

Original Research Article

Atopic dermatitis and its association with serum immunoglobulin E levels: our experience in KVG medical college and hospital, Karnataka

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ABSTRACT

Background: Eczema is an inflammatory skin reaction which presents as acute, subacute and chronic forms. Eczemas persisting for more than 6 weeks or characterized by thickening and discoloration of skin is typical of chronic eczema. Atopic dermatitis (AD) is a type of chronic or chronically relapsing eczematous skin disorder. To determine the percentage of AD in all forms of chronic eczema by using HRC. We also estimated serum immunoglobulin E (IgE) levels and determined its correlation with chronic eczemas and with various clinical parameters of HRC.

Methods: A total of 50 patients with chronic eczema meeting defined inclusion and exclusion criteria were enrolled in this cross-sectional study after taking an informed consent and approval of institutional ethical committee. All patients were subjected to a detailed history based on a questionnaire. A thorough clinical examination was done to determine all major and minor clinical parameters of HRC for AD. Blood samples were collected and AEC and total serum IgE levels were determined.

Results: Most of our study patients were females (64%). Majority of males (77.7%) were farmers and majority of females (56.2%) were housewives assisting in fieldwork activities. Various causes of chronic eczema were clinically diagnosed AD (34%), chronic actinic dermatitis (8%), polymorphic light eruption (4%), airborne contact dermatitis (10%), phyto-photodermatitis (10%), chronic hand and/or foot eczema (16%) and seborrheic dermatitis (2%). Thirty-two patients (64%) satisfied HRC. Among all clinical parameters of HRC, pruritus and xerosis were the commonest in AD patients. Serum IgE level was raised in 58% of chronic eczema and 68.7% of AD patients.

Conclusions: Serum IgE levels showed significant association with typical morphology and distribution of lesions, early age of onset and perifollicular accentuation.

Keywords: AD, IgE levels, Eczema

INTRODUCTION

Eczema is an inflammatory skin reaction characterized by redness, papulo-vesicles, oozing, crusting and scales. Clinically it is categorized as acute, subacute and chronic. Chronic eczemas are typified by persistent pruritus, thickening of skin and an indolent course. Eczemas are classified as 'exogenous' and 'endogenous'. Prevalence of all forms of eczema in a survey of 20,000 people in USA was 1.8%.¹ In India, study of 300 patients each, in

age group of below 17 years and above 60 years, found eczema in 24% and 37% respectively.² AD is a common, chronic relapsing inflammatory, multifactorial skin disease, which is characterized by intense pruritus.³⁻⁵ It affects up to 20% of children and 1-3% of adults.⁶ The mechanisms responsible for the onset and aggravation of AD involve skin barrier dysfunction and an atopic background. In patients with AD, the functions of the intercellular lipids of the stratum corneum are impaired because of abnormal reductions in ceramide levels.^{7,8} The

horny cell layer, which consists of keratin and filaggrin, is structurally tough. A loss-of-function mutation in filaggrin and filaggrin deficiency related to inflammation have been observed in patients with AD.^{9,10} A reduction in skin barrier function might allow stimuli and allergens to penetrate the skin more easily. Interleukin (IL)-33, IL-25, and thymic stromal lymphopoietin (TSLP), which are released from epidermal keratinocytes upon exposure to proteases, allergens, infections, or tissue damage, induce type-2 immune reactions, leading to the induction of allergen-specific IgE antibody production. With regard to the treatment of AD, topical corticosteroids and topical calcineurin inhibitors are the main treatments for inflammation, whereas the topical application of moisturizers is used to treat cutaneous barrier dysfunction.^{3,4,11} Systemic treatment, e.g., oral cyclosporin and UV irradiation, is an option for severe refractory cases.^{3,4,12} Several patient-specific complicating factors are seen in most cases. It is important to identify such factors and establish strategies to combat them. Hence, by this study, we aim to assess the prevalence of AD and its various diagnostic parameters in patients presenting with all forms of chronic eczema. With this evidence, guidelines for occupational and life-style counselling can be given to the patients with past or present history of AD which will help in reducing recurrence of the disease and improve quality of life.

