International Journal of Dermatology, Venereology and Leprosy Sciences

E-ISSN: 2664-942X
P-ISSN: 2664-9411
Impact Factor (RJIF): 5.67
www.dermatologypaper.com
Derma 2026; 9(1): 21-24
Received: 12-10-2025
Accepted: 16-11-2025

Shimaa El Sayed Hussein Eissa
Dermatology & Venereology, Faculty of Medicine, Tanta University, Egypt

Nesrin Sabry Radwan
Dermatology & Venereology, Faculty of Medicine, Tanta University, Egypt

Marwa Mohamed Atef
Dermatology & Venereology, Faculty of Medicine, Tanta University, Egypt

Zeinab Abd-Alsamad Ibrahim
Dermatology & Venereology, Faculty of Medicine, Tanta University, Egypt

Corresponding Author:
Shimaa El Sayed Hussein Eissa
Dermatology & Venereology, Faculty of Medicine, Tanta University, Egypt

Study of Serum Level of Lysosomal Associated Membrane Protein 2 in Psoriatic Patients

Shimaa El Sayed Hussein Eissa, Nesrin Sabry Radwan, Marwa Mohamed Atef and Zeinab Abd-Alsamad Ibrahim

DOI: https://www.doi.org/10.33545/26649411.2026.v9.i1a.256

Abstract
Introduction: Although psoriasis is a prevalent dermatological condition, its precise pathogenic mechanisms remain incompletely elucidated. Dysregulated autophagy has been increasingly recognized as contributing to the pathogenesis of certain skin disorders. LAMP2 is a key regulator within the autophagic machinery.
Objective: The objective of this work was to estimate serum level of LAMP2 in psoriatic individuals in a trial to elucidate its function in etiopathogenic pathways involved in the disease.
Patients and methods: This prospective case-control study encompassed 26 participants with different clinical types of psoriasis, and 26 healthy participants, matched for age and sex, were enrolled as the control group. Venous specimens drawn from peripheral sites (5 mL each) were obtained from all participants to determine serum LAMP2 levels using an ELISA assay.
Results: Serum LAMP2 levels were markedly elevated in patients (mean ± SD: 27.37 ± 33.25) compared to controls (3.03 ± 1.32) with high statistical significance (P<0.001).
Conclusion: LAMP2 could be incriminated in pathogenesis of psoriasis manifested by elevated serum levels of LAMP2 in psoriatic participants relative to the control group.

Keywords: Psoriasis, Serum, LAMP2

Introduction

Psoriasis is a prolonged inflammatory dermatological disorder defined by a complex, immune-mediated pathogenesis. It arises from the dynamic interaction of multiple genetic predispositions, environmental factors, and dysregulated immune responses. This condition is relatively common, affecting about 2% of the global populace [1].

The etiopathological processes driving psoriasis is mediated by dysregulation of various T-cell subsets, comprising T-helper (Th) 1, Th2, Th17, Th22, and regulatory T cells, leading to aberrant secretion of accompanied cytokines as IFN-γ, tumor necrosis factor (TNF)-α, and members of the IL-23 and IL-17 families, indicative of impaired immune regulatory mechanisms [2].

Autophagy is a cellular process that regulates cytokine release, supports lymphocyte viability and lineage specification, facilitates major histocompatibility complex (MHC) antigen presentation, promotes clearance of cells undergoing apoptosis, and modulates pro-inflammatory molecular pathways [3]. Impaired autophagy has been evidenced to promote the synthesis of pro-inflammatory cytokines and stimulate keratinocyte proliferation [4, 5].

Lysosome-associated membrane protein 2 (LAMP2), also referred to as CD107b, is a 410–amino acid, highly glycosylated lysosomal membrane protein. It serves as a key regulator of autophagy and, together with LAMP1, represents a substantial proportion of the total lysosomal membrane glycoproteins [6].

LAMP1 and LAMP2 have been observed to aggregate in primary keratinocytes experiencing lysosomal dysfunction following sustained contact with pro-inflammatory cytokine TNF-α. This may participate in the etiopathogenesis of persistent inflammation conditions like psoriasis [7].

