DOI: 10.7860/JCDR/2026/85039.22982

Original Article

Internal Medicine Section

Evaluation of Correlation between Psoriasis Severity and Proteinuria: A Cross-sectional Study

S KRISHNAPRIYA[1], DHANIN PUTHIYOTTIL[2], VP ANEEB RAJ[3], BIFI JOY[4]

ABSTRACT

Introduction: Psoriasis is a chronic inflammatory skin disease characterised by well demarcated erythematous scaly plaques. Psoriasis has been found to be associated with various systemic involvement including renal, metabolic syndrome and cardiovascular abnormalities. The association between psoriasis severity, chronicity and proteinuria is documented in literature. Understanding this relationship may help in early identification of subclinical renal involvement and guide timely intervention.

Aim: To assess the correlation between albuminuria with chronicity and severity of psoriasis.

Materials and Methods: The present cross-sectional study included 50 patients with psoriasis who fulfilled the inclusion and exclusion criteria, who were studied for a period of 12 months from April 2024 to March 2025. The study was conducted in the Department of Dermatology at the Government Medical College Kannur, Kerala, India. After obtaining consent, detailed history was taken and clinical examination was done. The severity of psoriasis was assessed by psoriasis Area Severity Index (PASI) and chronicity by the duration in days from the diagnosis. Urine Albumin Creatinine Ratio (UACR) was measured in these patients. The estimated Glomerular Filtration Rate (eGFR) was calculated in all patients using CKD-EPI equation. Descriptive statistics like frequency, percentage, mean and standard deviation were used for statistical analysis. Correlation between UACR values with PASI score were measured by Spearman rank correlation coefficient. All p-values <0.05 were considered significant.

Results: Out of the 50 patients who were enrolled in the study, 54% (n=27) were males. Duration of psoriasis ranged between 84-17885 days. The mean±SD of eGFR was found to be 87.5±22.97 in mL/min/1.73 m^2. The PASI score ranged from 1 to 12 with mean±SD of 3.9±2.28. The UACR ranged from 1.5 to 33.0 mg/g with a mean±SD of 7.76±7.33. Among the 50 patients, UACR levels very weak positive correlation with PASI which was not statistically significant (p=0.684). Among the 50 patients, UACR levels showed a very weak positive correlation with chronicity, which was not statistically significant (p=0.387).

Conclusion: The present study could find a very weak positive correlation between severity and chronicity of psoriasis and UACR levels. However, there was no statistically significant correlation between neither with chronicity nor with the severity. The take home message is at least in relatively less severe cohort of psoriasis UACR may not be a strong independent marker of renal involvement.

Keywords: Albuminuria, Psoriatic nephropathy, Psoriasis area and severity index score

INTRODUCTION

Psoriasis is a chronic inflammatory and hyperproliferative papulosquamous skin disease with approximately 60 million people affected worldwide characterised by erythematous papules and plaques with silvery white scales [1]. Histopathologically, it is characterised by parakeratosis, acanthosis with elongated rete ridges, suprapapillary thinning of epidermis, agranulosis and dilated dermal capillaries [2]. Initiation of immune process occurs with immune activation in susceptible individuals following environmental stimuli and/or loss of immune tolerance via the recognition of three psoriasis autoantigens; LL37/cathelicidin, ADAMTSL5, and PLA2G4D-generated neolipidantigens [3].

Psoriasis has been found to be associated with systemic diseases such as cardiovascular diseases (myocardial infarction, stroke), diabetes mellitus and metabolic syndrome [4]. Chronic inflammation by cell mediated immunity mediated by T cell activation and cytokines such as Tumour Necrosis Factor α (TNF α) is thought to be the pathophysiologic link between psoriasis and these systemic conditions [4]. Compared to other well-known cardiovascular and metabolic co-morbidities, the relation between psoriasis and renal disease is largely unclear and more research is required in this field [5].

