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ORIGINAL ARTICLE

Evaluation of The Role of Peroxisome Proliferator Activated Receptor Delta in Psoriasis and Atopic Dermatitis versus Control Subject

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ABSTRACT

Background: Psoriasis as well as atopic dermatitis (AD) are considered chronic inflammatory skin diseases with overlapping immune mechanisms. Peroxisome proliferator-activated receptor delta (PPARδ) has been implicated in skin homeostasis, but its serum levels and relationship to disease activity remain unclear. This work aimed for evaluation of the serum PPARδ levels among psoriatic cases and AD compared to healthy controls, and to investigate the correlation between PPARδ and disease severity.

Methods: This case-control study was performed on 43 individuals involving 14 patients with psoriasis, 14 with atopic dermatitis, and 14 healthy controls who were matched for age and sex. Disease severity was evaluated utilizing the Psoriasis Area and Severity Index (PASI) for psoriasis and Three Item Severity (TIS) score for AD. Serum PPARδ levels were quantified by Enzyme-Linked Immunosorbent Assay (ELISA)..

Results: Serum PPARδ levels were significantly lower in both psoriasis (479.77 ± 175.23 ng/mL) and atopic dermatitis (550.84 ± 174.76 ng/mL) groups compared to controls (794.56 ± 225.19 ng/mL) ($p < 0.001$). Serum PPARδ showed good diagnostic accuracy for distinguishing cases from controls (AUC = 0.855, 95% CI: 0.712–0.944, $p < 0.001$) and psoriasis from atopic dermatitis (AUC = 0.860, 95% CI: 0.718–0.947, $p < 0.0001$), both with high sensitivity and specificity. A strong negative correlation was exhibited between serum PPARδ with disease severity in both groups (psoriasis: $rs = -0.980$, $p < 0.001$; atopic dermatitis: $rs = -0.969$, $p < 0.001$).

Conclusion: Serum PPARδ levels are significantly lower among patients with psoriasis and atopic dermatitis and inversely correlated with disease severity. These findings indicate that serum PPARδ could be used as a useful biomarker for observing disease activity in both diseases.

Keywords: Peroxisome Proliferator Activated Receptor Delta, Psoriasis, Atopic Dermatitis.

INTRODUCTION

Psoriasis is a widespread non-contagious, immune-related skin disease affecting up to 4% of the global population across all age groups. It usually lasts for life, involving mainly the skin and sometimes the joints. Although the exact cause of psoriasis is still unclear, a genetic background has been noted, with HLA antigens (especially HLA-Cw6) strongly linked to increased risk of developing the condition [1]. Psoriasis has five main types: plaque, guttate, flexural, pustular, and erythrodermic. It can be grouped into nonpustular and pustular forms. Common sites for lesions include the scalp, knees, elbows, and folds of skin. Triggers such as stress, skin

trauma (Koebner phenomenon), and infections, particularly streptococcal, can precede disease onset. Factors like infections, stress, seasonal variations, smoking, and obesity may worsen symptoms [2].

The symptoms of psoriasis differ according to its type. Plaque psoriasis is recognized by red, well-defined patches and silvery scales. Other forms may show pustules or shiny lesions with less scaling, often in skin folds. Flare-ups can bring itching and pain, and patients may also experience eye redness or joint pain if comorbidities are present [3].

Atopic dermatitis (AD) is another chronic, relapsing, non-contagious, itchy skin disease. It is very frequent in children but can appear at any age, sometimes starting as early as a few months old and continuing into adulthood. AD is the most common dermatitis, often seen in people with an atopic tendency meaning a personal or family history of AD, asthma, or allergic rhinitis is typical [4]. The disease results from complex interactions between genetic as well as environmental factors, especially issues in the skin barrier, which make the skin more vulnerable to the irritants and infections, mainly by Staphylococcus aureus, which can further increase inflammation [5].

Clinically, AD can be acute or chronic, sometimes alternating between inflamed, red, blistered patches and dry, thickened, itchy skin. Persistent itching is often the main complaint. The appearance and location of eczema can vary by age group, with typical patterns on the cheeks, arms, and legs in infants, and on flexures or hands in older children and adults [6].

