Assessment of the Level of Serum Interleukin 17 in Patients with Atopic Dermatitis Compared to Normal Control before and after Treatment with Mometasone Furoate Cream 0.1% (Borgasone): A Case Control Study

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Abstract

Background: Atopic dermatitis (AD) is a chronic inflammatory skin condition characterized by itching, redness, and eczematous lesions. Interleukin 17 (IL-17) plays a crucial role in the pathogenesis of AD.

Objective: This study aimed to assess the level of IL 17 in AD before and after topical corticosteroid therapy mometasone furoate (Borgasone 1% cream) and compare that to normal control.

Methods: This diagnostic interventional case-control study included 30 patients with AD (Group A) and 30 healthy subjects as controls (Group B). Clinical severity scores were assessed using the SCORAD index, and subjective symptoms were evaluated using a visual analogue scale. Serum IL-17 concentrations were measured using ELISA. All patients received topical mometasone furoate cream for two weeks, and the SCORAD scores were reassessed after treatment.

Results: IL-17 serum levels had significantly decreased after treatment with topical mometasone furoate when compared with before treatment (126.86±10.21 vs 238.12±23.16 pg/ml; P<0.001). Patients with AD had considerably increased IL-17 serum levels when compared with controls (238.12±23.16 vs 101.79±10.94 pg/ml; P<0.001). ROC curve revealed that cut off point 211.5 pg/ml of IL-17 had significant discriminative ability to differentiate between cases with AD and controls with area under the curve 1 (P<0.001) with 96.7% sensitivity and 100% specificity.

Conclusions: The current investigation demonstrated a notable increase in the serum levels of IL-17 among individuals diagnosed with acute and subacute atopic dermatitis, as compared to the control group. Moreover, the topical mometasone furoate administration exhibited a substantial reduction in IL-17 serum levels in AD patients.

Keywords: Interleukin 17; Atopic Dermatitis; Mometasone Furoate; Borgasone.

Introduction

Atopic dermatitis is a chronic inflammatory skin condition characterized by increased levels of IgE in the blood, accumulation of eosinophils in tissues, and excessive growth of the outer skin layer, resulting in skin redness. While atopic dermatitis was initially believed to be driven by Th2 cells due to the presence of elevated eosinophils and IgE in the bloodstream, compelling evidence suggests that the development of skin

lesions in atopic dermatitis is triggered by the activation of Th2 cells in the lymph nodes initially, followed by the involvement of Th1-type cells in the chronic phase of the condition [1].

Human IL-17 was identified in 1995 and characterized as a glycoprotein weighing around 20 kDa (composed of 155 amino acids) that closely resembles murine IL-17. It was found to be primarily produced by activated T cells. IL-17 is released as a homodimer weighing 32 kDa and interacts with the IL-17 receptor (IL-17R), which is a transmembrane protein of type I. The IL-17R is widely distributed across various tissues [2]

In addition to IL-17A, there are five other members of the IL-17 family referred to as IL-17B to IL-17F. These members exhibit conservation in their C-terminal region and also exist as homodimers. It has been established that a specific subgroup of CD4+ T cells, known as Th17 cells, predominantly produce IL-17A and IL-17F. These cells represent a distinct lineage within the CD4+ T cell population [3].

IL-17 plays a diverse role in tissue inflammation by affecting various immune cells and tissue cells. It serves as a crucial mediator in this process. IL-17 is involved in the recruitment of neutrophils, both through the stimulation of granulopoiesis and CXC chemokine induction, as well as by promoting their local survival. Both IL-17A and IL-17F induce the production of different cytokines and chemokines, such as tumor necrosis factor α (TNFα), IL-1b, IL-8, IL-6, GROa, monocyte chemoattractant protein-1 (MCP-1), and granulocyte colony-stimulating factor (G-CSF). Additionally, IL-17A and IL-17F increase the expression of intercellular adhesion molecule-1 (ICAM-1) in monocytes, airway epithelial cells, vein endothelial cells, and fibroblasts [4].