Patients and Methods

This prospective case control study was executed on 52 subjects, 26 subjects presented with

different clinical types of psoriasis as patient's group and 26 apparently healthy individuals as controls who were selected from the Dermatology and Venereology Outpatient Clinic, Tanta University Hospital during the period from the 1st of July 2023 to the 31st of August 2024.

The authorization was attained from research Ethics Committee by Faculty of Medicine Tanta University Hospitals, authorization code No: (36264MS223/6/23).

Participants diagnosed clinically as psoriasis, who had not received any treatment for psoriasis within the four weeks preceding the study, who agreed to join the study and gave informed consent were included. While those with history of bleeding, lactating or pregnant females or participants with record of chronic debilitating diseases, autoimmune disorders, dermatological or other systemic disease which might alter serum LAMP2 level were excluded.

All participants underwent a comperehensive history taking, general and dermatological examination, dermoscopic examination of the lesions and Clinical assessment of the patients was made by the intensity of the disorder, as determined by Psoriasis Area and Severity Index (PASI) score.

Peripheral venous blood samples (5 mL) were obtained from all participants to determine serum LAMP2 levels via an enzyme-linked immunosorbent assay (ELISA). The samples were transferred into disposable plastic tubes and allowed to clot, followed by centrifugation at 1200 rpm for 10–15 minutes to obtain the serum. The resulting sera were divided into aliquots and preserved at –80 °C until analysis.

Estimation of serum LAMP2 level

Human LAMP2 ELISA Kit supplied by BT LAB, Jiaxing, Zhejiang, China Catalog No: E0348 Hu was applied for quantitative quantification of serum LAMP2 level

Test Principle

- The kit is an ELISA for the quantitative in vitro determination of LAMP2.
- The plate provided in this kit coated in advance with Human HLAMP-2 antibody.
- HLAMP-2 contained in the sample was incorporated and allowed to bind to antibodies immobilized on the wells.
- Biotinylated Human HLAMP-2 Antibody was introduced and allowed to bind to HLAMP-2 in the sample.
- Streptavidin-HRP was subsequently added to bind the biotinylated HLAMP-2 antibody.
- Unbound Streptavidin-HRP was washed away after incubation.
- A substrate solution was then applied, and the resulting colorimetric change occurred proportionally to the concentration of human HLAMP-2.

The reaction was stopped by adding an acidic stop solution, and the absorbance was measured at 450 nm.

Reagent Preparation

The standard solution (48 ng/mL, 120 µL) was prepared in 120 µL of the standard diluent to prepare a 24 ng/mL stock standard solution. The mixture was left to stand for 15 minutes with soft mixing before preparing serial dilutions. Duplicate standard points were generated by performing 1:2 serial dilutions of the stock solution using standard diluent to yield concentrations of 12 ng/mL, 6 ng/mL, 3 ng/mL, and 1.5 ng/mL. Standard diluent was used as the zero standard (0 ng/mL). The Wash Buffer (1×) was prepared by 25-fold dilution of 20 mL Wash Buffer Concentrate with deionized or distilled water, yielding a total volume of 500 mL.

1. Reagents, standards, and samples were prepared following the manufacturer's guidelines, and the assay was carried out at ambient temperature.
2. Fifty microliters of each standard solution was dispensed into the designated standard wells.
3. Forty microliters from each sample were dispensed to the sample wells, and then 10 µL of anti-HLAMP-2 antibody. Subsequently, 50 µL of streptavidin-HRP was dispensed to both sample and standard wells, and the contents were thoroughly mixed. After sealing, the plate underwent incubation at 37 °C for 60 minutes.
4. The plate underwent five consecutive washing steps using wash buffer, with each well soaked in 300 µL of buffer for 30 seconds to 1 minute per wash.
5. Fifty microliters of substrate solution A were dispensed to each well, and then by 50 µL of substrate solution B, and the plate underwent incubation for 10 minutes at 37 °C in the dark.
6. Fifty microliters of stop solution were dispensed to each well, resulting in an immediate color shift from blue to yellow was observed.
7. The optical density (OD) of each well was quantified at 450 nm via a microplate reader within 10 minutes of introducing the stop solution.

