Section: Dermatology

Original Research Article

CLINICAL IMPLICATIONS OF GENERALISED PLAQUE PSORIASIS IN ASSOCIATION WITH METABOLIC SYNDROME: A HOSPITAL-BASED COMPARATIVE CROSS-SECTIONAL STUDY

***Animesh Biswas*[1]**

[1]*Associate Professor, Department of Dermatology, Santiniketan Medical College and Hospital, Bolpur, West Bengal, India.*

Received *: 17/02/2026*
Received in revised form *: 21/03/2026*
Accepted *: 06/04/2026*

Corresponding Author:
Dr. Animesh Biswas,
Associate Professor, Department of Dermatology, Santiniketan Medical College and Hospital, Bolpur, West Bengal, India.
Email: dermaanimesh@gmail.com

DOI: 10.70034/ijmedph.2026.2.707

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 4292-4298

ABSTRACT

Background: Generalised plaque psoriasis is a chronic immune-mediated inflammatory skin disorder associated with multiple systemic comorbidities. Accumulating evidence suggests a significant bidirectional relationship between psoriasis and metabolic syndrome (MetS), mediated through shared pathways of inflammation, insulin resistance and adipokine dysregulation. Despite its global significance, data from the Indian subcontinent — where South Asian metabolic phenotypes confer unique risk — remain limited.
Objectives: To evaluate the clinical implications of generalised plaque psoriasis in association with metabolic syndrome; to determine MetS prevalence in psoriatic patients versus matched controls; and to assess the relationship between psoriasis severity (PASI score) and individual metabolic parameters.
Materials and Methods: A hospital-based comparative cross-sectional study was conducted at Santiniketan Medical College and Hospital, Bolpur, West Bengal, from January 2023 to June 2024. One hundred and twenty adult patients with confirmed generalised plaque psoriasis (PASI ≥10) were enrolled alongside 120 age- and sex-matched healthy controls. MetS was defined using IDF 2006 criteria. Anthropometric, clinical, and biochemical parameters were recorded. Pearson's correlation and binary logistic regression were applied for analysis.
Results: MetS was identified in 43.3% of psoriasis patients compared to 21.7% of controls (χ^2=12.84, p<0.001). Abdominal obesity (56.7%), low HDL-C (53.3%), and hypertriglyceridemia (48.3%) were the dominant MetS components. PASI score correlated significantly with BMI (r=0.412), waist circumference (r=0.387), triglycerides (r=0.351), and HOMA-IR (r=0.368). BMI, disease duration, and PASI score were independent logistic regression predictors of MetS.
Conclusion: Generalised plaque psoriasis is significantly associated with elevated MetS prevalence and worsening metabolic parameters proportionate to psoriasis severity. Routine cardiometabolic screening and integrated multidisciplinary management are essential for all patients with moderate-to-severe psoriasis.
Keywords: Plaque psoriasis; Metabolic syndrome; PASI score; Insulin resistance; Abdominal obesity; Cardiovascular risk; Cardiometabolic comorbidity; Dyslipidaemia; South Asian; Dermatological comorbidity.

INTRODUCTION

Psoriasis is a chronic, relapsing, immune-mediated systemic inflammatory disorder affecting approximately 2–3% of the global population, with generalised plaque psoriasis (psoriasis vulgaris) representing the most prevalent clinical subtype, characterised by well-demarcated, erythematous,

