Dermatology Practical & Conceptual

Association of Serum IgE and Peripheral Eosinophilia with Seborrheic Dermatitis: Evidence from a Case-Control Study

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ABSTRACT

Introduction: It is thought that some shared mechanisms may play a role in the pathophysiology of seborrheic dermatitis (SD) and atopic conditions, both of which are chronic, inflammatory, and recurrent in nature.

Objective: This study aimed to investigate the presence of atopic diseases in SD patients without atopic dermatitis or other inflammatory skin disorders and to assess their serum IgE levels and peripheral eosinophil values as a marker of atopy.

Methods: This prospective cross-sectional study included 83 SD patients and 68 age- and sex-matched healthy controls.

Results: There were no significant differences between the SD patients and the healthy control group regarding sex distribution (P=0.507) or mean age (P=0.436). No significant difference was observed between SD and control groups in terms of accompanying rhinitis, conjunctivitis, or asthma (P=0.307, P=0.487, and P=0.370, respectively). The median serum IgE levels were significantly higher in SD patients than in controls (123.1vs. 46.7; P=0.001) No significant difference was observed between the groups in terms of eosinophil percentage or absolute eosinophil count (P=0.678 and P=0.827, respectively). SD patients with concomitant rhinitis had significantly higher IgE levels than those without rhinitis (P=0.048).

Conclusion: In this study, total serum IgE levels were significantly higher in SD patients than in healthy controls, suggesting that allergens and hypersensitivity responses may play a greater role in the pathogenesis of SD than previously thought.

Introduction

Seborrheic dermatitis (SD) is a chronic, inflammatory, recurrent skin disorder in which various factors such as immune dysregulation, inflammatory processes, skin dysbiosis, genetic predisposition, and environmental triggers, particularly stress, are implicated in its etiopathogenesis [1]. Atopy is defined as a genetically determined tendency to develop exaggerated immune responses to environmental allergens, characterized by $CD4^+$ Th2 differentiation and overproduction of immunoglobulin E (IgE). It is associated with elevated serum IgE levels and an increased risk of developing atopic conditions such as atopic dermatitis, allergic rhinitis, and asthma [2,3].

Immunoglobulin E is a molecule that plays a central role in allergic and inflammatory responses, while eosinophils are multifunctional leukocytes involved in the regulation of immune responses, particularly in allergic inflammation [3]. Some shared mechanisms may contribute to the pathophysiology of SD and atopic conditions, both of which are chronic, inflammatory, recurrent conditions [4].

Objectives

The present study aimed to investigate the presence of atopic diseases such as allergic rhinitis, conjunctivitis, and asthma in SD patients who did not have atopic dermatitis (AD) or other inflammatory skin disorders, and to evaluate their serum IgE levels and peripheral eosinophilia as potential markers of atopy.

Materials and Methods

The study was reviewed and approved by the local ethics committee (protocol number: 171, date of approval: 12.08.2024). Written informed consent was obtained from all participants. The study was carried out in accordance with the principles expressed in the Declaration of Helsinki. A prospective cross-sectional study was conducted. A total of 151 participants were included: 83 patients with clinically diagnosed SD and 68 healthy controls with no evidence of SD or any other inflammatory diseases.

Patients above the age of 18 years were enrolled. Only patients who presented with active SD lesions at the time of enrollment were included in the study. Patients with a history of any inflammatory dermatoses, including atopic dermatitis, contact dermatitis, cutaneous lymphoma, or systemic diseases (including inflammatory, metabolic, endocrinological, and infectious diseases, or malignancy), and those who had received any systemic treatment such as antihistamines, anti-inflammatory, immunosuppressive, or biological medications and any topical treatment within the previous three months of the study were excluded. Pregnant or lactating females were excluded.

Healthy controls were recruited from individuals attending routine check-ups in the same hospital and had no history of SD, AD, or chronic inflammatory disease.

Clinical and Demographic Data

For all participants, demographic information (age, sex), disease duration, and history of atopic conditions (allergic rhinitis, conjunctivitis, asthma) were recorded using a standardized data collection form. Disease severity in SD patients was assessed using the Seborrheic Dermatitis Area Severity Index (SEDASI), which scores erythema, scaling, and pruritus separately in the facial and scalp regions. The SEDASI was calculated by a single dermatologist to prevent intra-observer inconsistency. The total SEDASI score was obtained by summing the facial and scalp scores [5]. Scores of 10 and below were classified as mild disease, and those above 10 were classified as moderate-severe disease.