Renal involvement in patients with psoriasis is increasingly being researched in the recent years. This has led to postulation of a new entity-psoriatic nephropathy [6]. Multiple studies shown greater prevalence of microalbuminuria, a sign of subclinical and early marker of glomerular dysfunction in patients with psoriasis [7-9]. Measuring 24-hour urine protein is a cumbersome procedure and could be replaced by UACR which is more sensitive than urine dipstick examination for albuminuria [10]. But potential confounders such as diabetes mellitus, systemic hypertension, and use of nephrotoxic drugs can also lead to nephropathy in psoriasis [11]. Furthermore, psoriatic arthritis and consuming Non-steroidal Anti-inflammatory Drugs (NSAIDs) also acts as a confounders increasing the risk of renal abnormality in psoriatic patients [12].Various glomerular disease like IgA nephropathy, mesangio proliferative glomerulonephritis have been histologically documented in psoriatic patients [13-16]. IgA nephropathy has been recognised as the most common glomerulonephritis in the list [13]. An immune mechanism was proposed to explain the close association between psoriasis and IgA nephropathy. High serum IgA levels were reported in up to 67% of psoriatic patients and this could reflect an abnormal immunological response, as in other autoimmune diseases, or may reflect an antibody response to a hypothetical infectious agent [17].

There is clear knowledge gap whether to screen or not for albuminuria in all patients with psoriasis as studies depicting conflicting results [5,7,18,19]. The aim of the study was to assess the correlation between albuminuria with chronicity and severity of psoriasis. The null hypothesis was that there is no correlation between albuminuria and severity and chronicity of psoriasis, and the alternate hypothesis was there is a correlation.

MATERIALS AND METHODS

The present hospital based cross sectional study was conducted at the Outpatient department of Dermatology, Venereology and Leprosy in Government Medical College Kannur, Kerala, India. Study was conducted for a period of 12 months (1 year) from April 2024 to March 2025. The study was initiated only after obtaining approval from Institutional Ethics Committee (IEC No 33/2023/GMCK).

Inclusion and Exclusion criteria: All adult patients (>18 years) with psoriasis were included in the study. Those with any other dermatological disease, pre-existing kidney disease, history of acute febrile illness within one week or urinary tract infection at the time of assessment were excluded. Patients with diabetes mellitus and systemic hypertension, those with psoriatic arthritis and patients receiving drugs which can cause nephrotoxicity like NSAIDS, methotrexate, ciclosporin were also excluded from the study.

Sample size calculation: The formula used for sample size calculation was:

$$n=\frac{Z(1-\alpha/2)+Z(1-b)}{(r^2/1-r^2)}$$

(where, n= the sample size, α - significance level, (1-β): power;

Z(1-α/2): 01.96, corresponding to 5% level of significance Z(1-b): 0.84, corresponding to 80% power. Substituting r=0.45 based on previous study by Dervisoglu E et al., the minimal sample size was found to be 12) [9].

Study Procedure

Patients with psoriasis fulfilling the inclusion criteria and consecutively attending Dermatology OPD were included in the study. Detailed history about the duration of the disease, treatment taken, age of onset, other co-morbidities, any family history acquired from the cases. Detailed dermatological and systemic examination done. All details recorded. The psoriasis Area and Severity Index (PASI) scores used to determine the severity and extent of psoriasis [20]. The PASI score accounts for both the extent of body surface area affected by the erythema, scaling, and thickness and the severity of these measures. The PASI score accounts for both the extent of body surface area affected by the erythema, scaling, and thickness and the severity of these measures. The score ranges from 0 (no disease) to 72 (maximal disease). Patients with a PASI score more than 12 are regarded as severe, score of 6 to 12 regarded as moderate and those with a PASI score less than six as mild. The PASI scoring was done by single person for all patients and reference photograph was used to reduce intra observer variability. Participants explained about the procedure of the study and written informed consent acquired in patient's regional language.