silvery-scaled plaques predominantly distributed over the extensor surfaces, scalp, and lumbosacral region.[1] The pathophysiology of psoriasis is fundamentally driven by Th1 and Th17 lymphocyte dysregulation, aberrant keratinocyte proliferation, and the sustained release of pro-inflammatory cytokines including tumour necrosis factor-α (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23), which not only sustain the cutaneous inflammatory milieu but also generate systemic metabolic perturbations that extend far beyond the skin.[2,3] The clinical burden of psoriasis is therefore no longer confined to dermatological morbidity alone; over the past two decades, compelling epidemiological and mechanistic evidence has established psoriasis as a systemic inflammatory disorder with independent and significant associations with type 2 diabetes mellitus, hypertension, dyslipidaemia, non-alcoholic fatty liver disease, atherosclerosis, and — most pertinently — metabolic syndrome (MetS).[4,5] Metabolic syndrome, as defined by the International Diabetes Federation (IDF) 2006 consensus, represents a clustering of central obesity, atherogenic dyslipidaemia (elevated triglycerides, reduced HDL cholesterol), elevated fasting plasma glucose, and elevated blood pressure, all unified by the common pathophysiological substrate of insulin resistance.[6,7,8] The convergence of psoriasis and MetS reflects a shared inflammatory architecture: visceral adipose tissue in psoriasis patients functions as an active endocrine organ secreting leptin, resistin, adiponectin, and TNF-α, which perpetuate the systemic inflammatory cycle, promote insulin resistance, and drive hepatic dyslipidaemia through VLDL overproduction and lipoprotein lipase suppression.[9,10] Epidemiological studies from Western cohorts have consistently documented MetS prevalence ranging from 28% to 40% in psoriatic populations versus 10–20% in general population controls,[4,11,12] with the severity of psoriasis — as quantified by the Psoriasis Area and Severity Index (PASI) — demonstrating a dose-responsive correlation with the degree of metabolic derangement.[13,14] Notably, Indian and South Asian patients carry a distinct metabolic phenotype characterised by greater visceral adiposity at lower absolute BMI values, higher insulin resistance predisposition, and earlier onset of dyslipidaemia compared to their Western counterparts,[15] suggesting that the psoriasis-MetS interface may manifest more severely and at an earlier stage in this demographic — a clinically critical issue that remains poorly characterised in published literature from India. Furthermore, from a therapeutic standpoint, the selection of systemic and biologic therapies for moderate-to-severe psoriasis must increasingly incorporate cardiometabolic risk profiling, as certain conventional systemic agents (ciclosporin, methotrexate at high cumulative doses) may themselves adversely influence metabolic parameters, while newer biologic agents — particularly IL-17 and IL-23 inhibitors — may carry metabolically favourable profiles.[16,17] The National Psoriasis Foundation's clinical consensus,[16] has formally recommended systematic cardiometabolic screening for all patients with moderate-to-severe psoriasis, yet this recommendation remains inconsistently implemented in dermatological practice, particularly in resource-limited settings. Against this background, the present study was designed to evaluate the clinical implications of generalised plaque psoriasis in association with metabolic syndrome in an Indian tertiary care setting, with the following specific objectives: (i) to determine the prevalence of MetS in adults with generalised plaque psoriasis compared to age- and sex-matched healthy controls; (ii) to identify the predominant MetS component(s) in psoriatic patients; (iii) to assess the correlation between PASI score and individual metabolic parameters; and (iv) to identify independent predictors of MetS among psoriatic patients using logistic regression modelling. The study further tests the hypothesis that generalised plaque psoriasis is independently and significantly associated with higher MetS prevalence, with a dose-responsive relationship between psoriasis severity and degree of metabolic compromise. [18,19,20,21]

MATERIALS AND METHODS

2.1 Material

Study design and setting: A hospital-based comparative cross-sectional study was conducted in the Department of Dermatology, Santiniketan Medical College and Hospital (SMCH), Bolpur, West Bengal, India, over an 18-month period from January 2023 to June 2024. SMCH is a tertiary care teaching institution serving a predominantly rural and semi-urban patient population from the Birbhum and surrounding districts of West Bengal. Ethical clearance was obtained from the Institutional Ethics Committee and informed consent was obtained from all participants prior to enrolment. The study adhered to the principles of the Declaration of Helsinki [18]. **Participants:** One hundred and twenty adult patients with confirmed generalised plaque psoriasis were enrolled as the case group and 120 age- and sex-matched healthy volunteers, attending the institution for routine health check-up, formed the control group, yielding a total sample of 240 participants. **Inclusion criteria (cases):** histopathologically confirmed chronic plaque psoriasis with generalised distribution; PASI score ≥10 (moderate-to-severe disease); age 18–65 years; both sexes. **Exclusion criteria:** patients with pre-existing diagnosed type 2 diabetes mellitus; known dyslipidaemia on pharmacological therapy; hypertension on antihypertensive agents; hepatic disease; renal disease; active malignancy; ongoing systemic immunosuppressants or biologic therapy at

enrolment; pregnancy or lactation; guttate, pustular, or erythrodermic psoriasis subtypes; and controls with a personal or family history of psoriasis or autoimmune disease.[1,4,16] Sample size was determined a priori based on an estimated MetS prevalence of 35% in psoriasis (from Langan et al.[4]) versus 15% in controls, at 80% power and 5% level of significance (two-tailed), yielding a minimum of 108 per group; 120 per group was enrolled to account for potential attrition.