The expression patterns influenced by IL-17 are often potentiated by TNFα and IFNδ. In the context of the skin, IL-17 has been found to impact the production of cytokines and the surface characteristics of epidermal keratinocytes. IL-17 promotes the production of IL-6 and IL-8 in keratinocytes and leads to a mild expression of ICAM-1 and HLA-DR. On the other hand, IL-17 inhibits the production of RANTES induced by IFNδ and TNFα to a significant extent. Moreover, IL-17 has been observed to modulate the function of fibroblasts by stimulating their production of IL-6, IL-8, IL-11, GROa, and G-CSF [5].

In acute lesions of atopic dermatitis, the expression of IL-17 is increased compared to unaffected skin. However, in chronic skin lesions, IL-17 levels are not elevated. While it is possible that IL-17 plays a role in the development of the disease by influencing fibroblasts, infiltrating cells, and keratinocytes, its role in atopic dermatitis appears to be different from other chronic inflammatory diseases where IL-17 levels are increased during the chronic phase [6].

Therefore, we aimed to assess the level of IL 17 in atopic dermatitis before and after topical corticosteroid therapy mometasone furoate (Borgasone 1% cream) and compare that to normal control.

Materials and Methods

This diagnostic interventional case-control study was conducted at the Dermatology Department of 6-October University between October 2022 and April 2023. The study included patients who met the inclusion criteria: those aged 18 years or older, of both genders, and diagnosed with acute and subacute atopic dermatitis.

Exclusion criteria were applied to exclude patients who had received topical or systemic therapy, including phototherapy, for at least 4 weeks prior to enrollment, patients with systemic diseases (such as autoimmune diseases, blood diseases, malignancy), and patients with other dermatological diseases (such as psoriasis).

The study population was divided into two groups: Group A consisted of 30 patients with atopic dermatitis, and Group B consisted of 30 healthy subjects matched for age and sex as the control group. Normal controls were recruited from patients attending plastic surgery for abdominoplasty.

The study received approval from the ethics committee of the Faculty of Medicine with approval number PMC-Me-2211.39. Additionally, approval for the study was obtained from the hospital's ethical committee. Informed written consent was obtained from all participants, and they were informed about the possible complications associated with biopsy procedures, including bleeding, bruising, scarring, and infection. Confidentiality and anonymity of the collected data were ensured.

All subjects underwent a series of assessments and examinations: This included obtaining informed consent, conducting complete history taking, and performing a comprehensive dermatological examination.

A general examination was carried out to assess vital signs such as blood pressure, temperature, heart rate, and respiratory rate, as well as to check for signs of pallor, cyanosis, jaundice, and lymph node enlargement.

Disease severity was evaluated using the SCORAD (SCORing Atopic Dermatitis) index, which provided clinical severity scores. The index utilized a six-area, six-sign scoring system and assessed various signs of atopic dermatitis across different regions of the body. Subjective symptoms, such as itch intensity and sleeplessness, were assessed using a visual analogue scale ranging from 0 (no itch/sleeplessness) to 10 (worst imaginable itch/sleeplessness). All patients were assessed at the first visit and subsequently received topical mometasone furoate cream (Borgasone 0.1%) for two weeks. After two weeks, patients were reassessed using the SCORAD score.

Serological examination was conducted to measure the serum concentration of Interleukin 17 (IL-17) using enzyme-linked immunosorbent assay (ELISA) kits (Human IL-17 Quantikine ELISA Kit D1700, R&D Systems, Minneapolis, MN). All procedures related to serum storage and ELISA kit usage followed the manufacturer's instructions. The quantitative analysis of IL-17 concentration was performed using the Epoch spectrophotometer (BioTek Instruments, Winooski, VT). To ensure result verification, each patient/control subject had two blood samples investigated to ensure good intraindividual reproducibility.

The study aimed to investigate primary outcomes, including the comparison of Interleukin 17 skin tissue levels in atopic dermatitis patients before and after topical mometasone furoate treatment, as well as the comparison of Interleukin 17 levels in atopic dermatitis patients compared to the control group. **Secondary outcome parameters** involved assessing the correlation between the severity and level of Interleukin 17 in the skin tissues of atopic dermatitis patients.