All participants have their blood drawn in red vacutainer tube for serum creatinine and serum urea estimation. All participants have midstream urine sample collected in a clean bottle and subjected to urine spot albumin, spot UACR and, for urine microscopy. Early morning sample is used to assess UACR and urine dipstick test. The blood investigations are also done at the same time. UACR was measured in an accredited clinical laboratory using an immunoturbidimetric assay on an automated analyser. The laboratory follows strict internal quality control procedures with daily calibration and participates in external proficiency testing. Jaffe reaction (spectrophotometry) was used for measuring serum creatinine. Urine dipstick >1+ and UACR value of >30 mg/g is treated as abnormal. The eGFR was calculated in all patients using CKD-EPI equation.

STATISTICAL ANALYSIS

Descriptive statistics like frequency, percentage used. Mean, standard deviation used for presenting UACR values. Median and interquartile range used for PASI score correlation. Spearman's rank correlation coefficient used for statistical analysis as both PASI score and chronicity data had significant skewing. The p-value <0.05 considered significant. Statistical analysis was done Statistical Package for Social Sciences (SPSS) 23 software.

RESULTS

The baseline characteristics are shown in [Table/Fig-1]. The UACR was expressed in mg/g. It ranged from 1.5 to 33.0 mg/g with a mean and standard deviation of 7.76 and 7.33, respectively (95% CI 5.68-9.85 mg/g). Only six patients had detectable urine albumin in dipstick (dipstick showing one +) examination. The UACR in these six patients ranged from 2.9 to 20.6mg/g with mean±SD of 8.2±6.59. The mean chronicity and mean PASI score in these six patients who had dipstick albumin positive was 1551.3 days 3.3, respectively. All patients had normal serum creatinine values with maximum being 1.3 mg/dL and mean eGFR±SD was 95.33±27.04 [Table/Fig-2]. Spearman's rank correlation coefficient showed only a very weak positive correlation (r<0.2 considered as very weak) between UACR and PASI values (r=0.059) which was not statistically significant (p=0.684) [Table/Fig-3a,b]. UACR and chronicity of psoriasis also showed a very weak positive correlation (r=0.125) which was not statistically significant (p=0.387). Scatter

Characteristics	Values
Age: n (%)	
21-40 years	17 (34)
40-60 years	19 (38)
>60 years	14 (28)
Males: n (%)	27 (54)
BMI: mean+SD (95% CI)	25.52±3.75 (24.45-26.58)
PASI score: mean+SD (95% CI)	3.9±2.28 (3.25-4.55)
Chronicity (days): mean+SD (95% CI)	1975.34±2690.5 (1210.7-2739.9)
Urine ACR (mg/g): mean+SD (95% CI)	7.76±7.33 (5.68-9.85)
Serum creatinine (mg/dL): mean+SD (95% CI)	0.96±0.21(0.95-1.05)
Serum urea (mg/dL): mean+SD (95% CI)	21.18±7.09 (19.16-23.20)
eGFR (mL/m 1.73 m^2): mean+SD (95% CI)	87.5±22.98 (80.97-94.03)
Psoriasis involvement: n (%)	
Scalp	16 (32)
Upper limb	26 (52)
Lower limb	20 (40)
Palms	19 (38)
Soles	6 (12)
Anterior trunk	22 (44)
Posterior trunk	31 (62)
Joint involvement	1 (2)
Nail changes: n (%)	
Pitting	18 (36)
Onycholysis	14 (28)
Longitudinal ridges	10 (20)
Subungual hyperkeratosis	11 (22)
Psoriasis type: n (%)	
Scalp psoriasis	8 (16)
Plaque psoriasis	28 (56)
Generalised pustular psoriasis	1 (2)
Palmoplantar psoriasis	4 (8)
Annular psoriasis	4 (8)
Guttate psoriasis	5 (10)
PASI score: n (%)	
Mild	42 (84)
Moderate	8 (16)
Severe	Nil

[Table/Fig-1]: Baseline characteristics.