Calculation of Results

A standard curve was built by plotting the mean OD of each standard on the vertical (Y) axis against its corresponding concentration on the horizontal (X) axis, with a fitted curve generated through the points. These calculations were carried out via computer-based curve-fitting software, and the optimal fit was determined via regression analysis.

Statistical analysis of the data

Data were entered and analyzed via IBM SPSS Statistics for Windows, version 20.0 (Armonk, NY: IBM Corp., 2011). Categorical variables were shown as frequencies and percentages. The Shapiro–Wilk test was applied to determine the normality of data distribution. Continuous variables were summarized via range (minimum–maximum), mean ± standard deviation, median, and interquartile range (IQR). Statistical significance was set at $p < 0.05$.

The used tests were

1. Chi-square test

For categorical variables, to relate among different groups

2. Monte Carlo correction

Correction for chi-square in cases where >20% of cells show anticipated counts <5

3. Student t-test

For normally distributed quantitative variables, to relate among two studied groups

4. Mann Whitney test

For abnormally distributed quantitative variables, to relate among two studied groups

5. Wilcoxon signed ranks test
For abnormally distributed quantitative variables, to relate among two periods.

Results
This study was executed on 26 psoriatic participants and 26 matched controls; Regarding the estimation of serum LAMP2 level in patients, it ranged between 3 and 114.5 with a mean ± SD of 27.37 ± 33.25. While in controls, it ranged between 0 and 5.1 with a mean ± SD of 3.03 ± 1.32.
There was a notable statistically significant difference among the psoriasis group and the control group in terms of serum level of LAMP2 (P<0.001) (Table 1).

Table 1: Comparison among the two studied groups in terms of serum LAMP2 level

	Patient (n = 26)	Control (n = 26)	U	p
Serum LAMP2 level				
Min. – Max.	3.0 –114.50	0.0 – 5.10	71.500	<0.001*
Mean ± SD.	27.37 ± 33.25	3.03 ± 1.32		
Median (IQR)	6.8 (4.7 – 50.2)	3.1 (2.0 – 4.0)		

Regarding the correlation between serum LAMP2 and clinical data of the studied participants there were non-significant link among serum LAMP2 and age or PASI score (r_s = 0.092 and r_s = 0.224) respectively, and (p = 0.655 and p =0.272) respectively (Table 2).

Table 2: Correlation among Serum LAMP2 level with different parameters in patient group (n = 26)

	Serum LAMP2 level	
	r_s	p
Age	0.092	0.655
PASI score	0.224	0.272

Discussion
Psoriasis represents an immune-mediated condition defiened by the stimulation of keratinocytes and immune cells, leading to keratinocyte hyperproliferation and the formation of well-demarcated erythematous scaly plaques [1] The underlying mechanisms contributing to the development of psoriasis is influenced by a range of factors, as immune dysregulation, genetic predisposition, exogenous stimuli, and metabolic disturbance [8] The role of autophagy in the emergence of psoriasis has recently gained recognition. Dysregulation of molecules involved in autophagy and lysosomal signaling cascades has also been observed in this condition [9].
The LAMP2 is a heavily glycosylated protein found primarily in the lysosomal membrane of nearly all cell types, with varying levels depending on tissue, cell type, and physiological condition. It is integral to the autophagy-lysosomal pathway (ALP), which is crucial for cellular homeostasis. Dysfunction in ALP has been implicated in psoriasis [9]. Chaperone-mediated autophagy dysfunction due to altered LAMP2 expression may lead to accumulation of cytotoxic protein aggregates, triggering inflammatory responses in the skin [10].
There is limited direct evidence specifically addressing LAMP2 expression in psoriasis skin. LAMP1 and LAMP2 have been observed to accumulate under conditions of lysosomal dysfunction in neurons exposed to stress. [11], [7] confirmed the lysosomal dysfunction in primary keratinocytes following prolonged exposure to the inflammatory cytokine TNF-α. in psoriasis and atopic dermatitis. There was TNF-α– induced induction of both LAMP1 and LAMP2 in primary KCs sequentially relative to controls which was significant.
The present study was the first, up to our knowledge, to provide information on the serum concentration of LAMP2 in Ps participants. In accordance with the results of this study similar data are reported in other pathologies of inflammatory conditions like small and medium vessel vasculitis and systemic lupus erythematosus. [12] reported that the serum LAMP2 levels in SMVV individuals were elevated relative with controls. Moreover, participants in the active disease stage had higher LAMP2 levels than those in remission. The inflammatory nature of vasculitis is somewhat comparable to psoriasis.

