Specific IgE Antibodies To *Sarcoptes Scabiei* In Scapetic Patients Also Recognize House Dust Mites Antigens

Muhammad M. Al-Ani*Ph.D

Summery

Back ground: *Scabies is relatively a highly contagious inflammatory skin disease caused by infestation with a parasite Saivoptes s_cabici . The pathogenesis of scabies is mainly due to & siarts after sensifi/ation of the host to the mite & its products which provokes an allergic response, responsible for a wide spectrum of clinical & histological manifestations .*

Objective: To determine whether specific IgE against scabies can show a cross reactivity with house dust mites allergens.

Study Design: *The level of specific IgE. against Sarcoptes scabiej_ var hominis , Dermatophagoides faiinae & Dermatophagoides pteronyssinus was measured in 30 scabietic patients by ELIZA method & (he results was compared using t test with that from the control group.*

Result: *There was a high statistical correlation (P<0.001) between the data of specific IgE against Sarcoptes scabiei var hominis , Dermatophagoides farinae & Dermatophagoides pteronyssinus in the patient group in comparison with that from the control group.*

Conclusion: *Some antigens from Sarcoptes scabiei body & its fecal pellets cross react with house dust mites body & feces , since they are related phylogenetically. So specific IgE against scabies can cross react with house dust mites, Dermatophagoides farinae & Dermatophagoides pteronyssinus, which are important allergens responsible for extrinsic asthma. This cross reactivity may explain the association of severe form of scabies infestation with atopic diseases & the persistence of symptoms in some cases even after proper treatment of the disease.*

J Fac Med Baghdad
2006; Vol. 48, No.2
Received Feb. 2004
Accepted april 2004

Introduction :

Scabies is a contagious inflammatory skin disease caused by infestation of Sarcoptes scabiei var hominis a parasite that colonizes the human epidermis [1].

The disease is undoubtedly an ancient problem, far in the period of Babylon & later on Roman era . Ruth in I lot Springs was well known to be curative. In the early Islamic era many Muslims physicians described & treated scabies by painting the whole body with yellow sulphur alone or mixed with oil [2].

Several centuries later in 1687 Bonomo & Cestoni in Italy described the parasite & marked scabies as the first infective agent to be related to a specific disease in man [3] , but unfortunately it took two centuries before this was generally accepted & definitely proven a mite Sarcoptes scabiei causes human mange by Raspail & his Corsican student Renacci in 1834 [4]

Transmission of scabies usually occurs by direct close physical contact including sexual contact, with an infested person's lesion . This contact must be enough to allow the acarus to pass from one host to another & to establish itself by burrowing in the skin [5]

* *Dcpt. Of microbiology , college of medicine , university of Baghdad*

However the surrounding area can be a source of living mites as well, these include bedding, clothes, underwear, blankets, dusts & rugs [3] .

Human scabies is caused by the mile Sarcoptes scabiei var hominis an anthrapod of the Class Arachnida , which differ from the insects in the absence of the wings. Order Acarina that includes ticks & mites where the head, thorax & abdomen are fused in an unsegmented body [6] .

Suborder Astigmata which includes the parasetic mite (Sarcoptes scabiei) & the free living mites (Dermatophagoides farinae & Dermatophagoides pteronyssus which are associated with house dust allergies [3].

Family Sarcoptidae , Genoits sarcoptes , which have more than 30 species & 15 varieties infesting human & other mammalians separately & named from the host in which they live as Sarcoptes scabiei var hominis, bovis, equi, suis & cams for human, cattle, horse, swine, & dog respectively [7].

When the mites enter the skin, it is quite accepted that, they evoke an immune response to scabies infestation which may be humoral &/ or cellular mediated immunity [8,9] however non of these reactions have been shown to eliminate all mites from the skin surface, though locally they may prevent the epidemic multiplication of scabies organisms on the skin [10] . The pathogenesis of scabies is mainly due to & starts after sensitization of the host to the mite & its products which provokes an allergic response, responsible for a wide spectrum of clinical & histological manifestations that ranges from subjective

symptoms with few parasites & hardly any visible lesions to a severe hyperkeratotic form of the disease, where several thousands of mites arc digging into the skin [11]. Moreover at least 6 antigens from Sarcoptes scabiej body & 3 antigens from its fecal pellets cross react with house dust mites body & feces respectively [12,13] since they arc related phylogenetically [12,14,15]. These mites, Dermatophagoides farinae & Dermatophagoides pteronyssinus, are important allergens responsible for extrinsic asthma, conjunctivitis & allergic rhinitis [16]. This cross reactivity may explain the association of severe form of scabies infestation with atopic diseases & the persistence of symptoms in some cases even after proper treatment of the disease [17]

