins and the pathogenetic role of anaphylatoxins C4a, C3a and C5a Vestn Dermatol Venerol 1989; 4: 4-7.
5. Leung DY. Role of IgE in atopic dermatitis**.** Curr Opin Immunol 1993; 5: 956-62.
6. Hill PB, Moriello KA, De Boer DJ. Concentrations of total serum IgE, IgA, and IgG in atopic and parasitized dogs. Vet Immunol Immunopathol 1995; 44: 105-13.
7. Okada S, Maeda K, Tanaka Y, Anan S, Yoshida H. Immunoglobulins and their receptors on epidermal Langerhans cells in atopic dermatitis. J Dermatol 1996; 23: 247-53.
8. Hill PB, Moriello KA, De Boer DJ. Concentrations of total serum IgE, IgA, and IgG in atopic and parasitized dogs. Vet Immunol Immunopathol 1995; 44: 105-13.
9. Morsy TA, Zohdi HW, Abdalla KF, Nasr ME, Ibrahim AA, el Said AM, Khalil HT. Immunoglobulins in patients with atopic dermatitis due to mites infestation in Qualyobia Governorate, Egypt. J Egypt Soc Parasitol 1994; 24(3):495-504.
10. Bjerke JR. Immunopathogenesis of atopic eczema. Tidsskr Nor Laegeforen 1994; 114: 1827-8.

Correspondence: Mustafa Şenol, MD
İnönü University School of Medicine
Department of Dermatology
44100 MALATYA
Phone: 422-323 1753
Fax : 422-341 0036
E-mail: mustafa.senol@ihlas.net.tr