Peroxisome proliferator-activated receptors (PPARs) are fatty acid-activated nuclear hormone receptors that regulate lipid and glucose metabolism and are vital for skin homeostasis. There are three main types: PPARα, PPARβ/δ, and PPARγ [7]. Of these, PPARδ is found throughout the skin in cells like keratinocytes, fibroblasts, and melanocytes, and is the most prevalent form in human keratinocytes. When activated, PPARδ promotes expression of skin differentiation markers such as transglutaminase 1 and involucrin [8].

Recent studies have shown that PPARδ is upregulated in psoriatic and atopic dermatitis lesions. However, its precise therapeutic and pathogenic role in these skin diseases remains underexplored. Interestingly, both activating and blocking PPARδ have been reported to reduce skin inflammation [8].

Despite advances in understanding the molecular basis of psoriasis and atopic dermatitis, the exact contribution of PPARδ to their pathogenesis and severity is still unclear, so this study aimed for evaluation of the serum PPARδ levels among psoriatic cases and AD compared to healthy controls, and to investigate the correlation between PPARδ and disease severity.

METHODS

This case-control study was performed on 42 individuals at the Dermatology, Venereology, and Andrology Department, Faculty of Medicine, Zagazig University Hospitals from June 2024 to June 2025, the sample size included three groups: Group A (14 patients who had psoriasis, 7 males and 7 females), Group B (14 patients who had atopic dermatitis, 7 males and 7 females), and Group C (14 apparently healthy controls, 5 males and 9 females), matched for age and sex.

Institutional Review Board (ZU-IRB#10401-7-2-2023) clearance was obtained and informed consent was collected from all patients who participated in the study. The research was performed following the World Medical Association's Code of Ethics (Helsinki Declaration) for studies involving human subjects.

The study involved subjects of both sexes aged 20 to 40 years were eligible. Groups A and B consisted of 14 patients each with persistent lesions of either atopic dermatitis or psoriasis. Group C included 14 healthy controls with no history of inflammatory skin disease. Diagnosis of atopic dermatitis was based on clinical criteria, including a history of chronic or relapsing pruritic eczema with typical

distribution and morphology, often supported by personal or family history of atopy . Persistent psoriatic lesions were diagnosed clinically by the presence of well-demarcated erythematous plaques with silvery scales, mainly over extensor surfaces [9,10] and confirmed by a dermatologist.

Patients who had received any systemic or topical therapy for six weeks prior to their clinic visit, those who had chronic systemic diseases like hypertension, diabetes mellitus, liver, kidney, or cardiac diseases, malignant skin disorders, as well as pregnant or lactating women were excluded.

Before collecting blood samples, all subjects went through a thorough process. Each participant provided informed consent after receiving information about the study. A detailed history was obtained, covering age, sex, occupation, and relevant demographic data. Further questions included disease onset, duration, course, previous and current treatments, other medical conditions, and any family history of psoriasis or atopic dermatitis.

The Psoriasis Area and Severity Index (PASI) was used to evaluate the severity of psoriasis in patients. This index takes into account the area of lesions and other plaque characteristics [9,10]. The Three Item Severity (TIS) score was used to compare the severity of erythema, edema/papulation, and excoriations across representative lesions in atopic dermatitis. A total score can be anything from 0 to 9, with each item receiving a score between 0 and 3.

Venous blood samples (4 mL) were collected from all subjects in clot-activator tubes, centrifuged at 3000 rpm for 15 minutes to separate the serum, and stored in Eppendorf tubes at -80°C until testing.

Serum PPARδ levels were assessed utilizing a quantitative sandwich enzyme-linked immunosorbent assay (ELISA) kit (Sunred Biotechnology, China; Cat. No. 201-12-4526). The standard assay range was 20–6000 pg/mL, with a sensitivity of 19.638 pg/mL. The assay followed the manufacturer's protocol.

First, standards were prepared by serial dilution. For each plate, standards and samples were added to appropriate wells, along with biotin-labeled antibodies and Streptavidin-HRP for immune complex formation. Plates were incubated, washed, and chromogen solutions A and B were added. After incubation in the dark, stop solution was added, causing a color change from blue to yellow. Optical density (OD) was read at 450 nm within 15 minutes. The concentration of PPARδ in each sample was determined using a standard curve plotted from OD values of the standards.