Aim of the study

Aim of study were to calculate the percentage of AD in patients of chronic eczema, to calculate the percentage of various diagnostic criterion of AD in patients of chronic eczema and to correlate serum IgE levels with various diagnostic criteria of AD.

METHODS

This cross-sectional observational study was conducted in department of dermatology venereology and leprosy, KVG medical college and hospital, on an out-patient basis, meeting mentioned inclusion and exclusion criteria from July 2019 to Jan 2021 for a total period of 18 months.

Source of data

During this period 50 number of cases of varicose veins were studied, which were admitted in department of general surgery at KVG medical college and hospital, Sullia and selected randomly.

Method of collection data

A total of 50 patients with chronic eczema meeting defined inclusion and exclusion criteria were enrolled in this cross-sectional study after taking an informed consent. In cases of minors, the informed consent of parents or guardians was taken. Chronic eczema was

regarded as any eczema persisting for more than 6 weeks or more, or having history of similar episode in past. Institutional Ethics committee approval was obtained. Detailed history of all patients based on a standard questionnaire was taken with emphasis on duration of present illness, age of onset, exacerbating factors and personal and family history of atopic disorders and documented in a structured proforma. A clinical diagnosis was made on the basis of distribution and morphology of lesions. A thorough clinical examination was carried out to determine all major and minor clinical parameters of Hanifin and Rajka's criteria for AD. Morphology and distribution of the lesions was also noted. Patients were also examined for other associated clinical features which were not part of HRC and frequency of such features was calculated. Diagnosis of AD was confirmed if patients satisfied three major and three minor criteria of Hanifin and Rajka. Blood samples were collected from all enrolled patients for absolute eosinophil counts (AEC) and total serum IgE levels. Each serum sample was subjected to total IgE measurement based on sandwich ELISA technique. Total serum IgE level less than 200 IU/ml was considered as normal. AEC was also performed manually for all patients by using Fuchs Rosenthal chamber and values below 350 cells/mm³ were considered normal. Results of investigations were reviewed in both chronic eczema and AD patients. After screening all the enrolled patients of chronic eczema, the percentage of patients satisfying HRC, i.e., diagnosed as AD, was calculated. Most common forms of chronic eczemas associated with AD were noted. The frequencies of all the clinical parameters of HRC were calculated. Elevated serum IgE levels were correlated with various clinical parameters of HRC. Hematological-absolute eosinophil count (AEC) and immunological serum IgE levels.

Inclusion criteria

Patient willing for the study. Patients with eczematous skin lesions of > 6 weeks duration. If the eczematous skin lesions are of <6 weeks duration, should have history of similar lesions in past or signs of lichen simplex chronicus*. (*Lichen simplex chronicus is a classical feature of chronic eczema and is characterized by thickening of skin with increased skin markings) were included in the study.

Exclusion criteria

Patients not willing for the study. Patient with immunocompromised status. Pregnant and lactating women. Patients on systemic corticosteroids and immunosuppressants were excluded from the current study.

RESULTS

The twenty-four percentages (12/50) patients were in age group 0-20 and 21-40 and more than 60 years each. The

28% (14/50) patients were in the age group 41 years to 60 years. The 64% (32/50) were the females whereas 36% (18/50) were males.

Table 1: Age and sex distribution in the study group, (n=50).

Age groups (years)	Males	Females	Total
0-20	2	10	12
21-40	3	9	12
41-60	4	10	14
More than 60	9	3	12
Total	18	32	50
Statistics	Mean age=42.04 years, X ² =10.834; p=0.028 [p<0.05 SS]		

The 36% (18/50) of patients were farmers by occupation. The 36% (18/50) of females were housewives with an agricultural background. The 24% (12/50) of patients were students. The 4% (2/50) of patients were a cook and a driver each.

Table 3: Conditions presenting as chronic eczema in the study group, (n=50).