Statistical analysis

SPSS software, version 25 (SPSS Inc., PASW statistics for windows version 25. Chicago: SPSS Inc.), was used for data analysis. Qualitative data were presented using numbers and percentages. For quantitative data, non-normally distributed data were described using median (minimum and maximum), while normally distributed data were described using mean ± standard deviation. Normality of the data was tested using the Kolmogrov-Smirnov test. The significance of the results was determined at the level of ≤ 0.05. To compare qualitative data between groups, Chi-Square and Monte Carlo tests were used as appropriate. For non-normally distributed data, the Wilcoxon signed rank test was used to compare two studied periods. The Student t-test was used to compare two independent groups for normally distributed data. Paired t-test was used to compare two paired readings of distributed data. The Spearman's rank-order correlation was employed to determine the strength and direction of a linear relationship between two non-normally distributed continuous variables and/or ordinal variables.

Results

Regarding demographic data of studied groups: No significant difference was reported between cases and controls regarding age, gender, residence or occupation. **Table 1**

Regarding clinical data of cases: Regarding the site of lesion among studied cases, upper limbs were the most commonly reported site of lesion in 23 (76.7%) patients followed by chest which was reported in 14 (46.7%) patients. The least common sites were face and hand each was reported in 5 (16.7%) patients. **Fig. 1**

Regarding the Extent of lesion, duration and recurrence distribution among studied cases, the median body surface area was 18 ranging between 7 and 60. The median disease duration was 18 years ranging between 5 and 20 years. Recurrence was reported in all studied cases. Regarding the exacerbating factors, *t*he most common exacerbating factor was dryness in 28 (93.3%) patients, followed by exposure to sun in 27 (90%) patients. The least common exacerbating factor was sweating in 5 (16.7%) patients. Regarding the treatment history, *s*ystemic steroids were previously prescribed for 11 (36.7%) patients. Topical cortisone and anti-histaminic drugs were prescribed for 22 (73.3%) patients while topical antihistaminic drugs were prescribed for 6 (20%) patients, 2 (6.7%) patients didn't receive any treatment. **Table 2**

Regarding comorbidities: The most common comorbidity was rhinitis which was reported in 28 (93.3%) patients, followed by asthma which was reported in 23 (76.7%) patients and eye disease which was reported

in 21 (70%) patients. Food allergy was reported in 17 (56.7%) patients. Urticria was reported in 5 (16.7%) patients and 3 (10%) patients had hepatic diseases. The least common comorbidities were diabetes, hypertension and cardiac diseases each was reported in 2 (6.7%) patients. **Fig. 2**

Cutaneous signs: Regarding Cutaneous signs: Pallor was found in 19 (63.3%) patients. LN enlargement was reported in 9 (30%) patients. **Fig. 3**

Comparison of skin manifestations before and after treatment with topical mometasone furoate: There was significant improvement of all skin manifestations including redness, swelling, crustation, scratch marks, Lichenfication, dryness, sleepiness and pruritis after treatment with topical mometasone furoate when compared with before treatment (P<0.001 for all). **Table 3**

Comparison of SCORAD score before and after treatment with topical mometasone furoate: SCORAD score significantly decreased after treatment with topical mometasone furoate when compared with before treatment (25.7 vs 59.7; P<0.001). **Figure 4**

Comparison of IL-17 serum levels before and after treatment with topical mometasone furoate: IL-17 serum levels significantly decreased after treatment with topical mometasone furoate when compared with before treatment (126.86±10.21 vs 238.12±23.16 pg/ml; P<0.001). **Table 4**

Comparison of IL-7 between studied groups: Patients with atopic dermatitis had significantly higher IL-17 serum levels when compared with controls (238.12±23.16 vs 101.79±10.94 pg/ml; P<0.001). **Fig. 5**

Receiver Operating characteristics of IL-7 in differentiating cases and control group: Receiver Operating characteristics revealed that cut off point 211.5 pg/ml of IL-17 had significant discriminative ability to differentiate between cases with atopic dermatitis and controls with area under curve 1 (P<0.001) with 96.7% sensitivity and 100% specificity. **Table 5**