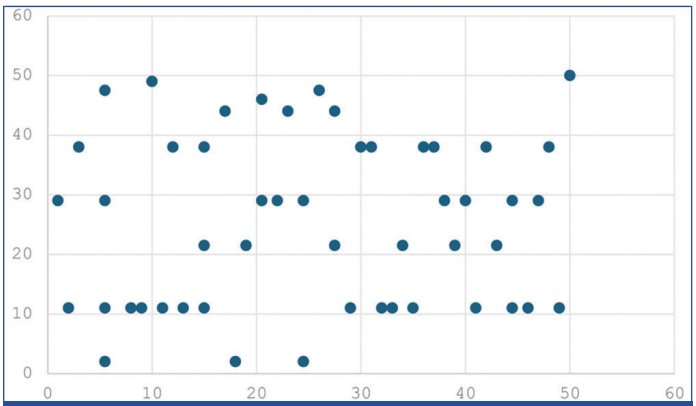
S. No.	Age	Gender	BMI	Chronicity (days)	PASI	UACR (mg/g)	Serum creatinine (mg/dL)	eGFR (mL/m 1.73m^2)
1	30	M	23.8	183	5	2.9	1.02	101
2	55	F	33.1	3285	2	20.6	1.2	71
3	35	F	30.4	730	4	3.61	1.03	97
4	41	F	23.9	1825	2	5.62	0.9	110
5	70	F	28.9	2555	2	10.3	1.3	59
6	22	F	21.5	730	5	6.3	0.7	134

[Table/Fig-2]: Characteristics of six patients who had urine dipstick albumin +.

plot showed in [Table/Fig-4a,b] demonstrates no significant positive correlation with either chronicity or PASI score with UACR. Subgroup analysis of PASI categories was not done as the number was very small.

Parameters	UACR	PASI
Spearman's rho UACR correlation coefficient	1.000	0.059
Sig. (2-tailed)		0.684
N	50	50
PASI correlation coefficient	0.059	1.000
Sig.(2-tailed)	0.684	
N	50	50

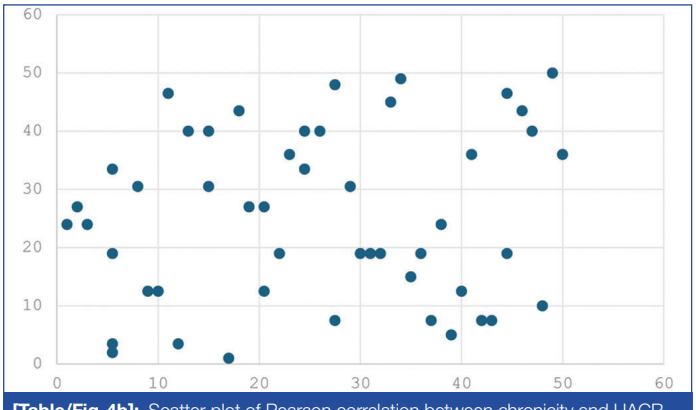
[Table/Fig-3a]: Pearson correlation between PASI score and UACR.



[Table/Fig-3b]: Scatter plot of Pearson correlation between PASI score and UACR. ranked data: X-axis-UACR, y-axis-PASI score

Parameters	UACR	Days
Spearman's rho UACR correlation coefficient	1.000	0.125
Sig. (2-tailed)		0.387
N	50	50
PASI correlation coefficient	0.125	1.000
Sig. (2-tailed)	0.387	
N	50	50

[Table/Fig-4a]: Pearson correlation between chronicity and UACR.



[Table/Fig-4b]: Scatter plot of Pearson correlation between chronicity and UACR. ranked data: X-axis-UACR, y-axis-chronicity

DISCUSSION

In recent years, there has been a growing number of reports describing the coexistence of psoriasis and renal diseases [5]. Several histopathological and immunological abnormalities have been reported in renal biopsies of patients with psoriasis. Although psoriasis can affect the kidney through multiple mechanisms, the present study aimed to evaluate the significance of proteinuria among patients with psoriasis.

