Psoriasis and its association with serum lipid profile: A hospital-based case-control study

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ABSTRACT

Introduction: Psoriasis is a chronic immune-mediated inflammatory dermatosis associated with significant cardiovascular comorbidity. Population-specific data on lipid abnormalities in Nepalese psoriasis patients are limited. This study aimed to determine the association of psoriasis with serum lipid profiles. **Methods:** This hospital-based case-control study enrolled 73 confirmed psoriasis cases and 73 age and sex-matched healthy controls. Fasting serum triglycerides, total cholesterol, low-density lipoprotein cholesterol, very low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol were measured. Disease severity was graded by the Psoriasis Area and Severity Index. **Results:** Psoriasis vulgaris was the commonest clinical pattern 40 (54.8%). Cases had significantly elevated median triglycerides (145.0 vs. 122.0 mg/dl; p<0.001) and very low-density lipoprotein cholesterol (37.0 vs. 21.0 mg/dl; p<0.001), and significantly lower high-density lipoprotein cholesterol (60.40 vs. 72.73 mg/dl; p<0.001) and low-density lipoprotein cholesterol (p<0.001). Total cholesterol did not differ significantly (p=0.703). Elevated very low-density lipoprotein cholesterol (odds ratio [OR] 9.06; p<0.001), low high-density lipoprotein cholesterol (OR 6.30; p=0.017), and hypertriglyceridemia (OR 3.03; p=0.003) were more prevalent among cases. No significant correlation was found between Psoriasis Area Severity Index and lipid parameters. **Conclusions:** Psoriasis is associated with a proatherogenic lipid profile. Routine fasting lipid profiling at diagnosis is recommended for early cardiovascular risk assessment.

Keywords: Cardiovascular risk, dyslipidemia, high-density lipoprotein, psoriasis, triglycerides, very low-density lipoprotein.

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INTRODUCTION

Psoriasis is a chronic, relapsing, immune-mediated inflammatory disorder of the skin and joints with a global prevalence of approximately 2–3%, including around 3% in Nepal.[1] It is characterized by keratinocyte hyperproliferation, angiogenesis, and a dysregulated T-helper (Th)1/Th17 immune response that drives persistent systemic inflammation.[2] Once regarded as purely a cutaneous disease, psoriasis is now firmly recognized as a systemic inflammatory condition associated with major comorbidities including cardiovascular disease (CVD), metabolic syndrome, hypertension, obesity, and type 2 diabetes mellitus.[3]

The cardiovascular burden in psoriasis is well established. Patients with severe psoriasis are up to 50% more likely to develop CVD than the general population, with risk escalating proportionally with disease severity.[4] This is mechanistically driven by chronic overproduction of pro-inflammatory cytokines—principally tumor necrosis factor-alpha (TNF-α), interleukin (IL)-17, IL-23, and IL-6—which promote endothelial dysfunction, oxidized low-density lipoprotein (LDL) formation, foam cell accumulation, and atherosclerotic plaque progression.[5] Persistently elevated TNF-α and IL-6 suppress lipoprotein lipase activity, impairing triglyceride

(TG) clearance and reducing high-density lipoprotein (HDL) synthesis, while simultaneously driving hepatic overproduction of very low-density lipoprotein (VLDL)—an atherogenic triad that amplifies cardiovascular risk independently of traditional risk factors.[6] Elevated plasma TG has also been shown to causally increase psoriasis susceptibility in a Mendelian randomization framework, suggesting a bidirectional pathogenic relationship.[7]

Despite this compelling evidence, population-specific data from South Asia—where distinct ethnic lipid profiles, dietary patterns, and a high baseline prevalence of atherogenic dyslipidemia further compound cardiovascular risk—remain limited. This hospital-based case-control study was therefore conducted to evaluate the fasting serum lipid profile in Nepalese psoriasis patients, compare it with age- and sex-matched healthy controls, and examine its association with disease severity as assessed by the Psoriasis Area and Severity Index (PASI).