Correlation between IL-17 before and after the treatment and age, duration, scored and skin manifestations: No significant correlation was reported between serum levels of IL-17 with any clinical parameter. There was a significant positive correlation between serum level of IL-17 with pruritis score (r=0.418; P=0.021). Otherwise, no significant correlation was reported between serum levels of IL-17 with other clinical parameters. **Table 6**

Table 1: comparison of sociodemographic characteristics of the studied groups

	Cases group N=30(%)	Control group N=30(%)	Test of significance
Age / years Mean ±SD	26.23±10.56	27.37±9.51	t=0.437 p=0.66
Sex Male Female	16(53.3) 14(46.7)	20(66.7) 10(33.3)	χ^2=1.11 P=0.430
Residence Urban Rural	18(60.0) 12(40.0)	21(70.0) 9(30.0)	χ^2=0.659 P=0.417
Occupation No Employee Physician Student	2(6.7) 6(20.0) 6(20.0) 16(53.3)	2(6.7) 9(30.0) 6(20.0) 13(43.3)	MC=0.910 P=0.823

MC;Monte Carlo test , χ^2= Chi-Square test , t:Student t test

Table 2: extent, duration and recurrence distribution, presence of Exacerbating/ exaggerating factors and treatment history among studied cases

	N=30	%
BSA (extent) Median (min-max)	18(7-60)	
Duration(years) Median (min-max)	18(5-20)	
Recurrence	30	100.0
Exacerbating factors		
Spring	11	36.7
Sweating	5	16.7
Winter	25	83.3
Dryness	28	93.3

Sun	27	90.0
Treatment History		
Systemic steroids	11	36.7
Other medications		
No	2	6.7
Topical cortisone and antihistamine	22	73.3
Topical antihistamine	6	20.0
Phototherapy	0	0.0

Table 3: Comparison of skin manifestations before and after treatment with topical mometasone furoate

	Before	After	Test of significance
Redness			
1	0	13(43.3)	P<0.001*
2	10(33.3)	17(56.7)	
3	20(66.7)	0	
Swelling			
0	2(6.7)	5(16.7)	P<0.001*
1	5(16.7)	23(76.7)	
2	15(50.0)	1(6.7)	
3	8(26.7)	0	
Crustration			
0	0	5(16.7)	P<0.001*
1	7(23.3)	17(56.7)	
2	18(60.0)	8(26.7)	
3	5(16.7)	0	
Scratch marks			
1	8(26.7)	3(10.0)	P<0.001*
2	10(33.3)	15(50.0)	
3	12(40.0)	12(40.0)	
Lichenfication			
0	3(10.0)	10(33.3)	P<0.001*
1	15(50.0)	20(66.7)	
2	12(40.0)	0	
Dryness			
1	3(10.0)	10(33.3)	P<0.001*
2	5(16.7)	14(46.7)	
3	22(73.3)	6(20.0)	
Sleepiness			
0	0	8(26.7)	P<0.001*
1	5(16.7)	14(46.7)	
2	3(10.0)	8(26.7)	
3	2(6.7)	0	
4	12(40.0)	0	
5	5(16.7)	0	
6	3(10.0)	0	
Pruritis			
1	0	5(16.7)	P<0.001*
2	0	11(36.7)	
3	5(16.7)	11(36.7)	
4	3(10.0)	3(10.0)	
5	19(63.3)	0	
7	3(10.0)	0	

#used test Stewart Maxwell test *statistically significant

Table 4: comparison of IL -7 before and after treatment with topical mometasone furoate

	Before	After	test of significance#

Il -17 (pg/ml) Mean±SD	238.12±23.16	126.86±10.21	P<0.001*

Paired t test , *statistically significant

Table 5: validity of IL-17 in differentiating between cases and control groups

	AUC (95%CI)	P value	Cut off point	Sensitivity%	Specificity%
Il -17 (pg/ml)	1.0(1.0-1.0)	<0.001*	211.5	96.7	100.0

AUC: Area under curve, CI: confidence interval.

