

Clinico-Histopathological Study of Psoriasis in Correlation with Lipid Profile

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Abstract

Background: Psoriasis is a chronic immune-mediated inflammatory dermatosis increasingly recognized as a systemic disease with multiple metabolic comorbidities, including dyslipidemia. Persistent inflammation in psoriasis is believed to influence lipid metabolism, thereby increasing cardiovascular risk. However, limited data are available correlating clinical severity, histopathological changes, and lipid profile abnormalities, particularly in the Indian population. The present study aimed to evaluate the clinico-histopathological spectrum of psoriasis and its correlation with lipid profile parameters.

Methods: This hospital-based cross-sectional observational study was conducted on 156 patients with clinically diagnosed psoriasis attending a tertiary care center. Detailed clinical evaluation, including assessment of disease severity using the Psoriasis Area and Severity Index (PASI), was performed. Skin biopsies were obtained for histopathological examination and graded for severity. Fasting serum lipid profile parameters, including total cholesterol, triglycerides, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) cholesterol, were analyzed. Associations between clinical severity, histopathological grading, and lipid abnormalities were statistically evaluated.

Results: The mean age of patients was 42.8 ± 13.6 years, with a male predominance. Psoriasis vulgaris was the most common clinical type. Based on PASI scoring, 39.7% of patients had mild, 34.6% moderate, and 25.6% severe psoriasis. Histopathological examination revealed moderate to severe changes in nearly 70% of cases. Dyslipidemia was present in 61.5% of patients. A significant increase in total cholesterol, triglycerides, and LDL levels and a decrease in HDL levels were observed with increasing PASI severity ($p < 0.05$). Dyslipidemia showed a strong association with severe histopathological changes ($p < 0.001$).

Conclusion: The study demonstrates a significant correlation between clinical severity, histopathological involvement, and lipid profile abnormalities in psoriasis. These findings support the concept of psoriasis as a systemic inflammatory disease with metabolic implications and highlight the need for routine lipid screening and integrated management to reduce cardiovascular risk.

Keywords: Psoriasis; Histopathology; Dyslipidemia; PASI score; Lipid profile; Systemic inflammation.

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Introduction

Psoriasis is a chronic, immune-mediated inflammatory dermatosis characterized by erythematous, scaly plaques with a relapsing–remitting course. It affects approximately 1–3% of the global population, with variable prevalence across geographic regions and ethnic groups [1]. Although traditionally regarded as a skin-limited disorder, psoriasis is now well recognized as a systemic inflammatory disease with multisystem involvement, significantly impacting quality of life and long-term morbidity [2].

The pathogenesis of psoriasis involves a complex interplay between genetic predisposition,

environmental triggers, and immune dysregulation, particularly involving Th1 and Th17 pathways [3]. Activated T-cells, dendritic cells, and pro-inflammatory cytokines such as tumor necrosis factor- α (TNF- α), interleukin-17, and interleukin-23 lead to keratinocyte hyperproliferation and abnormal epidermal differentiation, which are reflected histopathologically as acanthosis, parakeratosis, Munro microabscesses, and elongation of rete ridges [4].

In recent years, increasing evidence has linked psoriasis with several metabolic comorbidities, including dyslipidemia, obesity, insulin resistance,

hypertension, and cardiovascular disease, collectively contributing to an increased risk of atherosclerosis and premature cardiovascular events [5]. Chronic systemic inflammation is believed to be the common pathogenic mechanism linking psoriasis and metabolic abnormalities [6]. Pro-inflammatory cytokines implicated in psoriasis have been shown to interfere with lipid metabolism by altering hepatic lipid synthesis, increasing lipolysis, and promoting oxidative modification of lipoproteins [7].

Dyslipidemia in patients with psoriasis is commonly characterized by elevated total cholesterol, low-density lipoprotein (LDL), triglycerides, and reduced high-density lipoprotein (HDL) levels [8]. These lipid abnormalities may occur independent of traditional risk factors and have been reported even in younger patients and those with mild disease, suggesting a direct association with the inflammatory burden of psoriasis. The duration and severity of psoriasis have also been variably associated with the degree of lipid derangement in different studies, though findings remain inconsistent [9].