METHODS

This hospital-based case-control study was conducted at the Department of Dermatology of Nepalgunj Medical College and Teaching Hospital, Kohalpur, from July 20, 2025 to May 20, 2026. The study was approved by the Institutional Review Committee of Nepalgunj Medical College and Teaching Hospital (Ref. No. 77 / 082-083) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Sampling technique and study participants

Consecutive (non-probability) sampling was employed; all eligible patients attending the outpatient department of Dermatology during the study period who met the inclusion criteria were enrolled as cases, and age- and sex-matched healthy controls were recruited from attendants and outpatients of other departments. The sample size was calculated using the Kelsey formula[8] for unmatched case-control studies: $n = (Z\alpha/2 + Z\beta)^2 \times [p_1(1-p_1) + p_2(1-p_2)] / (p_1-p_2)^2$, where p_2 denotes the prevalence of dyslipidemia in controls (estimated at 30% from published literature), p_1 the expected prevalence in psoriasis cases (estimated at 55%), $Z\alpha/2 = 1.96$ (5% significance level, two-tailed), and $Z\beta = 0.84$ (80% power). This yielded a minimum sample size of 66 per group; accounting for a 10% non-response rate, 73 participants were enrolled in each case and control group, giving a total of 146 participants.

Inclusion and exclusion criteria

Cases included patients aged ≥15 years with clinically confirmed psoriasis who had not received systemic therapy (methotrexate, cyclosporine, acitretin, or biologics) within the preceding three months. Excluded from both groups were patients with known diabetes mellitus, hypertension, hypothyroidism, hepatic disease, chronic kidney disease, or pregnancy, and those on lipid-modifying agents, oral contraceptives, or corticosteroids, as these conditions independently alter lipid metabolism.

Clinical assessment and PASI scoring

A structured proforma recorded age, sex, family history, and clinical pattern of psoriasis. Disease severity was graded by the Psoriasis Area and Severity Index (PASI): mild (<5), moderate (5–10), and severe (>10).[9]

Biochemical analysis

After 12 hours of fasting, venous blood was collected in plain vacutainer tubes and serum separated by centrifugation. Serum total cholesterol (TC) and TG were estimated by enzymatic colorimetric methods; HDL cholesterol (HDL-C) by the direct method. LDL cholesterol (LDL-C) and VLDL cholesterol (VLDL-C) were calculated by the Friedewald formula (LDL-C = TC − HDL-C − TG/5) for samples with TG <400 mg/dl. Reference cut-offs applied: hypertriglyceridemia (TG ≥150 mg/dl), hypercholesterolemia (TC ≥200 mg/dl), elevated LDL-C (≥130 mg/dl), low HDL-C (<40 mg/dl), and elevated VLDL-C (>30 mg/dl) per National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) criteria.[10]

Statistical analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY). Normality was assessed by the Shapiro-Wilk test. Non-normally distributed continuous variables (TG, TC, LDL-C, VLDL-C) were compared by the Mann-Whitney U test; HDL-C (normally distributed) by independent samples t-test. Fisher's exact test was applied for categorical comparisons where expected cell counts were <5 (HDL-C and LDL-C rows); Yates'-corrected chi-square for remaining categorical comparisons. Odds ratios (OR) with 95% confidence intervals (CI) were calculated by Woolf's method. Spearman's rank correlation assessed PASI–lipid associations. $P<0.05$ was considered statistically significant.

RESULTS

Demographic and clinical characteristics

Seventy-three cases (36 males, 37 females) and 73 controls

(42 males, 31 females) were enrolled. Sex distribution was comparable between groups (chi-square = 0.991; p = 0.320). Mean age of cases was 41.82 ± 13.32 years (range 16–74) vs. 48.56 ± 16.54 years in controls (p = 0.018). A positive family history of psoriasis was noted in eight (10.96%) cases. Demographic details are shown in Table 1. Clinical patterns of psoriasis are shown in Figure 1. Psoriasis vulgaris was the commonest pattern (54.8%; n = 40), followed by palmoplantar psoriasis (24.7%; n = 18). The remaining six variants each accounted for ≤4.1%. Of 71 cases with recorded PASI, 43 (60.6%) had severe disease (PASI >10), 26 (36.6%) moderate (PASI 5–10), and two (2.8%) mild (PASI <5).