Table 6: correlation between IL-17 before and after treatment with topical mometasone furoate and age, duration, scored and skin manifestations

Before the treatment		IL.17
Age/years	r	-0.088
	p value	0.645
Duration /years	r	0.057
	p value	0.764
SCORAD	r	-0.049
	p value	0.798
Redness	r	-0.139
	p value	0.464
Swelling	r	-0.151
	p value	0.427
Crustation	r	-0.052
	p value	0.785
Scratch marks	r	-0.001
	p value	0.994
Lichenification	r	-0.042
	p value	0.825
Dryness	r	-0.097
	p value	0.611
Sleepinesss	r	-0.107
	p value	0.572
Pruritis	r	-0.021
	p value	0.910
		IL.17 after treatment
Redness	r	-0.023
	p value	0.903
Swelling	r	0.074
	p value	0.699
Crustation	r	-0.049
	p value	0.799
Scratch marks	r	0.359
	p value	0.051
Lichenification	r	0.094
	p value	0.621
Dryness	r	0.337
	p value	0.069
Sleepliness	r	0.066
	p value	0.729
Pruritis	r	0.418*
	p value	0.021*
SCORAD	r	0.352
	p value	0.056

r: Spearman correlation coefficient, *statistically significant, SCORAD: scoring atopic dermatitis