Diagnosis	Patients satisfy HRC				Total		Statistics
	Yes		No		F	%	
	F	%	F	%			
Clinically diagnosed AD*	14	82.3	3	17.7	17	34	X ² =3.882; p=0.049
Chronic actinic dermatitis	3	75	1	25	4	8	X ² =1.000; p=0.317
Airborne contact dermatitis	5	100	0	0	5	10	NA
Lichen simplex chronicus	5	62.5	3	37.5	8	16	X ² =0.254; p=0.614
Polymorphic light eruptions	0	0	2	100	2	4	NA
Phyto-photodermatitis	3	60	2	40	5	10	X ² =0.200; p=0.655
Chronic hand and/or foot eczema	2	25	6	75	8	16	X ² =2.000; p=0.0157
Seborrheic dermatitis	0	0	1	100	1	2	NA

*This diagnosis was made on basis of history and distribution pattern before evaluating HRC.

Table 4: AD in chronic eczema group, (n=50).

Variables	Patients satisfying HRC		Total
	Yes	No	
Frequency of patients	32	18	50
Percentage (%)	64	36	100
Statistics	X ² =3.920; p=0.048 (SS)		

The 64% (32/50) of patients of chronic eczema satisfied HRC and were diagnosed as AD. The 9 (28.1%) out of 32 of patients diagnosed as AD gave positive family history of atopy.

The 20 (20%) patients had allergic rhinitis. The 8 (25%) patients had wheezing. The 20 (62.5%) patients had history of itchy eyes. The four (12.5%) patients gave history of urticaria. The seventy-five percentages of patients of AD gave history of either one of the features of atopy.

Table 2: Occupation profile in study group, (n=50).

Cases	Occupation			
	Farmers	House wife	Students	Other
Males	14	0	2	2
Females	4	18	10	0
Total	18	18	12	2
Statistics	X ² = 29.263; p=0.000			

The 17 (34%) patients were diagnosed clinically as AD, of which 14 (82.3%) satisfied HRC. The 4 (8%) patients were diagnosed as chronic actinic dermatitis of which 75% satisfied HRC. Airborne contact dermatitis was noted in 5 (10%) of patients of which 100% satisfied HRC. Lichen simplex chronicus was present in 8 (16%) of which 62.5% satisfied HRC. Polymorphic light eruptions were in 4% of which none satisfied HRC. Phyto-photodermatitis in 5 (10%) of which 60% satisfied HRC. Chronic hand and/or foot eczema was in 8 (16%) of which 25% satisfied HRC. Seborrheic dermatitis was present in 1 (2%) of patient of which none satisfied HRC.

Table 5: Distribution of lesions in patients of chronic eczema with AD.

Site	Frequency	Percentages (%)
Flexures	27	84.3
Extensor	21	65.6
Seborrheic areas	12	37.5
Face	21	65.6
Hands	4	12.5
Feet	9	28.1
Trunk	11	34.3
Nipple	2	6.25
Generalized	3	9.30

The 27 (84.3%) of chronic eczema with AD had flexural distribution. The 21 (65.6%) each had facial and extensor distributions. The 12 (37.5%) had seborrheic area involvement. Hands and feet were involved in 4 (12.5%) and 9 (28.1%) respectively. Trunk was involved in 11 (34.3%) and nipple eczema was present in 2 (6.25%). The 3 (9.3%) patients had generalized distribution.

The 100% patients had pruritus. The 96.8% had typical morphology and distribution of lesions. 100% patients had chronic/chronically relapsing course of disease. The 75% patients gave personal and/or family h/o atopy.

The 68.75% of patients had elevated serum IgE levels with mean of 619.66 (SD=948.99) IU/mm³. 75% patients

of AD had raised AEC levels with a mean of 494.34 (SD=198.36) cells/mm³.

Typical morphology and distribution and early age of onset showed statistically significant correlation with elevated serum IgE levels.

Table 6: Major criteria of HRC in patients of chronic eczema with AD.