Laboratory Analyses

Venous blood samples were obtained from all participants after overnight fasting. Total serum IgE levels were measured using chemiluminescence method, and the results are expressed in kU/L. Eosinophil counts were obtained from complete blood counts performed on an automated hematology analyzer. Both the absolute eosinophil counts (cells/μL) and the eosinophil percentage (%) were recorded for each patient.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 27 (IBM Corp., Armonk, NY, USA).

Descriptive statistics were calculated as number, percentage, mean, standard deviation, median, and minimum-maximum values. Correlations between categorical variables were analyzed using Pearson's chi-square test. The normality of continuous variables was assessed using

Shapiro-Wilk tests. Non-normally distributed variables were analyzed using the Mann–Whitney U test and Kruskal-Wallis test. Correlations between continuous variables were analyzed using Spearman's rank correlation coefficient. A p-value of <0.05 was considered statistically significant.

Results

The demographic and clinical data of the participants are presented in Table 1.

There were no significant difference between SD patients and healthy controls regarding sex distribution (*P*=0.507) or mean age (*P*=0.436).

The disease duration among SD patients ranged from one month to 30 years, with a mean of 58.72 ± 66.31 months. A positive family history of SD was reported in 31.3% (N=26) of patients. Facial involvement was observed in 66.3% (N=55) of cases, while scalp involvement was almost universal, observed in 98% (N=81) of patients. The mean SEDASI face score was 3.31 ± 3.73, the mean SEDASI scalp score was

Table 1. Descriptive data of patients with SD and comparisons between SD and control groups.

	Seborrheic dermatitis (N=83)	Controls (N=68)	p
Sex, N (%)			0.507
Female	37 (44.6)	34 (50.0)	
Male	46 (55.4)	34 (50.00)	
Age (years), Mean ± SD, Median(min-max)	30.37±10.17 (18-62)	31.15±10.03 (18-65)	0.436
Rhinitis, N(%)			0.307
No	69 (83.1)	52 (76.5)	
Yes	14 (16.9)	16 (23.5)	
Conjunctivitis, N(%)			0.487
No	75 (90.4)	59 (86.8)	
Yes	8 (9.6)	9 (13.2)	
Asthma, N(%)			0.370
No	78 (94.0)	66 (97.1)	
Yes	5 (6.0)	2 (2.9)	
Disease duration (months) Mean ± SD, Median(min-max)	58.72±66.31 30 (1-360)		
Family history			
No	57 (68.7)		
Yes	26 (31.3)		
Face involvement			
No	28 (33.7)		
Yes	55 (66.3)		
Scalp involvement			
No	2 (2.4)		
Yes	81 (97.6)		
SEDASI, Face	3.31 ± 3.73 2(0-20)		
SEDASI, Scalp	8.3 ± 4.45 (8 (0-20)		
SEDASI, Total	11.42 ±6.2 10 (0-35)		
Pruritus			
0	16 (19.3)		
1	30 (36.1)		
2	28 (33.7)		
3	9 (10.8)		

Abbreviation: IgE: Immunoglobulin E, SEDASI: Seborrheic Dermatitis Area and Severity Index.

8.30 ± 4.45, and the mean total SEDASI score was 11.42 ± 6.20. Overall, 46 patients (55.4%) had a SEDASI score >10, indicating moderate-to-severe disease, while 37 patients (44.58%) had a score ≤10, consistent with mild disease. In terms of symptoms, 67 patients (80.72%) reported pruritus, whereas 16 patients (19.3%) did not experience itching.

No significant difference was observed between SD and control groups regarding the presence of rhinitis, conjunctivitis or asthma (*P*=0.307, *P*=0.487, and *P*=0.370, respectively)

The median serum IgE level was significantly higher in SD patients compared to controls (123.1 vs. 46.7; *P*=0.001). However, there was no significant difference between the groups in terms of eosinophil percentage or absolute eosinophil count (*P*=0.678 and *P*=0.827, respectively) (Table 2).

Among SD patients, those with concomitant allergic rhinitis had significantly higher serum IgE levels than those without rhinitis (*P*=0.048). No significant association was observed between serum IgE levels, eosinophil percentage, absolute eosinophil count, and clinical features, including disease duration, family history, scalp or facial involvement, and pruritus, as summarized in Table 3.

In patients with SD, no significant correlations were found between serum IgE levels and either eosinophil percentage (Spearman's correlation coefficient (r)=0.106, *P*=0.340) or absolute eosinophil count (r=0.167, *P*=0.132). Eosinophil percentage and absolute count showed a strong positive correlation (r=0.896, *P*<0.001).