The itching & scratching in scabies is mainly due to immediate type hypersensitivity reaction [14] , through the release of histamine & other cellular products from the skin, onward, the clinical manifestations of scabies are seen & felt after an induction phase from 2-8 weeks[3]. this is suggested by:

--- An elevation of total serum IgE antibodv among patients with seabies & fall after successful treatment[8,14,18] .Moreover there is a statistical correlation between IgE level & the degree of lesion extension [16]. Those with severe form of Norwegian scabies have an extreme elevation of scrum IgF level [19] , simulating those with atopicr diseases [20]

--- Antigen specific IgE in the scrum was elevated in scabietic patients as determinate by radio-allergio-sorbant test (RAST), using crude scabies antigen, that cross react wilh antigen from the common house dust mite [12,20,21]

--- Intra-cutaneous skin test with extracts of adult mite causes a wheal reactions in the individuals who have had scabies but not normal volunteers1[20] , & this can be passively transferred by a factor in the serum(Prausnylz-Kustner) [19].

--- Scabietie patients serum could cause histamine release using basophil degranulation test [22]

IgE may act to protect the host against parasites through mediators released by triggered mast cells, that affect the parasite directly or via increasing vascular permeability together with the releasing of eosinophil chemotactic factor . All these could lead to accumulating the necessary antibodies & cells to attack the parasite , in addition IgH immune complexes can induce macrophage mediated cytotoxicity to parasites its products [23,24,25]

Subjects:

This study was conducted in the , Baghdad teaching hospital /Department of Dermatology & in Al-Rasheed Military Hospital, in a period between August 2002, & February 2003, gathering a total sum of 60 individuals of both sexes, different age groups comprising all the patient group & the control group. From each individual a full history & clinical assessment was done. All the people in the stud}' & their family were free from history of allergic diseases &. Parasitic diseases.

The patient study grope (30 individual) were diagnosed clinically as having scabies by a history of contact with a satieties patient & finding the burrows in their skin.. All the patients had the disease for the first time, some of the patients developed the lesions recently, while other patients had the disease for more than one month without treatment. AH skin lesions were clinically free from secondary bacteria! Infection.

The control groups (30 individual) apparenllv normal persons were age & sex matched ill the patient group .

From each individual in the study 5 ml of blood was aspirated, few drops of blood were put on clean slides for blood film, the rest of the aspirated blood were allowed to clot, then ccntrifuged & the separated serum was divided into A aliquots & stored in a deep freeze at (-20 C°) till used. Thawing of each frozen serum was allowed only once at the time of its used in the test.

Method:

Ultra Allergy Enzyme Immunoassay specific IgE-Using Miniprep 75/2 (Biomaghreb) This procedure uses an enzyme immunoassay (ElA) method for the semi-quantitative determination of allergen-specific IgE concentration in human serum.

The serum is incubated in a first step with an allergen covalently bound to the solid support of a paper disc, if the sample contains antibody directed against this allergen it will be bound to the solid phase.

Washing the support with the washing solution removes all the antibodies having not reacted with the disc. Incubation of the washed paper disc with enzyme labeled antiserum human IgE allows the antiserum to fix the allergen-specific IgE antibodies to be bound to the solid support.

After a second washing oi' (he disc, a ehromogenic solution is added & incubated. This results in the development of a yellow color which is measured spectrophotometrically at 405 nm, after stopping the reaction & measuring in a colorimeter, the level of iillergen-specific IgE is calculated.

Compare the absorbanccs of each control & patient samples with the ibsorhances for the references sera IXC,B.A.I I. Assign the class value to he sample as follows:

Class	Count rate	Specific IgE conc.

5+	>H	Super high
4+	A-H	Very hgh
3+	B-A	Hgh
2+	C-B	Moderate
1+	D-C	Lo
0	<D	Non detectable

A semi-quantitative assessment can be done alternatively by assigning I.A,B.C&D the values of 52.5,1 7.5.3.5,0.7 & 0.35EU/ml respectively.
On semilog paper (included in (he kid) plot ()D average for each tandard(Y axis) versus concentration (X axis) of references sera. Read 'allies of controls or patients directly from the standard curve. Value telow 0.35 EU/ml are nor, detectable. Values increases with increasing concentration of allergen-specific IgE.