Histopathological examination remains the gold standard for confirming the diagnosis of psoriasis, especially in atypical or early cases [10]. Certain histological features may reflect disease activity and severity, and it has been postulated that more pronounced epidermal and dermal inflammatory changes could correlate with greater systemic inflammation and metabolic disturbances, including dyslipidemia [11]. However, data correlating clinical severity, histopathological features, and lipid profile abnormalities are limited and heterogeneous, particularly in the Indian population [12]. Given the rising burden of psoriasis and its associated metabolic complications, understanding the relationship between clinical presentation, histopathological characteristics, and lipid profile alterations is of significant clinical relevance. Early identification of dyslipidemia in psoriatic patients may allow timely intervention, reduce cardiovascular risk, and support a more holistic approach to disease management. Hence, the present study aimed to evaluate the clinico-histopathological spectrum of psoriasis and its correlation with lipid profile abnormalities.

Materials and Methods

This hospital-based observational cross-sectional study was conducted in the Department of Dermatology in collaboration with the Department of Pathology and the Department of Biochemistry at a tertiary care teaching hospital in India. The study was carried out over a period of 12 months, from June 2023 to May 2024. The objective was to evaluate the clinical and histopathological features

of psoriasis and to assess their correlation with serum lipid profile abnormalities.

Study Population and Sample Size: All consecutive patients attending the dermatology outpatient department and inpatient services with a clinical diagnosis of psoriasis during the study period were screened for eligibility. A total of 156 patients fulfilling the inclusion criteria were enrolled using a convenience sampling method. The sample size was based on feasibility and patient availability during the study period, with reference to previous similar hospital-based studies reporting dyslipidemia prevalence of approximately 30–50% among patients with psoriasis [13].

Inclusion and Exclusion Criteria: Patients of either sex aged ≥ 18 years with newly diagnosed or previously diagnosed, untreated or treatment-free psoriasis (no systemic therapy for at least 4 weeks and no topical therapy for at least 2 weeks prior to enrollment) were included in the study. Patients with clinical features suggestive of psoriasis vulgaris, guttate psoriasis, erythrodermic psoriasis, or pustular psoriasis were eligible.

Patients with known disorders affecting lipid metabolism such as diabetes mellitus, hypothyroidism, nephrotic syndrome, chronic liver disease, or established cardiovascular disease were excluded. Individuals receiving lipid-lowering drugs, systemic corticosteroids, retinoids, immunosuppressive therapy, or biologics in the preceding three months were also excluded. Pregnant and lactating women and patients unwilling to give informed consent were not included in the study.

Clinical Evaluation: After obtaining written informed consent, detailed demographic data including age, sex, duration of disease, family history, and associated comorbidities were recorded. A thorough dermatological examination was performed in all patients. The diagnosis of psoriasis was made based on classical clinical features such as well-defined erythematous plaques with silvery scales and characteristic distribution.

Disease severity was assessed using the Psoriasis Area and Severity Index (PASI) score. Based on PASI scores, psoriasis was categorized as mild (PASI < 10), moderate (PASI 10–20), or severe (PASI > 20). Nail involvement, scalp involvement, and joint symptoms suggestive of psoriatic arthritis were also documented.

Histopathological Examination: A skin biopsy was performed in all patients to confirm the diagnosis and assess histopathological features. A 4-mm punch biopsy was taken from an active, untreated lesion under local anesthesia, preferably from the edge of a well-developed plaque. The biopsy specimen was fixed in 10% neutral buffered

formalin, processed routinely, and embedded in paraffin.

Sections of 4–5 μm thickness were stained with hematoxylin and eosin (H&E) and examined under light microscopy by an experienced pathologist who was blinded to the clinical and biochemical findings. Histopathological parameters evaluated included hyperkeratosis, parakeratosis, acanthosis, elongation of rete ridges, thinning of suprapapillary plates, Munro microabscesses, spongiform pustules of Kogoj, dermal inflammatory infiltrate, and dilated capillaries. Histological severity was graded as mild, moderate, or severe based on the extent and prominence of epidermal and dermal changes.

Lipid Profile Assessment: After an overnight fast of at least 10–12 hours, venous blood samples were collected under aseptic precautions. Serum was separated and analyzed on the same day for lipid parameters, including total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol, using standard enzymatic methods on an automated biochemistry analyzer.