The study results showed no significant proteinuria among 50 patients with mild to moderate psoriasis as assessed by the PASI score. The Urinary Albumin-To-Creatinine Ratio (UACR) among patients with albuminuria ranged from 1.5 to 33.0 mg/g, with a mean±SD of 7.76±7.33 mg/g. These values indicate that none of the patients had clinically significant albuminuria (>30 mg/g). Among the 50 patients, UACR levels showed a very weak positive correlation with PASI score and disease chronicity, but this correlation was not statistically significant. Hence, the study accepted the null hypothesis stating no correlation between PASI and chronicity with UACR.

The findings are consistent with several previous studies reporting no significant association between psoriasis and proteinuria. Kaur I et al., found that the mean urine ACR in the disease group was13.36±26 mg/g compared to 5.66 mg/g (3.40-8.08) in the control group. Although there was a trend toward higher ACR in psoriatic patients, the difference was not statistically significant [21]. Similarly, Seena P et al., reported that among 104 patients with psoriasis, seven had elevated UACR levels, but this was also not statistically significant [18]. The same study, however, noted a positive association between the risk of developing Chronic Kidney Disease (CKD) and both the duration and severity of psoriasis. Tehranchinia Z et al., found no statistically significant difference in 24-hour urinary albumin between psoriatic patients and controls, nor an increased risk of kidney disease in cases [22]. A cross-sectional study by Friedland R et al., also reported no increased risk of CKD or End-Stage Renal Disease (ESRD) among paediatric psoriasis patients compared to controls [11]. Nowowiejska J et al., showed psoriasis does not impairs glomerular or tubular function by measuring urinary albumin and tubular markers like beta-2 microglobulin [23].

Conversely, a few studies have reported differing results. Conti A et al., demonstrated a significant positive correlation between lower eGFR and both psoriasis severity and disease duration [5]. Chiu HY et al., in a cohort study, also found a positive association between psoriasis and CKD compared with controls [19]. A large population-based study from Korea showed that psoriasis was associated with an increased risk of proteinuria, particularly among patients with newly developed proteinuria, although the inclusion of patients with other confounding risk factors was not clearly stated [7]. An earlier study (1992) reported that median albuminuria was significantly higher among 32 non-diabetic, non-hypertensive psoriatic patients compared to controls, particularly in those with more extensive skin involvement (PASI >11) [8]. In a study by Dervisoglu E et al., patients with psoriasis had an increased prevalence of pathologic albuminuria (30 mg/24 h) compared with controls and had significant correlation with PASI score [9].

From a clinical perspective, these findings suggest that routine screening for proteinuria may not be necessary in all patients with mild to moderate psoriasis in the absence of traditional CKD risk factors. Instead, renal evaluation may be more appropriately targeted toward patients with severe psoriasis, longer disease duration, or those with comorbidities known to independently increase the risk of kidney disease. This tailored approach may help avoid unnecessary investigations, reduce healthcare burden, and allow clinicians to focus resources on higher-risk subsets of psoriatic patients.

However, given that psoriasis is increasingly recognised as a systemic inflammatory disease, the possibility of subclinical renal involvement cannot be completely excluded. Longitudinal studies with larger cohorts, including patients with severe psoriasis, are warranted to better delineate the temporal relationship between psoriasis severity and kidney dysfunction. Incorporating biomarkers of renal injury and evaluating microalbuminuria trends over time may further enhance understanding of true renal risk in psoriasis.

Limitation(s)

Limitations of the study were no patients with severe psoriasis (PASI >12), cross-sectional design, having no control group in the study. The PASI scoring was done by single examiner at one time and the inter and intra examiner variability testing could not be calculated. The study was at a single time point measurement unable to assess variability or progression over time. The Dipstick sensitivity is low which may miss early proteinuria and spot UACR was less reliable compared to 24-hour UACR.