Discussion

In this case-control study, we evaluated serum IgE levels and peripheral eosinophilia in patients with SD compared with healthy controls and investigated the relationship of these laboratory parameters with clinical features and atopic comorbidities. Our findings revealed that serum IgE levels were significantly higher in SD patients, while no significant difference was observed in eosinophil percentage or absolute eosinophil count. Additionally, the prevalence of allergic rhinitis, conjunctivitis, and asthma did not significantly differ between SD and control groups, although SD patients with concomitant rhinitis had higher IgE levels. Importantly, IgE levels were not correlated with disease severity scores or disease duration, and no association was observed with eosinophil parameters.

SD is a common chronic skin disease characterized by erythematous, scaly lesions, and pruritus, typically affecting sebaceous gland–rich areas such as the face, scalp, and chest. Although its pathogenesis remains incompletely understood, immune dysregulation, inflammatory processes, skin dysbiosis, overgrowth of *Malassezia species*, genetic predisposition, and environmental factors, particularly stress, are believed to play key roles [1,2]. Previous studies have mainly focused on the role of *Malassezia* species, altered skin barrier function, and innate immunity in SD pathogenesis [2,6,7]. Increased colonization with *Malassezia* yeasts, particularly *M. globosa* and *M. restricta*, has been reported to trigger immune responses via toll-like receptors, leading to the release of proinflammatory cytokines such as IL-6, IL-8, and TNF-α [2,8].

Atopy and allergic diseases are characterized by an exaggerated immune response to otherwise harmless environmental agents, typically mediated by a type 2 immune response [9-11]. In routine clinical practice, total IgE levels and eosinophil counts are widely used biomarkers for the diagnosis and monitoring of atopic conditions due to their accessibility, low cost, and practicality [11,12].

Although IgE is not essential for the development of AD, numerous studies have shown that atopic sensitization is closely linked to disease severity and prognosis. Sensitization to microbial antigens such as *Malassezia furfur*, *Candida albicans*, and *Alternaria alternata* has been demonstrated in a substantial proportion of AD patients, particularly those with moderate-to-severe disease [13-15]. In SD, *Malassezia* species and broader alterations in the cutaneous microbiome have also been implicated in disease pathogenesis

Table 2. Comparisons of serum total IgE levels and peripheral eosinophilia between SD patients and controls.

	Seborrheic dermatitis (N=83) Mean ± SD Median (Q1-Q3)	Controls (N=68) Mean ± SD Median (Q1-Q3)	p
IgE	301.67 ± 935.65 123.1 (80.7-215)	70.34 ± 78.46 46.7 (22.2-88.8)	**0.001**
Eosinophil percentage	2.46 ± 1.87 1.9 (1.2-3.3-)	2.12 ± 1.2 2.05 (1.1-2.9)	0.678
Absolute eosinophil count	0.18 ± 0.16 0.13 (0.08-0.24)	0.16 ± 0.09 0.14 (0.1-0.27)	0.827

Abbreviation: IgE: Immunoglobulin E.

Table 3. Association between clinical characteristics and serum IgE levels and peripheral eosinophilia.

N= 83		IgE Median (min-max)	Eosinophilia % Median (min-max)	Absolute Eosinophil count Median (min-max)
Sex				
Female		133 (13.7-8440)	1.6 (0.18-6.3)	0.12 (0.03-0.9)
Male		118.5 (10.7-1231)	1.95 (0.1-9.4)	0,15 (0.01-0.87)
P		0.180	0.145	0.167
Age (years)	r	0.101	0.042	0.101
	p	0.361	0.706	0.362
Disease duration (months)	r	-0.087	-0.098	-0.127
	p	0.435	0.378	0.253
Family history	Yes	122.25 (11.2-1160)	1.75 (0.1-7.6)	0.12 (0.02-0.87)
	No	123.1 (10.7-8440)	2.0 (0.1-9.4)	0.14 (0.01-0.9)
P		0.517	0.455	0.307
Face involvement	No	141.05 (10.7-8440)	2. 1 (0.2-6,3)	0.13 (0.03-0.45)
	Yes	117.0 (11.2-1231)	1.8 (0.1-9.4)	0.13 (0.01-0.9)
P		0.122	0.985	0.606
Scalp involvement	No	172.0 (117-227)	0.95 (0.4-1.5)	0.09 (0.03-0.15)
	Yes	123.1 (10.7-8440)	1.9 (0.1-9.4)	0.13 (0.01-0.9)
P		0.602	0.170	0.382
SEDASI, Face	r	-0.069	0.048	0.108
	p	0.538	0.434	0.175
SEDASI, Scalp	r	0.207	0.063	0.067
	p	0.060	0.574	0.549
SEDASI, Total	r	0.174	0.118	0.138
	p	0.116	0.264	0.169
SEDASI < 10		128.31 (10.7-844)	1.90 (0.2-7.8)	0.120 (0.01-0.47)
SEDASI ≥ *10*		118.0 (230-1120)	1.85 (0.2-9.4)	0.145 (0.01-0.90)
P		0.694	0.169	0.207
Pruritus				
0		97.5 (13.7-859)	1.85 (0.9-7.6)	0.15 (0.06-0.87)
1		135.8 (22-1120)	1.95 (0.1-5.2)	0.15 (0.02-0.9)
2		126.2 (10.7-1231)	1.7 (0.1-7.8)	0.11 (0.01-0.47)
3		93.1 (25.7-8440)	3.4 (0.7-9.4)	0.22 (0.04-0.5)
P		0.138	0.327	0.138
Rhinitis				
No		118 (10.7-8440)	1.8 (0.1-9.4)	0.13 (0.01-0.9)
Yes		167.6 (56-1160)	2.35 (0.1-5.1)	0.14 (0.02-0.37)
P		**0.048**	0.715	0.543
Conjunctivitis				
No		123.1 (11.2-8440)	1.9 (0.1-9.4)	0.13 (0.02-0.90)
Yes		122.45 (10.7-1160)	1.4 (0.1-7.8)	0.12 (0.01-0.47)
P		0.963	0.605	0.902
Asthma				
No		123.1 (11.2-8440)	1.9 (0.1-9.4)	0.13 (0.02-0.9)
Yes		122.45 (10.7-1160)	1.4 (0.1-7.8)	0.12 (0.01-0.47)
P		0.484	0.622	0.496