Very-low-density lipoprotein (VLDL) cholesterol was calculated using Friedewald's formula where applicable. Dyslipidemia was defined according to standard cut-off values as per national and international guidelines, including elevated total cholesterol, LDL, triglycerides, and/or reduced HDL levels [14].

Correlation of clinical, histopathological, and biochemical parameters: Clinical severity (PASI score), duration of disease, and histopathological grading were correlated with individual lipid parameters to evaluate the relationship between cutaneous disease activity and systemic lipid abnormalities. Comparisons were also made between different clinical severity groups to assess trends in lipid profile alterations.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical

Package for the Social Sciences (SPSS) version 20.0. Continuous variables were expressed as mean \pm standard deviation, while categorical variables were presented as frequencies and percentages. The association between categorical variables was assessed using the Chi-square test or Fisher's exact test as appropriate.

Comparisons of mean lipid levels across severity groups were performed using Student's t-test or one-way analysis of variance (ANOVA). Correlation between PASI score, histopathological severity, and lipid parameters was analyzed using Pearson's or Spearman's correlation coefficient. A p-value of <0.05 was considered statistically significant.

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee prior to initiation of the study. All procedures were conducted in accordance with the Declaration of Helsinki, and informed written consent was obtained from all participants before enrollment.

Results

The study included 156 patients with psoriasis, with a mean age of 42.8 ± 13.6 years. The majority of patients belonged to the 31–45 year age group (37.2%), followed by 46–60 years (28.2%). Males constituted 65.4% of the study population, showing a male preponderance. The mean duration of disease was 6.2 ± 4.9 years, with nearly one-fourth (21.8%) having psoriasis for more than 10 years. A positive family history was noted in 17.9% of patients. Clinically, psoriasis vulgaris was the most common type (75.6%), while nail involvement was observed in 29.5% of cases. Assessment of disease severity using the Psoriasis Area and Severity Index (PASI) revealed that 39.7% of patients had mild psoriasis, 34.6% had moderate disease, and 25.6% had severe psoriasis. The overall mean PASI score was 14.6 ± 6.8 , indicating that a substantial proportion of patients had moderate to severe disease at presentation (Table 1).

Table 1: Baseline demographic and clinical characteristics of psoriasis patients (n = 156)

Variable	Frequency (%) / Mean \pm SD
Age (years)	42.8 \pm 13.6
Age group (years)	
18–30	32 (20.5%)
31–45	58 (37.2%)
46–60	44 (28.2%)
>60	22 (14.1%)
Gender	
Male	102 (65.4%)
Female	54 (34.6%)
Duration of psoriasis (years)	6.2 \pm 4.9
<5 years	68 (43.6%)
5–10 years	54 (34.6%)
>10 years	34 (21.8%)

Family history of psoriasis	28 (17.9%)
Clinical type of psoriasis	
Psoriasis vulgaris	118 (75.6%)
Guttate psoriasis	18 (11.5%)
Erythrodermic psoriasis	12 (7.7%)
Pustular psoriasis	8 (5.1%)
Nail involvement	46 (29.5%)
PASI severity category	
Mild (PASI <10)	62 (39.7%)
Moderate (PASI 10–20)	54 (34.6%)
Severe (PASI >20)	40 (25.6%)
Mean PASI score	14.6 ± 6.8

Histopathological examination demonstrated classical features of psoriasis in most cases. Hyperkeratosis (94.9%), acanthosis (91.0%), and elongation of rete ridges (88.5%) were the most frequently observed findings. Parakeratosis was present in 87.2% of cases, while Munro microabscesses were identified in 61.5%. Dermal lymphocytic infiltrate was observed in 83.3%, and

dilated dermal capillaries in 66.7%, supporting active inflammatory pathology. Based on composite epidermal and dermal changes, histopathological severity was graded as mild in 30.8%, moderate in 39.7%, and severe in 29.5% of patients. Thus, nearly two-thirds of cases demonstrated moderate to severe histopathological involvement, reflecting significant disease activity (Table 2).