Table 1: Demographic characteristics of cases and controls

Parameter	Cases (n=73)	Controls (n=73)	p-value
Mean age ± SD (years)	41.82 ± 13.32	48.56 ± 16.54	0.018*
Age range (years)	16–74	22–89	—
Male, n (%)	36 (49.3%)	42 (57.5%)	0.320**
Female, n (%)	37 (50.7%)	31 (42.5%)	
Positive family history, n (%)	8 (10.96%)	—	—

*Mann-Whitney U test. **Chi-square test. SD: standard deviation.

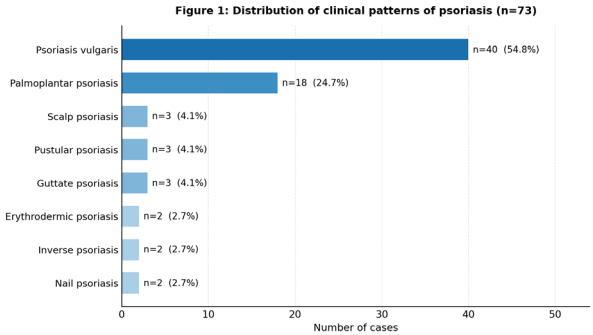

Figure 1: Distribution of clinical patterns of psoriasis in cases (n=73). Bars represent number of cases; percentages refer to proportion of total cases

Comparison of serum lipid parameters

TG, TC, LDL-C, and VLDL-C were non-normally distributed (Shapiro-Wilk p<0.05) and compared by the Mann-Whitney U test. HDL-C was normally distributed and compared by independent samples t-test. Results are summarized in Table 2.

Cases had significantly elevated median TG [145.0 (IQR 117.0–198.0) vs. 122.0 (82.0–146.0) mg/dl; U = 3766.5, p<0.001] and median VLDL-C [37.0 (26.0–56.4) vs. 21.0 (17.0–25.0) mg/dl; U = 4640.5, p<0.001]. Median LDL-C was significantly lower in cases [60.2 (44.0–84.0) vs. 90.0 (76.0–125.0) mg/dl; U = 1210.0, p<0.001]. Mean HDL-C was significantly lower in cases (60.40 ± 17.96 vs. 72.73 ± 19.31 mg/dl; t = −3.99, p < 0.001). TC did not differ significantly [142.0 vs. 135.0 mg/dl; U = 2762.5, p = 0.703].

Table 2: Comparison of serum lipid parameters between cases and controls

Parameter (mg/dl)	Cases Median (IQR) / Mean ± SD	Controls Median (IQR) / Mean ± SD	Test statistic	p-value
TG[1]	145.0 (117.0–198.0)	122.0 (82.0–146.0)	U=3766.5	<0.001*
TC[1]	142.0 (111.0–200.0)	135.0 (121.0–190.0)	U=2762.5	0.703
LDL-C[1]	60.2 (44.0–84.0)	90.0 (76.0–125.0)	U=1210.0	<0.001*
VLDL-C[1]	37.0 (26.0–56.4)	21.0 (17.0–25.0)	U=4640.5	<0.001*
HDL-C[2]	60.40 ± 17.96	72.73 ± 19.31	t=−3.99	<0.001*

[1]Median (IQR); Mann-Whitney U test (non-normally distributed). [2] Mean ± SD; independent samples t-test (normally distributed). *Statistically significant (p<0.05). IQR: interquartile range; SD: standard deviation; TG: triglycerides; TC: total cholesterol; LDL-C: low-density lipoprotein cholesterol; VLDL-C: very low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

Prevalence of dyslipidemia parameters

Prevalence of individual dyslipidemia components with OR and 95% CI is shown in Table 3 and Figure 2. Elevated VLDL-C (>30 mg/dl) was the most prevalent abnormality (61.6% vs. 15.1%; OR 9.06; 95% CI 4.09–20.08; p<0.001). Low HDL-C (<40 mg/dl) was present in 15.1% of cases vs. 2.7% of controls (OR 6.30; 95% CI 1.34–29.52; p = 0.017). Hypertriglyceridemia (TG ≥150 mg/dl) occurred in 47.9% of cases vs. 23.3% of controls (OR 3.03; 95% CI 1.49–6.18; p = 0.003). Elevated LDL-C (≥130 mg/dl) was paradoxically less frequent in cases (6.8% vs. 24.7%; OR 0.22; 95% CI 0.08–0.64; p = 0.006). Hypercholesterolemia (TC ≥200 mg/dl) did not differ significantly (p = 0.557).