CONCLUSION(S)

In this cohort of 50 patients with mild to moderate Psoriasis (as measured by PASI score), no clinically significant proteinuria or albuminuria was observed. The study could not find out a significant correlation between albuminuria and chronicity and severity of psoriasis. This underscores the need for larger longitudinal studies, ideally including patients with more severe disease or additional risk factors to better characterise whether and when kidney monitoring should be routinely recommended for psoriasis patients.

REFERENCES

[1] Raharja A, Mahil SK, Barker JN. Psoriasis: A brief overview. Clin Med (Lond). 2021;21(3):170-73. Doi: 10.7861/clinmed.2021-0257. PMID: 34001566; PMCID: PMC8140694

[2] Mihu C, Neag MA, Bocşan IC, Melincovici CS, Vesa ŞC, Ionescu C, et al. Novel concepts in psoriasis: Histopathology and markers related to modern treatment approaches. Rom J Morphol Embryol. 2021;62(4):897-906. Doi: 10.47162/RJME.62.4.02. PMID: 35673809; PMCID: PMC9289716.

[3] Hawkes JE, Yan BY, Chan TC, Krueger JG. Discovery of the IL-23/IL-17signaling pathway and the treatment of psoriasis. J Immunol. 2018;201(6):1605-13.

[4] Davidovici BB, Sattar N, Prinz JC, Puig L, Emery P, Barker JN, et al. Psoriasis and systemic inflammatory diseases: Potential mechanistic links between skin disease and co-morbid conditions. J Invest Dermatol. 2010;130(7):1785-96. Doi: 10.1038/jid.2010.103.

[5] Conti A, Giovannini L, Mandel VD, Odorici G, Lasagni C, Bigi L, et al. Chronic kidney disease in psoriasis: A cohort study. J Dtsch Dermatol Ges. 2020;18(5):438-45. Doi: 10.1111/ddg.14087. Epub 2020 Apr 20. PMID: 32311824.

[6] Grandinetti V, Baraldi O, Comai G, Corradetti V, Aiello V, Bini C, et al. Renal dysfunction in psoriatic patients. G Ital Nefrol. 2020;37(1):2020.

[7] Bae EH, Kim B, Song SH, Oh TR, Suh SH, Choi HS, et al. Proteinuria and psoriasis risk: A nationwide population-based study. J Clin Med. 2021;10(11):2356. Doi: 10.3390/jcm10112356. PMID: 34071993; PMCID: PMC8199156.

[8] Cecchi R, Seghieri G, Gironi A, Tuci F, Giomi A. Relation between urinary albumin excretion and skin involvement in patients with psoriasis. Dermatology. 1992;185(2):93-95.

[9] Dervisoglu E, Akturk AS, Yildiz K, Kiran R, Yilmaz A. The spectrum of renal abnormalities in patients with psoriasis. Int Urol Nephrol. 2012;44(2):509-14. Doi: 10.1007/s11255-011-9966-1. Epub 2011 Apr 20. PMID: 21505751.

[10] Lamb EJ, McTaggart MP, Stevens PE. Why albumin to creatinine ratio should replace protein to creatinine ratio: It is not just about nephrologists. Ann Clin Biochem. 2013;50(4):301-05.

[11] Friedland R, Kridin K, Cohen AD, Landau D, Ben-Amitai D. Psoriasis and renal disorders: A large-scale population-based study in children and adults. Dermatology. 2022;238(5):904-09.

[12] Lee E, Han JH, Bang CH, Yoo SA, Han KD, Kim H-N, et al. Risk of end-stage renal disease in psoriatic patients: Real-world data from a nationwide population-based cohort study. Sci Rep. 2019;9:16581. Doi: 10.1038/s41598-019-53017-4.