Table 2: Histopathological features and Histopathological severity grading observed in psoriasis patients (n = 156)

Variable	Frequency (%)
Histopathological feature	
Hyperkeratosis	148 (94.9%)
Parakeratosis	136 (87.2%)
Acanthosis	142 (91.0%)
Elongation of rete ridges	138 (88.5%)
Thinning of suprapapillary plates	122 (78.2%)
Munro microabscesses	96 (61.5%)
Spongiform pustules of Kogoj	58 (37.2%)
Dilated dermal capillaries	104 (66.7%)
Dermal lymphocytic infiltrate	130 (83.3%)
Histopathological severity	
Mild	48 (30.8%)
Moderate	62 (39.7%)
Severe	46 (29.5%)

Severity grading based on extent of epidermal hyperplasia, inflammatory infiltrate, and presence of characteristic features.

The mean total cholesterol level was 201.6 ± 38.4 mg/dL, while mean triglyceride and LDL cholesterol levels were 168.9 ± 52.7 mg/dL and 129.4 ± 34.6 mg/dL, respectively. Reduced HDL cholesterol levels were noted with a mean of 38.6 ± 8.2 mg/dL. Overall, 61.5% of patients had at least one abnormal lipid parameter, indicating a high prevalence of dyslipidemia among psoriasis patients (Table 3).

Table 3: Fasting lipid profile parameters in psoriasis patients (n = 156).

Lipid parameter	Mean ± SD	Frequency (%)
		Abnormal
Total cholesterol (mg/ dL)	201.6 ± 38.4	72 (46.2%)
Triglycerides (mg/ dL)	168.9 ± 52.7	78 (50.0%)
LDL cholesterol (mg/ dL)	129.4 ± 34.6	74 (47.4%)
HDL cholesterol (mg/ dL)	38.6 ± 8.2	82 (52.6%)
VLDL cholesterol (mg/ dL)	33.8 ± 10.5	76 (48.7%)
Any dyslipidemia	—	96 (61.5%)

Dyslipidemia defined using standard guideline cut-offs. LDL: Low-density lipoprotein; HDL: High-density lipoprotein; VLDL: Very-low-density lipoprotein; SD: Standard deviation.

A statistically significant worsening of lipid parameters was observed with increasing PASI severity. Mean total cholesterol, triglycerides, and LDL cholesterol levels progressively increased from mild to severe psoriasis ($p = 0.001$, $p < 0.001$, and $p = 0.003$, respectively).

Conversely, HDL cholesterol levels showed a significant decreasing trend with increasing disease severity ($p < 0.001$). These findings indicate a strong association between clinical severity of psoriasis and dyslipidemia (Table 4).

Table 4: Comparison of lipid profile parameters across PASI severity categories

Lipid parameter	Mild (n = 62)	Moderate (n = 54)	Severe (n = 40)	p-value
	Mean \pm SD			
Total cholesterol (mg/ dL)	186.4 \pm 32.1	204.8 \pm 35.6	223.6 \pm 41.2	0.001
Triglycerides (mg/ dL)	148.2 \pm 41.6	170.5 \pm 48.2	197.8 \pm 56.4	<0.001
LDL cholesterol (mg/ dL)	118.6 \pm 29.4	131.9 \pm 32.8	145.2 \pm 38.6	0.003
HDL cholesterol (mg/ dL)	41.8 \pm 7.6	38.2 \pm 7.9	34.1 \pm 6.8	<0.001

PASI: Psoriasis Area and Severity Index.

Dyslipidemia was present in 37.5% of patients with mild histopathological changes, 61.3% with moderate changes, and 87.0% with severe histopathological involvement. This association was statistically highly significant ($p < 0.001$), demonstrating a strong correlation between increasing histopathological severity of psoriasis and the presence of lipid abnormalities (Table 5).

Table 6: Association between histopathological severity and dyslipidemia (n = 156)

Histopathological severity	Dyslipidemia present	Dyslipidemia Absent	p-value
	Frequency (%)		
Mild (n = 48)	18 (37.5%)	30 (62.5%)	<0.001
Moderate (n = 62)	38 (61.3%)	24 (38.7%)	
Severe (n = 46)	40 (87.0%)	6 (13.0%)	

Discussion

The present clinico-histopathological study of psoriasis in correlation with lipid profile highlights the close interplay between cutaneous disease activity, histopathological severity, and systemic metabolic abnormalities. Psoriasis in our cohort predominantly affected middle-aged adults with a male preponderance, a finding consistent with several Indian hospital-based studies by Asokan et al., and Raghuvver et al., that have reported higher male attendance, possibly reflecting healthcare-seeking behavior rather than true sex predilection [15,16]. The predominance of psoriasis vulgaris and the substantial proportion of patients with long-standing disease further underscore the chronic and progressive nature of psoriasis in real-world clinical settings [17].