Table 3: Prevalence of dyslipidaemia parameters and odds ratios in psoriasis cases versus controls

Parameter	Cases n(%)	Controls n(%)	OR	95% CI	p-value
Hypertriglyceridaemia (TG ≥150 mg/dl)	35(47.9%)	17(23.3%)	3.03	1.49–6.18	0.003*a
Hypercholesterolaemia (TC ≥200 mg/dl)	19(26.0%)	15(20.5%)	1.36	0.63–2.94	0.557a
Elevated LDL-C (≥130 mg/dl)	5(6.8%)	18(24.7%)	0.22	0.08–0.64	0.006*b
Low HDL-C (<40 mg/dl)	11(15.1%)	2 (2.7%)	6.30	1.34–29.52	0.017*b
Elevated VLDL-C (>30 mg/dl)	45(61.6%)	11(15.1%)	9.06	4.09–20.08	<0.001*a

*Statistically significant (p<0.05). OR: odds ratio (Woolf's method); CI: 95% confidence interval. a Yates' chi-square with continuity correction. b Fisher's exact test.

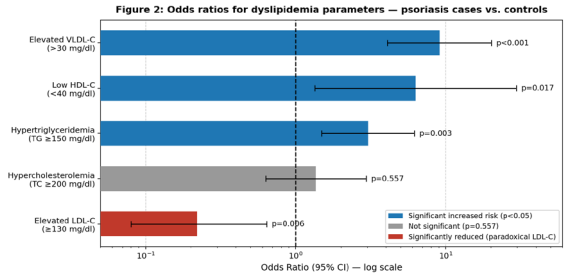

Figure 2: Odds ratios (95% CI) for dyslipidemia parameters in psoriasis cases vs. controls (log scale). Blue bars: significant increased risk; grey bar: non-significant; red bar: paradoxically reduced odds. The dashed reference line

indicates OR = 1.0

Correlation between PASI and lipid parameters

Spearman's rank correlation between PASI scores and lipid parameters was assessed in 71 cases with available PASI data. No statistically significant correlation was identified: TG (ρ = 0.074; p = 0.537), TC (ρ = −0.002; p = 0.990), LDL-C (ρ = 0.157; p = 0.192), VLDL-C (ρ = −0.027; p = 0.825), or HDL-C (ρ = 0.042; p = 0.730).

DISCUSSION

This hospital-based case-control study from Nepal demonstrated that psoriasis is associated with a proatherogenic lipid phenotype of elevated TG and VLDL-C, and reduced HDL-C, corroborating and extending the growing South Asian evidence base on psoriasis and lipid dysregulation.

The significantly elevated median TG in cases (145.0 vs. 122.0 mg/dl; p<0.001; OR 3.03; 95% CI 1.49–6.18) aligns with the atherogenic dyslipidemia pattern—high TG, low HDL, and small dense LDL—predominant in South Asian populations per the ICMR-INDIAB survey.[11] Kafle et al., in the only prior Nepalese case-control study from Patan Hospital, reported more than 95% dyslipidemia prevalence in 30 psoriasis patients with TG significantly elevated (p = 0.001).[3] Our study, enrolling 73 cases, confirms this with greater statistical power. Basnet et al. from Western Nepal similarly reported significantly higher dyslipidemia in 52 psoriasis patients vs. controls (OR 1.709; p<0.001).[12] In India, Nakhwa et al. from JSS Medical College, Mysore, found mean TG of 219.69 mg/dl in cases and VLDL-C of 42.99 mg/dl vs. 28.0 mg/dl in controls (p = 0.001), concordant with our data.[13] Mechanistically, TNF-α and IL-6 suppress lipoprotein lipase and impair TG clearance while driving hepatic VLDL overproduction.[6]