Criteria	Cases				Statistics
	Yes		No		
	F	%	F	%	
Pruritus	32	100	0	0	NA
Typical morphology and distribution	31	96.8	1	3.2	X ² =28.12; p=0.000
Chronic or chronically relapsing course	32	100	0	0	NA
Personal and/or family H/o atopy	24	75	8	25	

Table 7: Minor criteria of HRC in patients of chronic eczema with AD, (n=32).

Criteria	Cases				Statistics
	Yes		No		
	F	%	F	%	
Xerosis	30	93.75	2	6.25	X ² 24.50; p=0.000
Orbital darkening	15	46.8	17	53.2	X ² =0.125; p=0.724
Hyper-linearity of palms	13	40.6	19	50.4	X ² =1.125; p=0.289
Early age of onset	9	28.1	23	71.9	X ² =6.125; p=0.013
Raised serum Ig-E	22	68.75	10	31.25	X ² =4.500; p=0.034
Tendency towards cutaneous infection	3	9.3	29	90.7	X ² =21.125; p=0.000
Facial pallor	3	9.3	29	90.7	X ² =21.125; p=0.000
Anterior neck folds	1	3.1	31	96.9	X ² =28.125; p=0.000
Intolerance to wool	4	12.5	28	87.5	X ² =18.00; p=0.000
Food intolerance	5	15.6	27	84.4	X ² =15.125; p=0.000
Nipple eczema	2	6.25	30	93.75	X ² =24.50; p=0.000
Cheilitis	9	28.1	23	71.9	X ² =6.125; p=0.013
Cataract	9	28.1	23	71.9	X ² =6.125; p=0.013
DM fold	6	18.75	26	81.25	X ² =12.50; p=0.000
Recurrent conjunctivitis	17	53.1	15	56.9	X ² =0.125; p=0.724
Non-specific hand and/ foot eczema	22	68.75	10	31.25	X ² =4.500; p=0.034
Pityriasis alba	5	15.6	27	84.4	X ² =15.125; p=0.000
Itch when sweating	14	43.75	18	56.25	X ² =3.125; p=0.077
Perifollicular accentuation	14	43.75	18	56.25	X ² =0.500; p=0.480
Course influenced by environmental and emotional factors	17	53.1	15	56.9	X ² =0.125; p=0.724

Table 8: Investigations in patients of chronic eczema with AD, (n=32).

Cases with raised serum IgE (%)		Cases with raised AEC level (%)	
Yes	No	Yes	No
22 (68.75)	10 (31.25)	24 (75)	8 (25)
Mean=619.66; SD=948.99 IU/mm ³		Mean=494.34; SD=198.36 cells/mm ³	

Table 9: Correlation of clinical parameters of HRC with elevated serum IgE levels.

Criteria	Statistics		Interpretation
	P value	CC	
Pruritus	0.409	0.116	No
Typical morphology and distribution	0.031	0.293	Yes
Chronically relapsing course	0.768	0.042	No

Continued.

Criteria	Statistics		Interpretation
	P value	CC	Statistically significant
Personal or family H/o atopy	0.083	0.238	No
Xerosis	0.768	0.042	No
Orbital darkening	0.643	0.065	No
Hyper linearity of palms	0.721	0.050	No
Early age of onset	0.021	0.311	Yes
Tendency towards cutaneous infection	0.318	0.140	No
Facial pallor	0.523	0.090	No
Anterior neck folds	0.768	0.042	No
Intolerance to wool	0.089	0.234	No