Clinically, nearly 60% of patients had moderate to severe disease based on PASI scores, indicating that a large proportion presented with significant disease burden. This is comparable to observations by Arora et al. and Gosai et al., who reported moderate-to-severe psoriasis in approximately 45–65% of tertiary-care attendees in India [18,19]. Higher PASI scores reflect greater inflammatory load, which is increasingly recognized as a systemic rather than skin-restricted phenomenon [20].

Histopathological evaluation in the present study demonstrated classical psoriatic features such as hyperkeratosis, parakeratosis, acanthosis, elongation of rete ridges, and Munro microabscesses

in the majority of cases. Importantly, nearly 70% of patients exhibited moderate to severe histopathological involvement, paralleling clinical severity. Similar histological patterns have been described in prior studies by Kiran et al., and Girisha et al., supporting the reliability of histopathological grading as a marker of disease activity [21,22]. The presence of dense dermal inflammatory infiltrate and dilated capillaries reflects persistent cytokine-mediated inflammation and angiogenesis, both of which are central to psoriasis pathogenesis [23].

A key finding of this study is the high prevalence of dyslipidemia (61.5%) among psoriasis patients. Elevated total cholesterol, triglycerides, LDL cholesterol, and reduced HDL cholesterol were commonly observed. This prevalence aligns with Indian studies by Kumari et al., Karne et al., and Anupama et al., reporting dyslipidemia rates ranging from 40% to 70% in psoriasis [24,25,26]. Chronic inflammation in psoriasis is known to alter lipid metabolism through cytokines such as TNF- α and interleukin-6, which increase hepatic lipid synthesis, impair reverse cholesterol transport, and promote oxidative modification of LDL particles. These mechanisms collectively contribute to an atherogenic lipid profile [27].

The most clinically relevant observation was the significant association between PASI severity and lipid abnormalities. As disease severity increased, total cholesterol, triglycerides, and LDL cholesterol levels rose significantly, while HDL cholesterol levels declined. Similar severity-dependent lipid

derangements have been reported by Kumari et al., and Karne et al., who demonstrated positive correlations between PASI scores and atherogenic lipid fractions [24,25]. These findings suggest that worsening cutaneous inflammation is accompanied by escalating systemic metabolic risk, reinforcing the concept of psoriasis as a systemic inflammatory disorder [27].

Furthermore, the study demonstrated a strong correlation between histopathological severity and dyslipidemia, with nearly 87% of patients with severe histological changes exhibiting lipid abnormalities. This observation is particularly important, as it suggests that microscopic inflammatory burden mirrors systemic metabolic derangement, even beyond what is apparent clinically [28]. While limited studies have explored this triad of clinical severity, histopathology, and lipid profile together, similar trends have been hinted at in smaller cohorts [29,30]. Histopathological severity likely reflects cumulative inflammatory exposure, which may exert sustained effects on lipid metabolism over time [30].

Taken together, the findings of this study support the growing body of evidence that psoriasis is a multisystem inflammatory disease with significant metabolic implications [31]. The parallel increase in PASI score, histopathological severity, and dyslipidemia underscores the need for a holistic approach to psoriasis management [32]. Routine screening for lipid abnormalities, especially in patients with moderate-to-severe disease or marked histopathological changes, may help in early identification of cardiovascular risk and timely intervention [32].

These findings underscore the importance of routine lipid profile screening in patients with psoriasis, particularly those with moderate to severe disease or pronounced histopathological involvement. Early identification and management of dyslipidemia may help reduce long-term cardiovascular risk in this population [33]. Dermatologists should adopt a multidisciplinary approach, collaborating with physicians and cardiologists to ensure comprehensive care. Incorporating metabolic risk assessment into routine psoriasis management may improve overall patient outcomes and reduce systemic complications associated with chronic inflammation [34].