Results:

Total o(30 scabietic patients were studied their ages ranged between 12-47 years with a mean age ±S.D. 21.45.19.18 years . The predominance of 20(66.6%)male patients over 10(33.3%)) female patients made the male to female ratio2:l The distribution of cases by their age groups is shown in Fig. 1 , which shows the maximum number of patients were in their second decade of life (10-19 years).

Serum specific IgO against Sarcoptes scabiei var hominis was elevated in all patients (100%) above the upper normal value of the control group (3.5 EU/ml) & there was a high statistical significance P<0.001 between the mean value of serum specific IgF in patient group when compared with that of the control group. Moreover this specific IgF significantly cross react with specific IgF against house dust mites (Tab.l) .

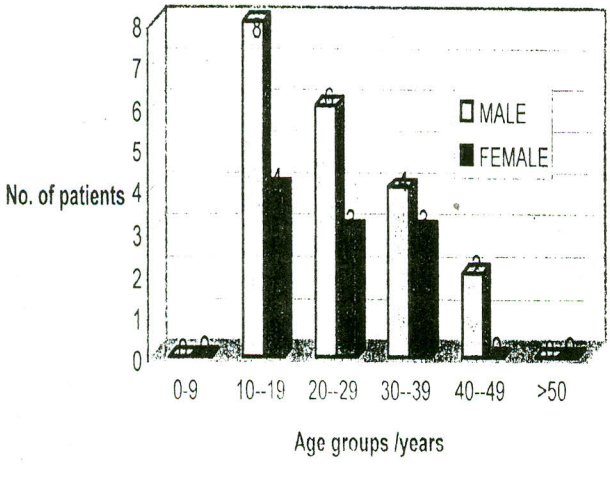

Fig.(1) The pattern of age groups distribution by their sex in (30) scabies patients.

Serum specific IgE(mean±SD)	Patients group	Control group	P value
Sarcoptes scabie	18.6±16.6	0.99+1.15	<0.001
D.farinae	14.1+14.67	1.57+1.39	<0.001
D.pteronyssus	12.36+13.17	1.77+1.43	<0.001

Discussion :

Scabies is undoubtedly an ancient disease. & in the present work some of the clinico-immunological characteristics of the disease were evaluated; Our study showed that scabies still affecting most age groups but it tends to have a higher incidence in younger people than elderly & the prevalence was decreased with increasing age groups, this drop in incidence may be due to acquired resistance , similar results were reported by some other studies Buxton (1941)(l), Beaver et al (1984)(5), , while different from another study by Christopherson (!()78)who reported that scabies was common in all age groups'"6'. This study also noticed that scabies was more frequent at the second decade of age because most of the patients were at 10-l(> years of age, while previous study by Al-Rawi (1900) reported that scabies was more predominant in children below 10 years(39.5%), & only (27.5%) were at second decade of life (ll> , the reason for this difference may be due to that , the previous study did not included the military hospitals in their work, while in this work (50%) of the patients included in the test were military persons, & for the same reason in this study there was a male predominance (2:1), which is agree with !;pstein(1 955 f , SrLvastara et al (!9S0)':6) , while Al-Rawi (1990) reported that the disease was equal between males & females' '.

Most of the female patients were students , while most of the males were military persons which is in agree with what was reported by Rpstein(1955){27).

Specific serum IgF was elevated in scabies patients which is coincided with what was reported previously

itching & skin lesions in scabies max be mainly due to immune hypersensitivity reactions through the release of histamine & other cellular mediators from triggered mast cells which may act to help the host against parasites either directly or via increasing vascular permeability to enhance the accumulation of the necessarv antibodies &cells that attack the

parasites(l5)(19)((20)(23)(25).