DISCUSSION

Bannister et al reported that 245 of 2604 patients (9%) who attended a contact clinic were diagnosed as AD which began for the first time at 20 years of age or older with no contact factor present.¹³ Ozkaya et al also reported 63 of 376 patients (16.8%) allocated to adult-onset AD with the age of 18 years as the cut-off mark.¹⁴ Alrichter et al recently found that sera from 28% of AD patients showed IgE autoreactivity directed against epidermal or epithelial cell line-derived proteins including cytoplasmic and cell membrane-associated moieties. This autoreactivity in AD patients was significantly correlated with the severity of the disease, defined by the total serum IgE levels and by clinical scoring indexes.¹⁵ IgE is involved in auto-immune reactivity in two ways. Firstly, it is an antigenic target for IgG anti-IgE antibodies and, secondly, it can also be autoreactive, with specificity for self-proteins. In a study from East India majority of individuals (40%) had summer exacerbations of this disease.¹⁶ Winter exacerbations were noted in majority of subjects (62%) from a North Indian study.¹ In a study of 89 subjects of AD, 74 (83.1%) experienced itch at the time of examination, and all the patients had experienced pruritus in the past. The majority of patients had generalized pruritus. The character of itch described by patients was as tickling, burning, stinging and pricking sensation with some not able to define the character. The most intense itching experienced by the majority of patients occurred in the evening (n=47, 52.8%) or at night (n=34, 38.2%). Only a small subgroup of AD patients reported the maximum severity of itch in the morning (n=10, 11.2%).¹⁷ Regarding factors influencing the frequency of itching, sweating (96%), physical effort (73%), skin dryness (71%) and stress (71%) were the most frequently mentioned parameters aggravating pruritus.¹⁸ Other relevant factors associated with itching severity are patients' age and disease duration however the association is not very strong. The 'infantile phase' of AD is seen in 45-60% of children, with an onset during the first 6 months of life and this may run until 2-3 years of age.¹⁹ Lesions are more of erythematous papules and vesicles. Oozing and crusting is seen in many cases. In this phase, the typical distribution pattern is that of a balaclava, with eczematous and highly pruritic lesions on the head and neck, sparing the periorbital and perioral regions.²⁰ 'Childhood phase' or juvenile AD is from 2-3

years of age until puberty. The rashes are more of lichenified papules and plaques. The classic areas of involvement are hands, feet, wrists, ankles and popliteal and anti-cubital fossae. Face, if involved tends to localize the lesion in periorbital and perioral areas in contrast to infantile phase. An inverse pattern has also been noticed, where a complete extensor distribution may be present. 'Adult phase starts' from puberty. Here, the predominant areas of involvement are flexures, face and neck, upper arms and back, and dorsal aspects of hands, fingers, feet and toes. Eruptions are characterized by dry, scaly, erythematous papules and plaques and the formation of large lichenified plaques. Typical morphology and distribution is reported to be a significant pointer of AD and is reported in 99.7%, 104. A frequency of 100% has been observed in 320 patients of AD giving history of chronic or chronically relapsing course of the disease.²¹ Nassif in his study, to know relation between AD and chronic idiopathic urticaria (CIU), suggested that CIU is part of the atopic diathesis since 96.5% of CIU patients examined by the author had a personal or familial history of atopic diseases.²² In another study results were contrasting.²³ Wahab et al conducted a study to evaluate minor features of HRC in 210, 1-12 years old Bangladeshi children. Assessment of 20 criteria revealed, cutaneous infection in 168 (80.0%) patients followed by coursed influenced by environmental factor in 140 (66.7%), high IgE level in 126 (60.0%), intolerance of wool in 105 (50.0%), xerosis in 92 (43.8%), infra-orbital fold in 83 (39.5%), ichthyosis in 72 (34.3%), early age of onset in 65 (31.0%), itch on sweating in 56 (26.7%), palmary hyper-linearity in 52 (24.8%), food hypersensitivity in 40 (19.0%), keratosis pilaris in 31 (14.8%), pityriasis alba in 30 (14.3%), facial erythema in 25 (1.9%), cheilitis in 22 (10.5%), hand eczema in 19 (9.0%), foot eczema in 16 (7.6%), intolerance of lipid solvent in 14 (6.70/o), scalp scaling in 11 (5.20/o) and infra-auricular fissure in 10 (4.8%).²⁴ A Turkish study, observed raised serum IgE levels in 57.5% whereas, in an Indian study it was found in 880/o of patients of AD.^{25,26} Dhar et al reported serum IgE levels ranging from 22 to 1188 IU with mean of 278.2 (SD 324.85) in 102 AD patients with mean age of 4.5 years.²⁷ The IgE serum concentration in an atopic patient is dependent on both the extent of the allergic reaction and hyperresponsiveness to a number of different allergens, both inhaled and ingested, to which the patient is sensitized. Non-atopic normal individuals have IgE