Limitations

This study has certain limitations that should be considered while interpreting the findings. First, the cross-sectional design limits the ability to establish a temporal or causal relationship between psoriasis severity and dyslipidemia. Second, the study was conducted at a single tertiary care center, which may introduce referral bias and limit the generalizability of results to the broader community. Third, a control

group of non-psoriatic individuals was not included, restricting direct comparison of lipid profile abnormalities with the general population. Fourth, potential confounding factors such as dietary habits, physical activity, socioeconomic status, and subclinical metabolic disorders were not evaluated in detail. Additionally, inflammatory markers and cytokine levels were not assessed, which could have further strengthened the mechanistic link between cutaneous inflammation and lipid derangements. Finally, the effect of long-term treatment and its impact on lipid profile could not be evaluated due to the study design.

Conclusion

The present study demonstrates a significant association between the clinical and histopathological severity of psoriasis and abnormalities in lipid profile, reinforcing the concept of psoriasis as a systemic inflammatory disorder rather than a purely cutaneous disease. The progressive worsening of atherogenic lipid parameters with increasing PASI scores and histopathological severity highlights the close link between disease activity and metabolic dysfunction.

References

1. Raharja A, Mahil SK, Barker JN. Psoriasis: a brief overview. *Clin Med (Lond)*. 2021;21(3):170-173.
2. Campanati A, Marani A, Martina E, Diotallevi F, Radi G, Offidani A. Psoriasis as an Immune-Mediated and Inflammatory Systemic Disease: From Pathophysiology to Novel Therapeutic Approaches. *Biomedicines*. 2021;9(11):1511.
3. Zeichner JA, Armstrong A. The Role of IL-17 in the Pathogenesis and Treatment of Psoriasis. *J Clin Aesthet Dermatol*. 2016;9(6 Suppl 1):S3-S6.
4. Harden JL, Krueger JG, Bowcock AM. The immunogenetics of Psoriasis: A comprehensive review. *J Autoimmun*. 2015;64:66-73.
5. Toussiroit E, Gallais-Sérézal I, Aubin F. The cardiometabolic conditions of psoriatic disease. *Front Immunol*. 2022;13:970371.
6. Bellinato F, Maurelli M, Geat D, Girolomoni G, Gisondi P. Managing the Patient with Psoriasis and Metabolic Comorbidities. *Am J Clin Dermatol*. 2024;25(4):527-540.
7. Nowowiejska J, Baran A, Flisiak I. Aberrations in Lipid Expression and Metabolism in Psoriasis. *Int J Mol Sci*. 2021;22(12):6561.
8. Sorokin AV, Remaley AT, Mehta NN. Oxidized Lipids and Lipoprotein Dysfunction in Psoriasis. *J Psoriasis Psoriatic Arthritis*. 2020;5(4):139-146.
9. Pietrzak A, Michalak-Stoma A, Chodorowska G, Szepietowski JC. Lipid disturbances in psoriasis: an update. *Mediators Inflamm*. 2010;2010:535612.