The most striking finding was markedly elevated VLDL-C (median 37.0 vs. 21.0 mg/dl; OR 9.06; 95% CI 4.09–20.08; p<0.001), the largest effect size in this study. VLDL-C is the direct precursor of atherogenic small dense LDL particles and its elevation reflects hepatic inflammatory overproduction and impaired catabolism. Praneeth et al. from Tirupati, Andhra Pradesh, reported VLDL-C of 49.6 mg/dl vs. 22.9 mg/dl (p<0.001) with an Atherogenic Index of Plasma (AIP) of 0.74 vs. 0.29.[14] Rajendran et al. from Sri Ramachandra Institute, Chennai, documented dyslipidemia prevalence of 74.3% vs. 41.4% in controls, with TG and VLDL-C as predominant contributors.[2]

The significantly lower mean HDL-C in cases (60.40 vs. 72.73 mg/dl; t = −3.99, p < 0.001; OR 6.30; 95% CI 1.34–29.52 for HDL-C <40 mg/dl) is clinically important given HDL's central role in reverse cholesterol transport and vascular protection. Kafle et al. identified HDL suppression as the most consistent lipid abnormality in their Nepalese cohort (p = 0.012).[3] Asha et al. from New Delhi documented significantly lower HDL and elevated oxidized LDL in 150 psoriasis patients, concluding that qualitative HDL dysfunction not merely quantitative reduction drives cardiovascular risk.[15] Sahoo et al. from VIMSAR, Burla, confirmed reduced HDL-C alongside elevated IL-6.[16] Kuberan et al. from Chettinad Hospital, Chennai, reported 35% of young adult psoriasis patients had low HDL-C by NCEP-ATP III criteria.[17]

A notable paradox was the significantly lower LDL-C in cases (median 60.2 vs. 90.0 mg/dl; p<0.001; OR 0.22; 95% CI 0.08–0.64). This psoriatic lipid paradox is attributed to IL-6-mediated upregulation of hepatic LDL receptors, accelerating LDL clearance despite an overall proatherogenic state. Nakhwa et al. similarly found no significant LDL elevation in their Indian cohort.[13] Wang et al. confirmed in a meta-analysis that apolipoprotein B is significantly elevated in psoriasis despite normal LDL-C, highlighting the inadequacy of standard lipid panels alone.[18]

TC did not differ significantly (p = 0.703), consistent with Kafle et al.[3] This is explained by the counterbalancing effect of elevated VLDL-C against simultaneously reduced LDL-C. No significant PASI–lipid correlation was identified (all p>0.05), concordant with Kafle et al., Basnet et al., Nakhwa et al., and Sahoo et al.[3,12,13,16] This likely reflects the multifactorial determinants of lipid dysregulation, disease duration, prior systemic therapy, and baseline metabolic risk that a single PASI measurement cannot capture.

Given that psoriasis affects approximately 3% of Nepal's population[1] yet cardiovascular risk assessment is not routinely integrated into dermatological practice, the markedly elevated ORs for VLDL-C (OR 9.06) and low HDL-C (OR 6.30) strongly support routine fasting lipid profiling for all newly diagnosed psoriasis patients, particularly those with moderate-to-severe disease. Actionable dyslipidemia findings should prompt dietary counselling, physical activity, and where clinically appropriate statin therapy, which carries additional anti-inflammatory benefits.[19]

Limitations include the cross-sectional design precluding causal inference, absence of body mass index and metabolic syndrome data, use of the Friedewald formula at high

TG values, non-measurement of advanced lipid markers (apolipoprotein B, oxidized LDL, paraoxonase-1), and incomplete disease duration records. Future prospective Nepalese studies with larger samples and comprehensive metabolic assessment are warranted.

CONCLUSIONS

Psoriasis is associated with a proatherogenic lipid profile of significantly elevated TG (OR 3.03; 95% CI 1.49–6.18) and VLDL-C (OR 9.06; 95% CI 4.09–20.08), and reduced HDL-C (OR 6.30; 95% CI 1.34–29.52). TC did not differ significantly and LDL-C was paradoxically lower in cases. No significant PASI–lipid correlation was found. Routine fasting lipid profiling at the time of psoriasis diagnosis is strongly recommended to enable early cardiovascular risk detection and timely preventive intervention in Nepalese psoriatic patients.

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CONFLICTS OF INTEREST: None declared

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AUTHORS' CONTRIBUTIONS

KP: concept, design, statistical analysis and interpretation; KJS: data collection and clinical assessment; SP: supervised manuscript drafting; all authors critically revised and approved the final version for submission.

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