concentrations that vary widely and increase steadily during childhood, reaching their highest levels at age 15 to 20, and thereafter remaining constant until about age 60, when they slowly decline.²⁸ Dhar et al found AEC ranging from 34 to 3453 cells/mm³ with a mean of 624 (SD=590) in patients of AD and 10 to 512 cells/mm³ with a mean of 105 (SD=83) in controls.²⁹ High AEC levels in AD patients are associated with personal or family history of atopy and are significantly raised with the severity of the disease.^{11,6} It has also shown significant covariance with disease severity.²⁹

CONCLUSION

Chronic eczema predominantly involved females and middle-aged persons. It presented flexurally in most patients. AD (64%) was the commonest cause for chronic eczema implying a strong association between chronic eczema and atopic diathesis. Pruritus, xerosis, elevated serum IgE levels and non-specific hand and/or foot dermatitis showed a statistically significant association with atopic dermatitis in patients with chronic eczema. Agricultural occupation was a confounding factor for the very high frequency of hyper-linearities of palms observed in our study. Serum IgE levels were raised in majority (58%) of chronic eczema patients irrespective of they satisfying Hanifin and Rajka's criteria for AD. Among the various parameters of Hanifin and Rajka, typical morphology and distribution of lesions, early age of onset and perifollicular accentuation showed statistically significant association with raised serum IgE levels.

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REFERENCES

1. Johnson ML-T, Roberts J. Prevalence, morbidity and cost of dermatological disease. *J Invest Dermatol.* 1979;73:395-401.
2. Sayal SK, Bal AS, Gupta CM. Pattern of skin diseases in paediatric age group and adolescents. *Indian I Dermatol Venereol Leprol.* 1998;64:17-9.
3. Katoh N, Ohya Y, Ikeda M, Ebihara T, Katayama I, Saeki H et al. Clinical practice guidelines for the management of atopic dermatitis 2018. *J Dermatol.* 2019;46:1053-101.
4. Ring J, Alomar A, Bieber T, Deleuran M, Fink-Wagner A, Gelmetti C et al. Guidelines for treatment of atopic eczema (atopic dermatitis) Part I *J Eur Acad Dermatol Venereol.* 2012;26:1045-60.
5. Eichenfield LF, Tom WL, Chamlin SL, Feldman SR, Hanifin JM, Simpson EL et al. Guidelines of care for the management of atopic dermatitis: Section 1 diagnosis and assessment of atopic dermatitis. *J Am Acad Dermatol.* 2014;70:338-51.
6. Williams HC. Atopic dermatitis. In *The Epidemiology, Causes and Prevention of Atopic Eczema*; Williams, HC Ed, Cambridge University Press: Cambridge, UK. 2000.
7. Elias PM. Stratum corneum defensive functions: An integrated view. *J Gen Intern Med.* 2005;125:183-200.
8. Melnik B, Hollmann J, Plewig G. Decreased stratum corneum ceramides in atopic individuals-a patho-biochemical factor in xerosis? *Br J Dermatol.* 1988;119:547-9.
9. Cabanillas B, Novak N. Atopic dermatitis and filaggrin. *Curr Opin Immunol.* 2016;42:1-8.
10. Kono M, Nomura T, Ohguchi Y, Mizuno O, Suzuki S, Tsujiuchi H et al. Comprehensive screening for a complete set of Japanese-population-specific filaggrin gene mutations. *Allergy.* 2014;69:537-40.
11. Eichenfield LF, Tom WL, Berger TG, Krol A, Paller AS, Schwarzenberger K et al. Guidelines of care for the management of atopic dermatitis: Section Management and treatment of atopic dermatitis with topical therapies. *J Am Acad Dermatol.* 2014;71:116-32.
12. Sidbury R, Davis DM, Cohen DE, Cordoro KM, Berger TG, Bergman JN et al. Guidelines of care for the management of atopic dermatitis: Section 3. Management and treatment with phototherapy and systemic agents. *J Am Acad Dermatol.* 2014;71:327-49.
13. Bannister MJ, Freeman S. Adult-onset atopic dermatitis. *Australas J Dermatol.* 2000;41:225-8.
14. Ozkaya E. Adult-onset atopic dermatitis. *J Am Acad Dermatol.* 2005;52:579-82.
15. Altrichter S, Kriehuber E, Moser J, Valenta R, Kopp T, Stingl G. Serum IgE autoantibodies target keratinocytes in patients with atopic dermatitis. *J Invest Dermatol.* 2008;128:2232-9.
16. Dhar S, Mandal B, Ghosh A. Epidemiology and clinical patterns of atopic dermatitis in 100 children seen in city hospital. *Indian J Dermatol.* 2002;47:202-4.
17. Danuta Chrostowska-Plak, Joanna Salomon, Adam Reich, Jacek CS. Clinical Aspects of Itch in Adult Atopic Dermatitis Patients. *Acta Derm Venereol.* 2009;89:379-83.
18. Bos JD, Sillevius Smitt JH. Atopic dermatitis. *J Eur Acad Dermatol Venereol.* 1996;7:101-14.
19. Dold S, Wjst M, Mutius Ev, Reitmeir P, Stiepel E. Genetic risk for asthma, allergic rhinitis, and atopic dermatitis. *Arch Dis Childhood.* 1992;67:1018-22.
20. Rudikoff D, Lebwohl M. Atopic dermatitis. *Lancet.* 1998;351:1715-21.
21. Halkjaer LB, Loland L, Buchvald FF, Agner T, Skov L, Strand M et al. Development of atopic dermatitis during the first 3 years of life: the Copenhagen