STATISTICAL ANALYSIS

Statistical analysis was performed using IBM SPSS Version 25. Data normality was assessed with the Shapiro-Wilk test. Descriptive statistics were reported as mean ± SD, median (range), or frequency (%). Student's t-test, Mann-Whitney U, one-way ANOVA, and Kruskal-Wallis tests were used for group comparisons as appropriate, while chi-square tested associations between categorical variables. Correlations between quantitative variables were analyzed, and ROC curves evaluated diagnostic accuracy. When p value was lower than 0.05 it was deemed as significant.

RESULTS

The psoriasis group patients had significantly higher mean age (32.50 ± 7.27 years) compared to the atopic dermatitis (24.57 ± 6.41 years) and control groups (25.00 ± 4.22 years) ($p < 0.001$). Gender distribution did not differ significantly between the 3 studied groups ($p = 0.681$), with equal numbers of males and females in the psoriasis and atopic dermatitis groups, and a slightly higher proportion of females in the control group (64.3%). Regarding serum PPARδ levels, the control group had significantly higher mean levels (794.56 ± 225.19 ng/mL) than both the psoriasis (479.77 ± 175.23 ng/mL) and atopic dermatitis groups (550.84 ± 174.76 ng/mL) ($p < 0.001$) (Table 1).

In the psoriasis group, the mean PASI score was 9.54 ± 6.29 (median 8.65; range 1.40–22.40), with 35.7% of patients classified as mild, 35.7% as moderate, and 28.6% as severe. Psoriasis vulgaris was the most common

clinical variant (78.6%), followed by guttate (14.3%) and palmoplantar psoriasis (7.1%). The most frequently affected sites were the elbows (78.6%) and knees (57.1%), while lower back, trunk (21.4% each), nails (14.3%), palms and soles, full-body involvement, and scalp (7.1% each) were less commonly involved (Table 2).

In the atopic dermatitis group, the mean Tis score was 6.50 ± 1.22 (median 6.00; range 5.00–9.00), with 57.1% of patients classified as moderate and 42.9% as severe. The most commonly affected areas were the face (57.1%), feet (50.0%), inner elbows (42.9%), and behind the knees (42.9%), while the hands (35.7%) and neck (7.1%) were less frequently involved (Table 3).

The duration of illness was significantly longer in the atopic dermatitis group (mean 9.93 ± 4.32 years) compared to the psoriasis group (mean 5.07 ± 5.22 years; $p < 0.001$) with non-significant differences in family history between the groups, with most patients reporting a positive family history in both psoriasis (71.4%) and atopic dermatitis (78.6%) ($p = 1.000$) or the extent of disease with a mean extent of 35.0% ± 21.75 in psoriasis and 29.64% ± 24.37 in atopic dermatitis ($p = 0.574$) (Table 4).

Serum PPARδ demonstrated good diagnostic validity for distinguishing cases from controls, with an AUC of 0.855 (95% CI: 0.712–0.944, $p < 0.001$) at a cutoff value of ≤655.98 ng/mL, achieving both sensitivity and specificity of 85.7%. For differentiating psoriasis from atopic dermatitis, the AUC was 0.860 (95% CI: 0.718–0.947, $p < 0.0001$), with the same cutoff yielding a sensitivity of 89.3% and specificity of 85.7% (Table 5, Figure 1).

Significant negative correlations were exhibited between serum PPARδ levels and disease severity, as indicated by PASI in the psoriasis group ($rs = -0.980$, $p < 0.001$) and Tis in the atopic dermatitis group ($rs = -0.969$, $p < 0.001$), so lower levels of serum PPARδ were associated with greater disease severity in both psoriasis and atopic dermatitis (Table 6).