This study showed that there was cross reaction between specific IgE against scabies & anyigens present in house dust mites , since they are related phylogenetically1 .These mites, Dermatophagoides farinae & Dermatophagoides pj^ronyssjnus, are important allergens responsible for extrinsic asthma, conjunctivitis & allergic rhinitis'16'. This cross reactivity may explain the association of severe form of scabies infestation with atopic diseases & the persistence of symptoms in some cases even after proper treatment of the disease(l

References:

1. *Buxton P A . The parasitology of scabies Br.Mcd.J.(1941); scp. 20,397-401*
2. *Al-Rawi JR . Clinico-epidemiological study of scabies in Baghdad teaching hospital M.Sc. thesis faculity of medicine . Mustansrya university. (1990)*
3. *Marquardt W C . Demaree R S & Grieve R B . Parasitology of vector biology second Fd. Ilarcouri Academic press. (2000)*
4. *Beaver P C Jung R C & Cupp F W . Clinical parasitology ninth Fd. lea & Febiger Philadelphia (1984)*
5. *Carter LD. Treatment of scabies Br.Med.J.(1941); sep. 20,401-403*
6. *Belding DL. Subjec! in textbook of parasitology third edition New York. Applenton Latary-Frofts (1965): 1001-1004*
7. *Sulsberger B M & Kaminstein I . Avian itch mite is a cause of human disease dermatosis Arch. Dermato!. Syph. (1936); 33 : 61-72 \S. Falk IS. Serum immunoglobulin value in patients with scabies Br. J. Dermatol. (1980) ; 102 : 57-61*
9. *Nasser N F , Mukaled K M . Flzayat F A & Sannad M M . Humoral & cell mediated immune response in scabietic patients. J. Egypt. Soc. Parasitol. (1901) Dcc.;21(3) :765-770*
10. *Cabrera R, Agar A, Dahi MV. The immunology of scabies. Semin Dermatol. (1993) Mar.; 12(1) : 1 5-21*
11. *Van Neste D . Immunology of scabies. Parasitology today (1986) ; 2 : 194-196*
12. *Arlian L G , Vyszenski-Moher DL & Gllmone H M . Cross antigenecity between Sarcoptes scabie & house dust mite Dermatophagoides farinae J.Med. Entamol. (1988); 25 : 240-247*
13. *Mellanby KS. Scabies. Hamton .England W. dassey Limited (1972).*
14. *Falk. E.S. & Bolle, R. In vetro demonstration of specific immunological hypcrsensitivity lo scabies mite. Br. J. Dermatol. (1980); 101 :l67*
15. *Morsy TA & cl Fakahany A F. Seru immunoglobulins & complements values in scabietic patients. J. Egypt. Soc. Parasitol. (1991) Apr. ; 23(1) : 221-229*
16. *Stenius B & Wide L. Reaginic antibody (IgF) skin & provocation test to Dermatophagoides furinae & house dust mite in respiratory allergy. Lancet (1969) ; ii : 455*
17. *Mostafa Ell , el-Kadi MA, al-Zeftawy AH, Singer HM & Khalil KA. The relation between scabies & hypcrsensitivity to antigen of house dust mites & storage mites . J. Egypt. Soc. Parasitol. (1998) Dec. ; 28(1) : 777-787*
18. *Falk F S . Serum immunoglobulins before i^: after treatment of scabies. Allergy (1982) ; 36 ; 167-174 I1).*
19. *Burks JW, Jiinu R & (ieorue \VM. Norwegian scabies Arch. Dermatol. (1956) ;7 1 :131*
20. *Falk LS & Bolle R . IgE antibodies to house dusl mites in patients with scabies. Br. J. Dermatol. (1980); 102 : 281-288 •*
21. *Iloefling KT & Schroeter AL. Dermato-immuno-pathology of scabies. J. Ann. Acad. Dermatol. (1<>XO) ; 1 : 237*
22. *Van Neste D & Lachapella JM . Host parasite relationship in hyperkeratotic(Norwegian) scabies, pathological & immunological finding Br. Med. J. (1981) ;105 :667-678*
23. *McLaren D J . Role of eosinophils in tropical diseases. Seminars hematology vol.(19) No. (2). Apr. (1982) : 100-106*
24. *Morsy T A , el-Alfy M S , Arafa M A , Salama M M & Habib K S . Serum level of tumor necrosis factor alpha(TNF-a) versus immunoglobulins IgM, IgG & Igli in Fgyption scabietic children J. Egypt. Soc. Parasitol. (1995) jDec.^SO) : 783-786*
25. *Braunweld E , Fssclbacher K S , Jean D W , Mortin J B & Fauci A S . Harrison's principle of internal medicine forth edition . Me Graw-Mill book company (1988).*
26. *Christopherson J . The epidemiology of scabies in Denmark 1900-1975 Arch. Dermatol. (1978) ; 1 14 ; 747-75,0*
27. *Epstein E . Treds in scabies Arch. Dermatol. (1955); 84 : 14()*