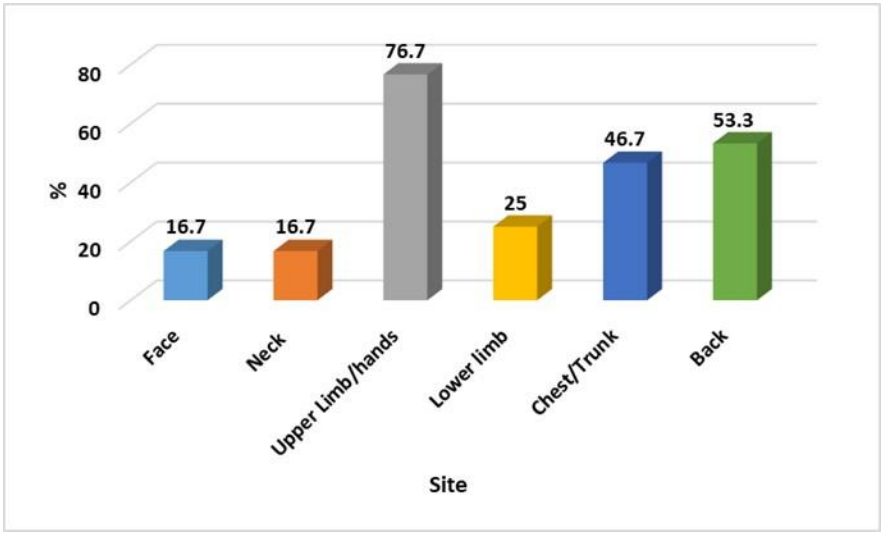

Figure 1: site of the studied lesion

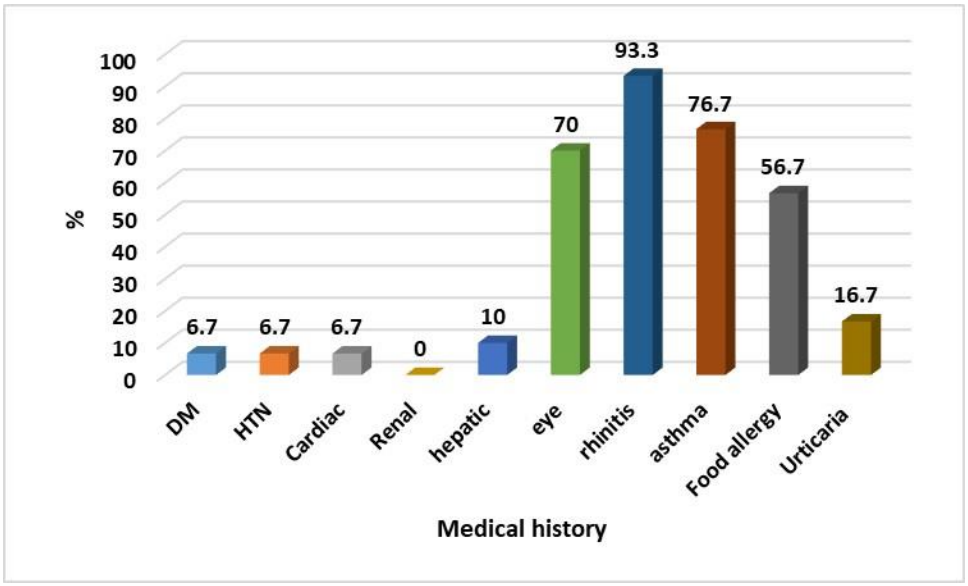

Figure 2: distribution of the studied cases according to medical history

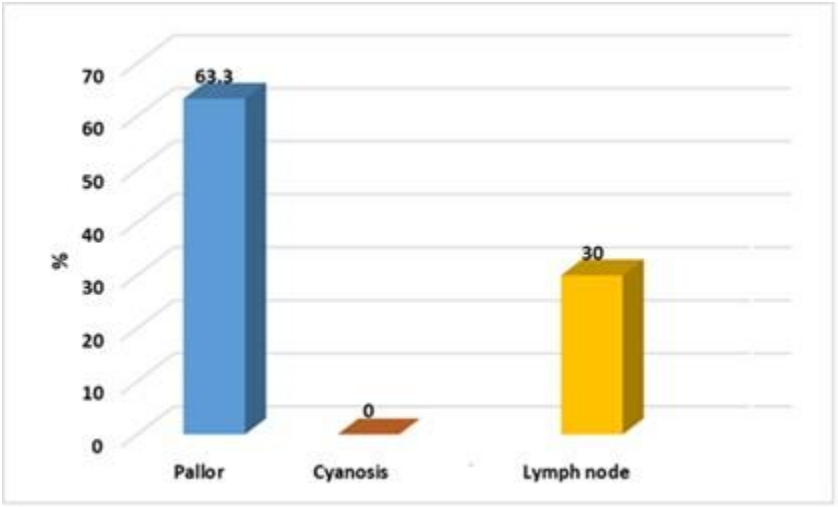

Figure 3: signs of the studied cases

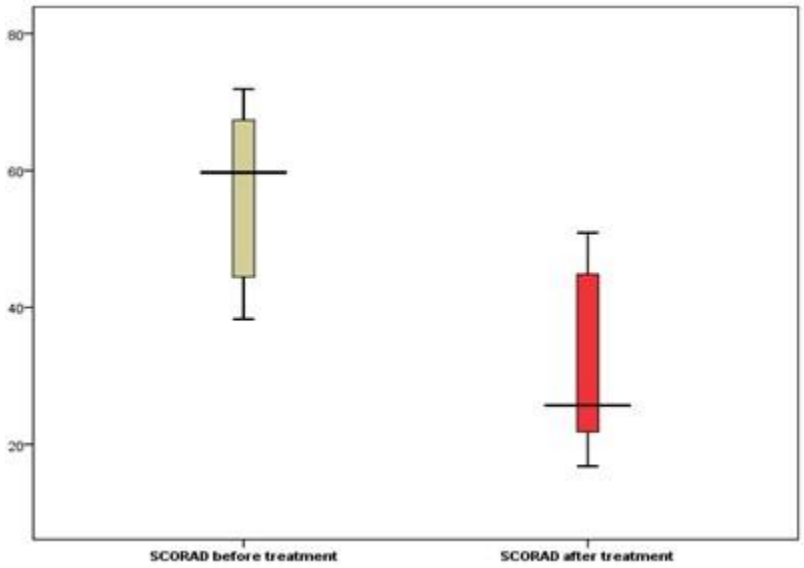

Figure 4: BOX& Whisker plot showing median SCORAD before and after treatment with topical mometasone furoate