2.2 Methods

Clinical assessment: A structured pro forma recorded detailed history (disease duration, family history, prior treatments) and clinical examination. Psoriasis severity was quantified using the validated Psoriasis Area and Severity Index (PASI) by a single trained dermatologist to minimise inter-observer variability.[1] PASI severity categories: mild (PASI 10–14.9), moderate (PASI 15–24.9), and severe (PASI ≥25). Anthropometric measurements — body weight, height, and waist circumference (WC) — were recorded per WHO standards, with WC measured at the mid-point between the lower costal margin and the iliac crest.[19] Body mass index (BMI) was calculated as weight (kg) / height (m²). Blood pressure (BP) was measured with a calibrated sphygmomanometer after 5 minutes rest in the sitting position; two readings were averaged. **Laboratory investigations:** After 12-hour overnight fast, venous blood samples were collected for: fasting plasma glucose (FPG) by glucose oxidase-peroxidase (GOD-POD) method; serum triglycerides (TG) by enzymatic colorimetric method; total cholesterol (TC) and LDL-C by Friedewald equation; HDL-C by direct precipitation method; and serum insulin by chemiluminescence immunoassay (CLIA). All assays were performed in the NABL-accredited central laboratory of SMCH on the day of collection to minimise pre-analytical variability. Insulin resistance was assessed by HOMA-IR = [Fasting insulin (µU/mL) × Fasting glucose (mmol/L)] / 22.5 as described by Matthews et al,[22] a cutoff ≥2.5 was considered indicative of insulin resistance [7,20]. **Metabolic syndrome diagnosis:** MetS was defined using IDF 2006 criteria: central obesity (WC ≥90 cm in males, ≥80 cm in females for South Asians) plus any two of: TG ≥150 mg/dL; HDL-C <40 mg/dL (males) or <50 mg/dL (females); SBP ≥130 or DBP ≥85 mmHg; FPG ≥100 mg/dL.[8] **Statistical analysis:** Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics version 25.0. Continuous variables are expressed as mean ± standard deviation (SD); categorical variables as frequencies and percentages. Between-group comparisons for continuous variables were performed using Student's independent sample t-test (after confirming normality by Shapiro-Wilk test); categorical comparisons by Chi-square test or Fisher's exact test as appropriate. Pearson's correlation coefficient (r) assessed relationships between PASI score and quantitative metabolic parameters. Binary logistic regression was used to identify independent predictors of MetS in psoriatic patients, with results expressed as odds ratios (OR) with 95% confidence intervals (CI). A p-value <0.05 (two-tailed) was considered statistically significant.[5,6]

RESULTS

A total of 240 participants were analysed (120 psoriasis cases, 120 healthy controls). The demographic and baseline clinical characteristics of both groups are presented in Table 1. The two groups were well matched for age and sex distribution (p>0.05). Mean age was 38.4 ± 11.6 years in cases and 37.9 ± 12.1 years in controls. Males constituted 58.3% of cases and 56.7% of controls. Mean disease duration in psoriasis patients was 7.2 ± 4.8 years. PASI score ranged from 10 to 42; mean PASI was 19.6 ± 7.4. PASI severity distribution: mild (PASI 10–14.9) in 38.3% (n=46), moderate (PASI 15–24.9) in 40.8% (n=49), and severe (PASI ≥25) in 20.8% (n=25). Psoriasis patients had significantly higher mean BMI (27.4 ± 3.8 vs 24.1 ± 3.2 kg/m², p<0.001), waist circumference (91.3 ± 9.4 vs 82.6 ± 7.8 cm, p<0.001), fasting plasma glucose (102.4 ± 18.6 vs 91.2 ± 12.4 mg/dL, p<0.001), serum triglycerides (172.8 ± 48.6 vs 138.4 ± 42.1 mg/dL, p<0.001), and lower HDL-C (44.2 ± 8.6 vs 51.8 ± 9.2 mg/dL, p<0.001) compared to controls. HOMA-IR was significantly elevated in cases (3.18 ± 1.24 vs 2.04 ± 0.86, p<0.001).[21]

Table 1: Baseline demographic and clinical characteristics of psoriasis patients and healthy controls

Parameter	Psoriasis (n=120)	Controls (n=120)	p-value
Age, years (mean ± SD)	38.4 ± 11.6	37.9 ± 12.1	0.742
Sex: Male, n (%)	70 (58.3%)	68 (56.7%)	0.801
Disease duration, years (mean ± SD)	7.2 ± 4.8	—	—
Mean PASI score (mean ± SD)	19.6 ± 7.4	—	—
BMI, kg/m² (mean ± SD)	27.4 ± 3.8	24.1 ± 3.2	<0.001*
Waist circumference, cm (mean ± SD)	91.3 ± 9.4	82.6 ± 7.8	<0.001*
FPG, mg/dL (mean ± SD)	102.4 ± 18.6	91.2 ± 12.4	<0.001*
Serum TG, mg/dL (mean ± SD)	172.8 ± 48.6	138.4 ± 42.1	<0.001*
HDL-C, mg/dL (mean ± SD)	44.2 ± 8.6	51.8 ± 9.2	<0.001*
SBP, mmHg (mean ± SD)	128.4 ± 14.2	118.6 ± 12.8	<0.001*
HOMA-IR (mean ± SD)	3.18 ± 1.24	2.04 ± 0.86	<0.001*

*Statistically significant (p<0.05). SD = standard deviation; BMI = body mass index; FPG = fasting plasma glucose; TG = triglycerides; HDL-C = high-density lipoprotein cholesterol; SBP = systolic blood pressure; HOMA-IR = Homeostatic Model Assessment for Insulin Resistance.