Table 1: Comparison between studied groups according to demographic data and serum PPARδ (n=42)

		Psoriasis	**Atopic dermatitis**	**Control**	**Test , p value**
		n=14	**n=14**	**n=14**	
Age (years)	Mean±SD	32.50 ± 7.27	24.57 ± 6.41	25.00 ± 4.22	F: 10.011, p<0.001*
	Median (Range)	35.00 (20.00-40.00)	21.00 (20.00-40.00)	24.50 (20.00-33.00)	
Gender	Female	7(50.0%)	7(50.0%)	9(64.3%)	X2: 0.769, p=0.681
	Male	7(50.0%)	7(50.0%)	5(35.7%)	
Serum PPARδ (ng/mL)	Mean ± SD	479.77 ± 175.23	550.84 ± 174.76	794.56 ± 225.19	H: 14.952, p<0.001*
	Median (Range)	505.27 (117.64-738.36)	577.43 (188.66-808.36)	855.70 (308.64-1010.22)	

F: One way ANOVA test, X2: Chi square test, H: Kruskal wallis test, * for significant p value (<0.05)

Table 2: PASI score, Clinical variants, and affected areas in psoriasis group (n=14).

		Psoriasis
		n=14
PASI	Mean ± SD	9.54 ± 6.29
	Median (Range)	8.65 (1.40-22.40)
PASI grades	Mild	5(35.7%)
	Moderate	5(35.7%)
	Severe	4(28.6%)
Clinical variant	Guttate Psoriasis	2(14.3%)
	Palmoplantar Psoriasis	1(7.1%)
	Psoriasis Vulgaris	11(78.6%)

Sites affected	Elbows	11 (78.57%)
	Knees	8 (57.14%)
	Lower back	3 (21.43%)
	Trunk	3 (21.43%)
	Nails	2 (14.29%)
	Palms and soles	1 (7.14%)
	Full-body involvement	1 (7.14%)
	Scalp	1 (7.14%)

Table 3: Atopic dermatitis classification, affected areas according to Tis score (n=14).

		Atopic dermatitis
		n=14
Tis	Mean ± SD	6.50 ± 1.22
	Median (Range)	6.00 (5.00-9.00)
Tis grades	Moderate	8(57.1%)
	Severe	6(42.9%)
Sites affected	Face	8 (57.14%)
	Feet	7 (50.00%)
	Inner elbows	6 (42.86%)
	Behind knees	6 (42.86%)
	Hands	5 (35.71%)
	Neck	1 (7.1%)

Table 4: Comparison between cases groups according to duration of illness, percentage of family history, percentage of extent of disease (n=28).

		Psoriasis	**Atopic dermatitis**	**Test , p value**
		n=14	**n=14**	
Duration (Years)	Mean ± SD	5.07 ± 5.22	9.93 ± 4.32	Z: -2.711, p<0.001*
	Median (Range)	3.00 (0.04-16.00)	9.00 (5.00-20.00)	
Family history	Negative	4(28.6%)	3(21.4%)	X2: 0.000, p=1.000
	Positive	10(71.4%)	11(78.6%)	
Extent (%)	Mean ± SD	35.00 ± 21.75	29.64 ± 24.37	Z: 0.574, p=0.574
	Median (Range)	35.00 (10.00-70.00)	20.00 (10.00-90.00)	

Z: Mann Whitney test , X2: Chi square test, * for significant p value (<0.05)

Table 5:Validity of Serum PPARδ for discrimination between study groups.