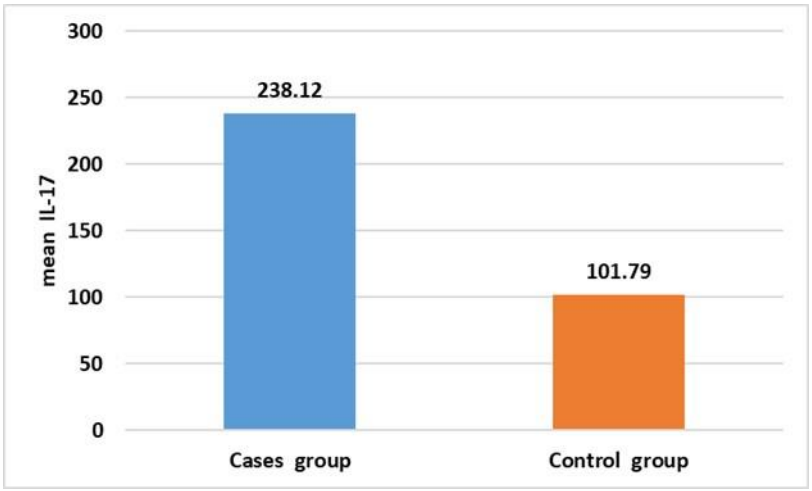

Figure 5: mean IL-17 between cases and control groups

Discussion

The current study aimed to assess level of IL 17 in atopic dermatitis (AD) compared to normal control and to assess the effect of treatment with topical mometasone furoate on patients with AD. To obtain this aim, 30 patients with AD who were attending to 6 October university dermatology department were included and were compared with 30 age and sex matched healthy subjects as control group.

The most common exaggerating factor in our study was dryness in 93.3% of patients, followed by exposure to sun in 90% of patients. In Murota et al. study, typical aggravating factors of AD were irritant dermatitis, food allergy in children, sweating, and psychological stress in adults [7].

Sweating was found to be the least common exacerbating factor in our study, affecting only 16.7% of patients. Sugawara et al. conducted a study and found that individuals with AD had significantly lower levels of sodium, potassium, lactate, urea, and pyrrolidone carboxylic acid in their sweat compared to healthy individuals. This indicates that impaired sweating could potentially decrease the levels of natural moisturizing factors, leading to dry skin in AD patients [8].

Systemic steroids were previously prescribed for 36.7% of patients in our study. Systemic corticosteroids are commonly used as a first-line systemic treatment of AD [9]. A significant number of patients with moderate-to-severe AD require systemic treatment to effectively manage their disease activity. Based on

data obtained from routine clinical care, it has been observed that over 10% of all AD patients receive systemic anti-inflammatory treatment [10].

In the current study, topical cortisone and anti-histaminic drugs were prescribed for 73.3% of patients while topical antihistaminic drugs were prescribed for 20% of patients, 6.7% of patients didn't receive any treatment. The primary or first-line treatment for addressing inflammation in AD is the use of topical glucocorticosteroids [11].

Phototherapy was not prescribed for AD patients in the current study. Phototherapy has been considered an option, based on narrow-band UVB or UVA ultraviolet light [12]. This type of treatment is not free from adverse effects, including photodamage or in long-term effects skin carcinogenesis mainly due to the application of UV light [13].

The most common comorbidity in the current study was rhinitis which was reported in 93.3% of patients, followed by asthma which was reported in 76.7% of patients and eye disease which was reported in 70% of patients. Food allergy was reported in 56.7% of patients. The least common comorbidities were diabetes, hypertension and cardiac diseases each was reported in 6.7% of patients. Consistent with our own findings, Silverberg's research indicates that AD is linked to a higher risk of developing other atopic disorders, such as asthma and food allergies. Additionally, individuals with AD may have a predisposition towards these conditions [14].

According to the present study, there was significant improvement of all skin manifestations including redness, swelling, crustation, scratch marks, Lichenfication, dryness, sleepiness and pruritis after treatment with topical mometasone furoate when compared with before treatment ($P<0.001$ for all). In line with our finding, in a study on 60 patients with AD, Khan et al. reported that topical mometasone furoate 0.1% cream showed a significant improvement in mean scores of erythema, excoriation and papules among patients with AD ($p<0.05$) [15].

SCORAD score in the present study significantly decreased after treatment with topical mometasone furoate when compared with before treatment (25.7 vs 59.7; $P<0.001$). In accordance with our finding, Dähnhardt et al. in a study on 20 patients with AD reported that mean local SCORAD significantly decreased in the mometasone furoate treatment group [16].