- prospective study on asthma in childhood cohort study in high-risk children. *Arch Dermatol.* 2006;142:561-6.
22. Yazganoglu KD, Ozkaya E. Non-typical morphology and localization in Turkish atopic dermatitis patients with onset before the age of 18 years. *Indian J Dermatol Venereol Leprol.* 2011;77:23-7.
 23. Nassif A. Is chronic urticaria an atopic condition? *Eur J Dermatol.* 2007;17:545-6.
 24. Augey F, Goujon-Henry C, Berard F, Nicolas JF, Gunera-Saad N. Is there a link between chronic urticaria and atopy? *Eur J Dermatol.* 2007;17:545-6.
 25. Wahab MA, Rahman MH, Khondker L, Hawlader AR, Ali A, Hafiz MA et al. Minor criteria for atopic dermatitis in children. *Mymensingh Med J.* 2011;20:419-24.
 26. Yazganoglu KD, Ozkaya E. Non-typical morphology and localization in Turkish atopic dermatitis patients with onset before the age of 18 years. *Indian J Dermatol Venereol Leprol.* 2011;77:23-7.
 27. Somani VK. A study of allergen-specific IgE antibodies in Indian patients of atopic dermatitis. *Indian J Dermatol Venereol Leprol.* 2008;74:100-4.
 28. Dhar S, Malakar R, Chattopadhyay S, Banerjee R, Ghosh A. Correlation of severity of atopic dermatitis with eosinophil counts in peripheral blood and serum IgE levels. *Indian J Dermatol Venereol Leprol.* 2005;71:246-9.
 29. Bratton DL, Hamid Q, Boguniewicz M, Doherty DE, Kailey JM, Leung DYM. Granulocyte macrophage colony-stimulating factor contributes to enhanced monocyte survival in chronic atopic dermatitis. *J Clin Invest* 1995;95:211-8.

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