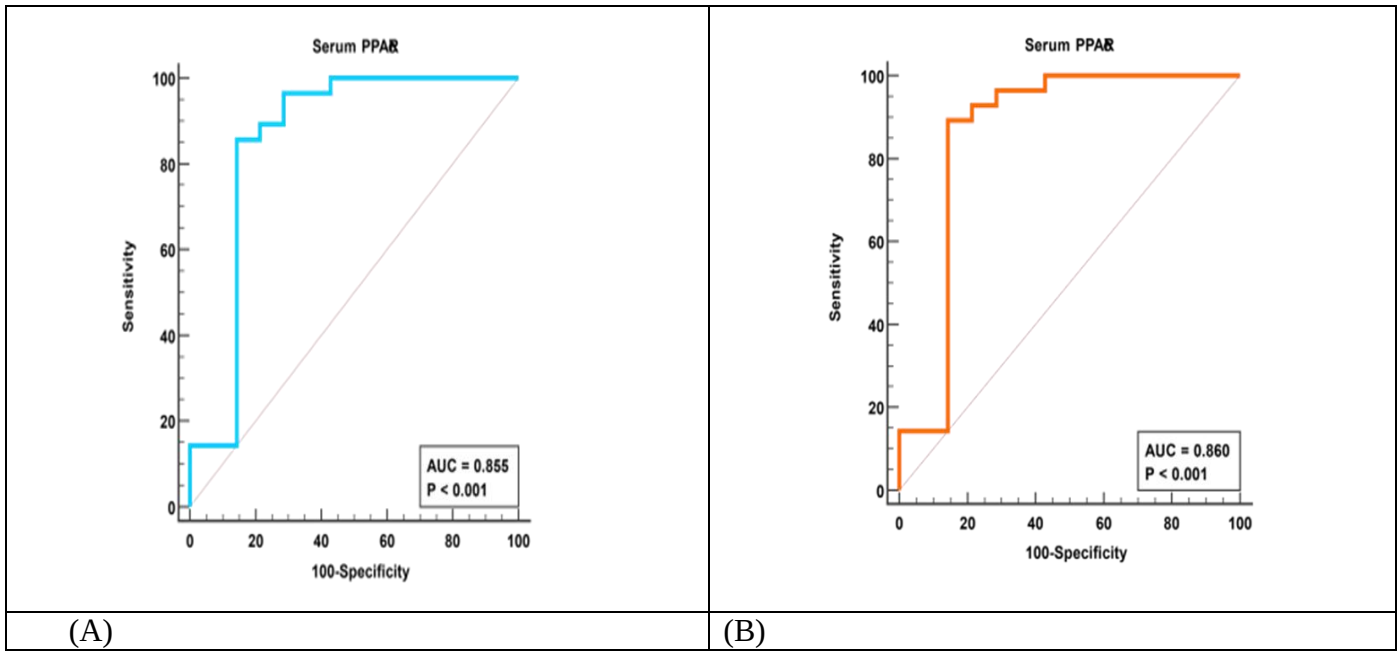
	AUC	**95% CI**	**p**	**Cutoff**	**Sensitivity (%)**	**Specificity (%)**
Cases vs control	0.855	0.712 to 0.944	<0.001*	≤655.98	85.71	85.71
Psoriasis vs atopic dermatitis	0.860	0.718 to 0.947	<0.0001	≤655.98	89.29	85.71

AUC, area under ROC curve; CI, confidence interval; *: Significant ≤0.05

Table 6: Correlation between Serum PPARδ and disease severity.

	rs	p
PASI in psoriasis group	-0.980	<0.001*
Tis in atopic dermatitis	-0.969	<0.001*

rs: Spearman correlation coefficient, *: Significant ≤0.05



(A) (B)

Figure 1: (A) Validity of Serum PPARδ for discrimination betweencases and control, (B) Validity of Serum PPARδ for discrimination between psoriasis and atopic dermatitis groups.

DISCUSSION

As a systemic inflammatory disease, psoriasis affects around 2% to 3% of the global population. The onset and progression of psoriasis depend on a complex combination of genetic and environmental factors that push individuals towards developing the characteristic psoriatic features, as described by Orzan et al. [12].

Atopic dermatitis, on the other hand, is a long-lasting and recurrent skin disorder with many causes. It arises from interactions between factors like skin barrier dysfunction, immune system imbalance, nervous system involvement, and different reactions to environmental antigens. All these are more likely to occur in people who are genetically susceptible, according to Criado et al. [13].

The peroxisome proliferator-activated receptors are a group of ligand-activated nuclear hormone receptors. This family includes PPARα, PPARβ/δ, and PPARγ. Once activated by a ligand, PPARs bind together with retinoid X receptor (RXR), forming a complex that can regulate the expression of different genes, as stated by Qiu et al. [14].

Analysis of the current results showed that the average age of patients with psoriasis was 32.5 years, while those with atopic dermatitis averaged 24.6 years, and controls had a mean age of 25 years. Gender distribution was balanced in both psoriasis and atopic dermatitis groups, with the control group having a slight female predominance (64.3%). Similar age patterns were observed in the study by Armstrong et al. [5], who found the mean age of psoriasis patients to be 32.8 years. Pala et al. [15] also reported that psoriasis cases had a mean age of 39.6 years, with a little more than half being women. El Eishi et al. [16] reported

a mean age of 38.1 years among their psoriasis cases. For atopic dermatitis, Holm et al. [17] found that patients had an average age of 26 years, with females making up the majority.