[13] Grewal SK, Wan J, Denburg MR, Shin DB, Takeshita J, Gelfand JM. The risk of IgA nephropathy and glomerular disease in patients with psoriasis: A population based cohort study. Br J Dermatol. 2017;176(5):1366-69.

[14] Jiao Y, Xu H, Li H, Li X. Mesangial proliferative glomerulonephritis with or without IgA deposits: The morphological characters in psoriasis vulgaris. Nephron Clin Pract. 2008;108:c221-c225. Doi: 10.1159/000119716. 15.

[15] Hiki Y, Kokubo T, Horii A, Yokouchi S, Satoh M, Kuwao S, et al. A case of severe IgA nephropathy associated with psoriatic arthritis and idiopathic interstitial pneumonia. Acta Pathol Jpn. 1993;43:522-28.

[16] Yamamoto M, Yorioka T, Kawada M, Nishimura K, Kumon Y, Yasuoka N, et al. A case of IgA nephropathy associated with psoriasis vulgaris. Nihon Jinzo Gakkai Shi. 1994;36:779-83.

[17] Hall RP, Peck GL, Lawley TJ. Circulating IgA immune complexes in patients with psoriasis. J Invest Dermatol. 1983;80(6):465-68.

[18] Seena P, George S, Narayanan B, Poornimamba M, Shabna CV, Gopinath A. Chronic kidney disease in patients with psoriasis–A hospital based cross sectionalstudy. Indian Dermatol Online J. 2021;12(6):864.

[19] Chiu HY, Huang HL, Li CH, Yin Y-J, Chen H-A, Hsu S-T, et al. Increased risk of glomerulonephritis and chronic kidney disease in relation to the severity of psoriasis, concomitant medication, and comorbidity: A nationwide population-based cohort study. Br J Dermatol. 2015;173(1):146-54.

[20] Manchanda Y, De A, Das S, Chakraborty D. Disease assessment in psoriasis. Indian J Dermatol. 2023;68(3):278-81. Doi: 10.4103/ijd.ijd_420_23. PMID: 37529444; PMCID: PMC10389140.

[21] Kaur I, Gandhi V, Raizada A, Bhattacharya SN, Tripathi AK, Jakhar D. Psoriaticnephropathy and its correlation with hs-CRP: A case control study. Indian Dermatol Online J. 2020;11(1):29.

[22] Tehranchinia Z, Ghanei E, Mohammadi N, Partovi-Kia M, Rahimi H, Mozafari N. No relation between psoriasis and renal abnormalities: A case-control study. Scientific World Journal. 2018;2018(3):01-05.

[23] Nowowiejska J, Baran A, Hermanowicz JM, Sieklucka B, Koper-Lenkiewicz OM, Kamińska J, et al. Psoriasis does not seem to impair glomerular and tubular function - the comprehensive study on serum and urine. Psoriasis (Auckl). 2025;15:285-99. Doi: 10.2147/PTT.S530313. PMID: 40697928; PMCID: PMC12282536.

PARTICULARS OF CONTRIBUTORS:

1. Resident, Department of Dermatology, Government Medical College, Kannur, Kerala, India.
2. Assistant Professor, Department of Nephrology, Government Medical College, Kannur, Kerala, India.
3. Associate Professor, Department of Nephrology, Government Medical College, Kozhikode, Kerala, India.
4. Professor, Department of Dermatology, Government Medical College, Kannur, Kerala, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dhanin Puthiyottil,
B1S1 Quarters Government Medical College, Pariyaram, Kannur-670503, Kerala, India.
E-mail: dhanin48@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Nov 04, 2025
- Manual Googling: Feb 05, 2026
- iThenticate Software: Feb 05, 2026 (10%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Oct 24, 2025**
Date of Peer Review: **Dec 01, 2025**
Date of Acceptance: **Feb 10, 2026**
Date of Publishing: **Apr 01, 2026**