In the current study, IL-17 serum levels significantly decreased after treatment with topical mometasone furoate when compared with before treatment (126.86±10.21 vs 238.12±23.16 pg/ml; $P<0.001$). The effect of mometasone furoate on IL-17 was previously reported in allergic diseases. For example, in a study on 50 children with allergic rhinitis, Meng and Ying. (2017) reported that mometasone furoate nasal spray can effectively decrease IL-17 levels. Moreover, in ex vivo human nasal mucosal tissue model, Zhang et al. reported that the use of mometasone furoate decreased the secretion of IL-17 [17].

Patients with AD in the present study had significantly higher IL-17 serum levels when compared with controls (238.12±23.16 vs 101.79±10.94 pg/ml; $P<0.001$).

In agreement with our study, Dewi. found that IL-17 serum levels were higher in AD patients than in control [18].

Moreover, in Baioumy et al. study on 83 patients with atopic dermatitis and 83 controls, serum levels of IL-17A in patients with AD were significantly higher than controls [19].

Furthermore, Tan et al. conducted a study involving 87 children with AD and 60 healthy control subjects. Their findings revealed a significant increase in IL-17 expression within the skin lesions of individuals with AD. However, they were unable to detect IL-17 in the serum of the subjects [20].

In a study on 181 children with atopic eczema/dermatitis disorder, Leonardi et al. found that IL-17 concentrations were significantly higher in the patients with AD than healthy controls [21].

In study by Batista et al., which was conducted on 33 AD patients and 25 controls, they found higher IL17 serum levels in AD patients compared to controls [22].

Additionally, Ma et al. conducted a study involving 181 AD patients and 218 healthy control subjects. Their findings indicated that the levels of IL-17 mRNA and serum concentration were elevated in AD patients. Moreover, they observed a correlation between the levels of IL-17 and the severity of AD, suggesting that IL-17 may play a role in the pathogenesis and progression of the disease [23].

Furthermore, Koga et al. suggested that IL-17 is involved in the development of AD. Through immune-histochemical examination, they observed a more pronounced infiltration of IL-17+ cells in the papillary dermis of atopic eczema, particularly in acute lesions compared to chronic lesions. This finding suggests that IL-17 may play a role in the early stages of AD and its associated inflammatory processes [24].

Moreover, Toda et al. conducted a study to examine the expression of IL-17 in skin biopsy samples obtained from both acute and chronic skin lesions of individuals with AD as well as from healthy volunteers. Their findings revealed increased levels of IL-17 in the skin biopsy specimens from acute AD lesions compared to both chronic AD lesions and the skin samples from healthy volunteers [25].

In the current study, there was a significant positive correlation between serum level of IL-17 with pruritis score after treatment with topical mometasone furoate (r=0.418; P=0.021). In accordance with our study, Ma et al. showed that IL-17 mRNA level and serum concentration were correlated with the severity of AD [23].

Additionally, Dewi, found that the IL-17 serum levels in moderate-severe AD were higher than the mild AD patients [18].

The current study didn't report significant correlation between IL-17 and SCORAD index. In contrast with our study, Dewi, found a robust positive correlation between IL-17 serum level and SCORAD index in adult patients. This finding suggests that IL-17 is a potential targeted therapy to cure AD and the prognostic biomarker to evaluate the severity [18]. Furthermore, Leonardi et al., found that IL-17 serum levels in children with AD were correlated positively with disease severity and SCORAD index [21].

Receiver Operating characteristics revealed that cut off point 211.5 pg/ml of IL-17 had significant discriminative ability to differentiate between cases with AD and controls with area under curve 1 (P<0.001) with 96.7% sensitivity and 100% specificity. In Baioumy et al. study, in ROC curve analysis, IL-17A had a poor predictive ability for the severity of the disease that increased when IL-17A was combined to total IgE [19].

Conclusion

Based on our findings, the present study revealed significant elevation of IL-17 serum levels in patients with acute and subacute atopic dermatitis when compared with controls. In addition, treatment with topical mometasone furoate had significant lowering effect on IL-17 serum levels in patients with atopic dermatitis.

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