Abbreviation: IgE: Immunoglobulin E, SEDASI: Seborrheic Dermatitis Area and Severity Index.

[2,15,16]. Our study demonstrated that SD patients had significantly higher total IgE levels compared to healthy controls, which may be related to microbiome changes observed in SD. Elevated IgE levels suggest that SD may be more 'allergy-related' than previously assumed, indicating a potential overlap with atopic immune pathways.

A previous study involving 53 SD patients found no significant difference in serum total IgE levels between SD patients and controls; however higher levels of IgE were observed in patients with a disease duration of three to five years compared to those with shorter disease duration [18]. We did not observe such a relationship in our study.

Eosinophils are key effector cells in AD and asthma, mediating chronic tissue inflammation via cytokines and cytotoxic proteins. In contrast, eosinophils appear to play little or no role in SD. The absence of eosinophilia in our SD cohort suggests that although IgE elevation may occur, eosinophils are not major contributors to SD pathogenesis, highlighting immunologic differences between SD and classical atopic diseases.

In this study, we specifically examined SD patients without concomitant AD or other inflammatory dermatoses to assess the presence of atopic diseases such as allergic rhinitis, conjunctivitis, and asthma and to evaluate serum IgE levels and peripheral eosinophil counts as markers of atopy. Our results showed no significant difference in the prevalence of these conditions between SD patients and controls. However, the finding that SD patients with allergic rhinitis had higher IgE levels suggests that a subset of SD patients may share immunological mechanisms with atopic disorders. This supports the concept of disease heterogeneity in SD, where overlapping but distinct immune pathways may coexist.

This study has some limitations including its small sample size, cross-sectional design, and the fact that most patients presented with mild disease severity. Additionally, as serum IgE levels were measured only during active disease, IgE dynamics during remission remain unknown. Factors such as smoking, parasitic exposure, and seasonal allergy variation were not systematically recorded; however, these influences are expected to be similarly distributed across both patients and controls.

Although total IgE is not routinely used in the clinical management of SD, our findings raise the possibility that IgE may help identify an atopy-prone subgroup of SD patients. In selected cases, elevated IgE could support risk stratification and prompt evaluation for coexisting atopic conditions or guide further allergy-focused diagnostic pathways. Longitudinal studies evaluating IgE levels during flare and remission phases and in response to therapy would clarify whether IgE serves as a meaningful biomarker that tracks with disease trajectory or treatment response. Such data would enhance the clinical relevance of IgE measurements in SD. Taken together, our findings suggest that SD involves not only innate immune dysregulation but also systemic immune alterations. Elevated IgE levels in the absence of eosinophilia distinguish SD from AD and point to alternative immunopathological mechanisms. The observed IgE elevation point to the possibility that allergens and hypersensitivity responses may play a greater role in SD pathogenesis than previously recognized. Further studies with larger cohorts and longitudinal designs are warranted to clarify the clinical relevance of IgE elevation and to elucidate potential links between microbial antigens, allergic sensitization, and systemic immune modulation in SD.

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