Most psoriasis cases in this study were psoriasis vulgaris (78.6%), while guttate psoriasis made up 14.3%, and palmoplantar psoriasis was seen in 7.1%. The most affected sites in the psoriasis group were the elbows (78.6%) and knees (57.1%), followed by lower back, trunk (21.4% each), and nails (14.3%). Less common locations included palms and soles, full body, and scalp, each seen in just one case (7.1%). Chen [18] reported different rates, with 55% plaque, 14% palmar, and 23% nail involvement.

The mean PASI score for psoriasis patients was 9.54, ranging from 1.40 to 22.40, which shows a broad variability in disease severity. About 35.7% of psoriasis patients had mild disease, 35.7% moderate, and 28.6% severe psoriasis. These results are in line with the findings by Hägg et al. [19], who showed mean PASI scores of 7.5 for men and 5.4 for women. Yeung et al. [20] also described variability in severity with 51.8% mild, 35.8% moderate, and 12.4% severe cases.

For the atopic dermatitis group, the most commonly affected areas were the face (57.1%), feet (50.0%), inner elbows and behind the knees (42.9% each), hands (35.7%), and the neck (7.1%). Chovatiya and Silverberg [21] similarly observed common involvement of flexural areas (58%), face and neck (42%), and hands and feet (36%).

When looking at serum PPARδ levels, the control group had significantly higher average values (794.56 ng/mL) compared to both the psoriasis group (479.77 ng/mL) and the atopic dermatitis group (550.84 ng/mL). This means that patients with psoriasis and atopic dermatitis had notably lower serum PPARδ levels than healthy individuals.

Analysis of the diagnostic value of serum PPARδ showed good accuracy in distinguishing between the groups. The area under the curve (AUC) was 0.855 for separating patients (psoriasis and atopic dermatitis) from controls, and 0.860 for telling psoriasis apart from atopic dermatitis, showing high diagnostic ability in both situations.

Further, when we checked the link between serum PPARδ levels and disease severity, there were significant negative correlations. In other words, lower serum PPARδ was associated with higher PASI scores in psoriasis patients and higher TIS scores in those with atopic dermatitis, pointing to more severe disease as PPARδ dropped.

As far as we know, this is the first study at Zagazig University to measure serum PPARδ in both psoriasis and atopic dermatitis patients together and compare it with healthy controls. Most previous research focused on tissue levels of PPARδ in normal and diseased skin, not the serum. In fact, nearly all earlier studies looked at PPARδ activity in lesions, not the blood.

For instance, Nijland et al. [22] found that PPARδ seems to be highly activated in psoriatic skin, as its target genes, like CD36 and FABP5, are strongly upregulated in these lesions. Morin et al. [23] also reported increased PPARδ expression in human psoriatic plaques compared to normal skin, and Kim et al. [24] demonstrated in mouse models that activating PPARδ worsens psoriasiform inflammation. Moreover, El Eishi et al. [16] compared PPARδ levels in skin tissue of psoriasis cases before and after management using methotrexate and PUVA, showing a significant drop after treatment, but not back to normal control levels. Töröcsik et al. [25] noted that certain endogenous ligands of PPARδ, such as arachidonic acid, PGF2, and 5-HETE, are elevated in the lesional skin of atopic dermatitis than healthy skin.

Some limitations exist in this study. Including the small sample size. The study was also limited in its subject diversity due to the fact that it was carried out at a single center.In addition, only serum PPARδ levels were assessed, without parallel tissue or genetic analyses, so we could not directly compare blood findings with changes at the skin level. Larger multicenter studies, including both serum and tissue samples, are needed to further

clarify the role of PPARδ in psoriasis as well as atopic dermatitis.

CONCLUSION

Serum PPARδ levels are significantly lower in patients with psoriasis and atopic dermatitis and inversely correlated with disease severity. These findings indicate that serum PPARδ could serve as a useful biomarker for observing disease activity in both diseases.

Conflict of Interest or financial disclosure: No potential conflict of interest to be reported by the authors.

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