10. Rohl R, Bax D, Schierer S, Bogner PN, Hernandez-Ilizaliturri F, Paragh G. A case for histologic verification of the diagnosis of atypical psoriasis before systemic therapy. *JAAD Case Rep.* 2018;4(5):465-467.
11. Bai S, Srinivasan S. Histopathologic diagnostic parameters of psoriasis; a clinicopathological study. *Int J Res Med Sci.* 2017;4(6):1915-1920.
12. Chandra KS, Bansal M, Nair T, et al. Consensus statement on management of dyslipidemia in Indian subjects. *Indian Heart J.* 2014;66 Suppl 3(Suppl 3):S1-51.
13. Lakshmi S, Nath AK, Udayashankar C. Metabolic syndrome in patients with psoriasis: A comparative study. *Indian Dermatol Online J.* 2014;5(2):132-137.
14. Sawhney JP, Ramakrishnan S, Madan K, et al. CSI clinical practice guidelines for dyslipidemia management: Executive summary. *Indian Heart J.* 2024;76 Suppl 1(Suppl 1):S6-S19.
15. Asokan N, Prathap P, Rejani P. Severity of Psoriasis Among Adult Males is Associated with Smoking, Not with Alcohol Use. *Indian J Dermatol.* 2014;59(3):237-240.
16. Raghuvver C, Shivanand DR, Rajashekar N. Clinico-histopathological Study of Psoriasis. *Int J Sci Stud.* 2015;3(7):176-179.
17. Ponikowska M, Vellone E, Czaplá M, Uchmanowicz I. Challenges Psoriasis and Its Impact on Quality of Life: Challenges in Treatment and Management. *Psoriasis (Auckl).* 2025;15:175-183.
18. Arora K, Hazarika N, Kumari R, Chawla H. Quality of Life in Psoriasis: A Cross-Sectional Study from North India. *Indian J Dermatol.* 2024;69(1):38-43.
19. Gosai A, Sao R, Mahaseth BK. A Study of Dyslipidemia in Psoriasis Patients: A Case-Control Study. *J Cardiovascular Disease Res.* 2024;15(5):1234-1242.
20. Man AM, Orăsan MS, Hoteiuc OA, Olănescu-Vaida-Voevod MC, Mocan T. Inflammation and Psoriasis: A Comprehensive Review. *Int J Mol Sci.* 2023;24(22):16095.
21. Kiran A, Babar Z, Naheed A, Mirza U. Frequency of Dyslipidemia in Patients with Psoriasis and Association with Disease Severity. *Pakistan J Med Health Sciences.* 2021;15(12):3430-3432.
22. Girisha BS, Thomas N, Rao GS. Metabolic Syndrome in Psoriasis among Urban South Indians: A Case Control Study Using SAM-NCEP Criteria. *J Clinical Diagnostic Res.* 2017;11(2):WC01-WC04.
23. Samotij D, Nedoszytko B, Bartosińska J, et al. Pathogenesis of psoriasis in the "omic" era. Part I. Epidemiology, clinical manifestation, immunological and neuroendocrine disturbances. *Postepy Dermatol Alergol.* 2020;37(2):135-153.
24. Kumari KMK. Psoriasis and significance of clinicopathological correlation in a tertiary care hospital. *IP Arch Cytol Histopathol Res.* 2017;2(2):23-26.
25. Karne PP, Pawar SM, Bargale AB. Study of lipid profile in psoriasis patients. *Int J Clin Biochem Res.* 2015;2(1):1-4.
26. Anupama YG, Patil SB. A retrospective clinicohistopathological study of psoriasis. *IP Indian J Clin Exp Dermatol.* 2020;6(3):222-226.
27. Sahoo SR, Das K, Panda B, et al. Serum Interleukin (IL)-6, Lipid Profile, and Association With Disease Severity in Patients With Psoriasis: A Cross-Sectional Study. *Cureus.* 2024;16(9):e69599.
28. Shah S, Parikh K, Shah ML, Kothari A. Unveiling the Metabolic Burden: A Clinicopathological Study of Chronic Plaque Psoriasis and Its Association With Metabolic Syndrome. *Cureus.* 2025;17(8):e89289.
29. Babu AR, S. A, R. RR, C. S, Shankar V, Mohamed M. A study on metabolic syndrome in patients with Psoriasis. *IP Indian J Clin Exp Dermatol.* 2017;3(2):69-74.
30. Mathew C, Sridharan R, Sudhamani B. A study of the association of psoriasis with metabolic syndrome from a tertiary care centre in Kerala, South India. *IP Indian J Clin Exp Dermatol.* 2021;7(1):18-23.
31. Gokani R, Nayak M, Janki Desai J, Patani S. Study of lipid profile in psoriasis. *Int J Acad Med Pharm* 2025;7(2):356-359.
32. Ganjre S. A study of Clinico-histopathological study of psoriasis in correlation with lipid profile. *J Cardiovascular Disease Res.* 2024;15(10):19-26.
33. Gupta A, Paneri S, Lohokare R, Sarkar PD, Jain A. Clinico-biochemical correlation between psoriasis and lipid profile. *Int J Res Med Sci.* 2016;4:1966-1969.
34. Nazir S, Haleem MA. A Study of Clinico Histopathological Correlation of Psoriasis. *Sch J App Med Sci.* 2019;7(11):3760-3765.