Conclusions
LAMP2 might contribute to the pathogenesis of psoriasis manifested by elevated serum levels of LAMP2 in psoriatic patients in comparison to control group.

Financial support and sponsorship: Nil

Conflict of Interest: Nil

References
1. Yamanaka K, Yamamoto O, Honda T. Pathophysiology of psoriasis: A review. Dermatol J. 2021;48:722-31.
2. Deng Y, Chang C, Lu Q. The Inflammatory Response in Psoriasis: a Comprehensive Review. Clin Rev Allergy Immunol. 2016;50:377-89.
3. Doria A, Gatto M, Punzi L. Autophagy in human health and disease. N Engl J Med. 2013;368:1845.
4. Lee H-M, Shin D-M, Yuk J-M, Shi G, Choi D-K, Lee S-H, *et al*. Autophagy negatively regulates keratinocyte inflammatory responses via scaffolding protein p62/SQSTM1. J Immunol. 2011;186:1248-58.
5. Yin H, Wu H, Chen Y, Zhang J, Zheng M, Chen G, *et al*. The Therapeutic and Pathogenic Role of Autophagy in Autoimmune Diseases. Front Immunol. 2018;9:1512.
6. Sarafian V, Jadot M, Foidart JM, Letesson JJ, Van den Brûle F, Castronovo V, *et al*. Expression of Lamp-1 and Lamp-2 and their interactions with galectin-3 in human tumor cells. Int J Cancer. 1998;75:105-11.
7. Klapan K, Frangež Ž, Markov N, Yousefi S, Simon D, Simon H-U. Evidence for lysosomal dysfunction within the epidermis in psoriasis and atopic dermatitis. J Invest Dermatol. 2021;141:2838-48.
8. Liang N, Zhang K. The link between autophagy and psoriasis. Acta Histochemica. 2024;126:152-66.
9. Kuczyńska M, Moskot M, Gabig-Cimińska M. Insights into Autophagic Machinery and Lysosomal Function in Cells Involved in the Psoriatic Immune-Mediated Inflammatory Cascade. Arch Immunol Ther Exp. 2024;72:1-14.
10. Xue S, Lin Y, Chen H, Yang Z, Zha J, Jiang X, *et al*. Mechanisms of autophagy and their implications in dermatological disorders. Frontiers in Immunology. 2024;15:1486627.
11. Hossain MI, Marcus JM, Lee JH, Garcia PL, Singh V,

Shacka JJ, *et al*. Restoration of CTSD (cathepsin D) and lysosomal function in stroke is neuroprotective. Autophagy. 2021;17:1330-48.
12. Li N, Zhu B, Zhu Q, Heizati M, Wu T, Wang G, *et al*. Serum lysosomal-associated membrane protein-2 levels are increased in small and medium-vessel vasculitis, especially in polyarteritis nodosa. Clin Exp Rheumatol. 2019;37:S79-S85.

How to Cite This Article
Eissa SESH, Radwan NS, Atef MM, Ibrahim ZAA. Study of Serum Level of Lysosomal Associated Membrane Protein 2 in Psoriatic Patients. International Journal of Dermatology, Venereology and Leprosy Sciences. 2026; 9(1): 21-24