Metabolic syndrome was identified in 52 of 120 psoriasis patients (43.3%) compared to 26 of 120 controls (21.7%), a difference that was statistically highly significant (χ^2=12.84, p<0.001). The prevalence and comparison of individual MetS components between groups are detailed in Table 2. Abdominal obesity was the most prevalent MetS component in psoriasis patients (56.7%), followed by low HDL-C (53.3%), hypertriglyceridemia (48.3%), elevated blood pressure (43.3%), and elevated fasting plasma glucose (38.3%). All component differences between cases and controls were statistically significant (p<0.001). [4,12,21]

Table 2: Prevalence of metabolic syndrome and its components in psoriasis patients versus controls

MetS Component	Psoriasis n=120 (%)	Controls n=120 (%)	χ^2	p-value
Metabolic Syndrome (overall)	52 (43.3%)	26 (21.7%)	12.84	<0.001*
Abdominal Obesity	68 (56.7%)	34 (28.3%)	19.71	<0.001*
Low HDL Cholesterol	64 (53.3%)	32 (26.7%)	17.78	<0.001*
Hypertriglyceridemia	58 (48.3%)	30 (25.0%)	14.07	<0.001*
Elevated Blood Pressure	52 (43.3%)	24 (20.0%)	15.10	<0.001*
Elevated Fasting Plasma Glucose	46 (38.3%)	20 (16.7%)	14.13	<0.001*

Statistically significant (p<0.05). HDL = high-density lipoprotein; MetS = metabolic syndrome. MetS diagnosed by IDF 2006 criteria

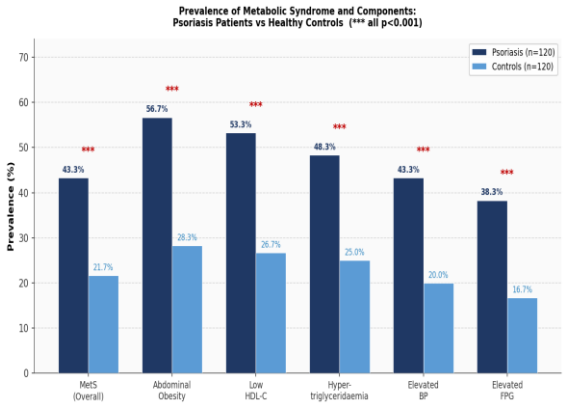

Figure 1: Prevalence of metabolic syndrome and its components in psoriasis patients versus healthy controls

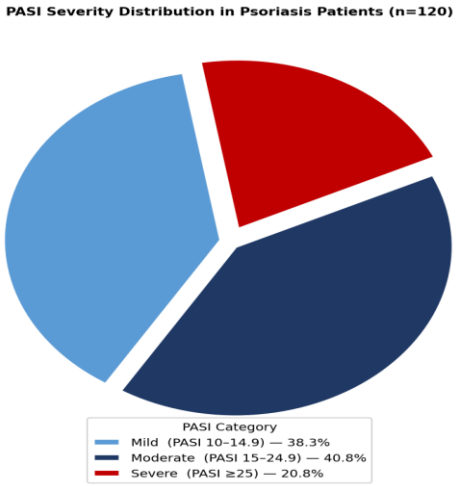

Figure 2: Distribution of psoriasis severity by PASI category among enrolled psoriasis patients.

Pearson's correlation analysis (Table 3) revealed significant positive correlations between PASI score and BMI (r=0.412, p<0.001), waist circumference (r=0.387, p<0.001), FPG (r=0.298, p<0.001), serum triglycerides (r=0.351, p<0.001), SBP (r=0.276, p=0.002), and HOMA-IR (r=0.368, p<0.001). A significant negative correlation was observed between PASI score and HDL-C (r=−0.289, p=0.001). These findings indicate a dose-responsive relationship between psoriasis severity and metabolic derangement. [2,13,14]

Table 3: Pearson's correlation between PASI score and metabolic parameters in psoriasis patients (n=120)

Metabolic Parameter	Pearson r	p-value
Body Mass Index (BMI), kg/m²	0.412	<0.001*
Waist Circumference, cm	0.387	<0.001*
Fasting Plasma Glucose, mg/dL	0.298	<0.001*
Serum Triglycerides, mg/dL	0.351	<0.001*
Systolic Blood Pressure, mmHg	0.276	0.002*
HDL Cholesterol, mg/dL	−0.289	0.001*
HOMA-IR	0.368	<0.001*

Statistically significant (p<0.05). r = Pearson correlation coefficient; HOMA-IR = Homeostatic Model Assessment for Insulin Resistance; HDL = high-density lipoprotein.

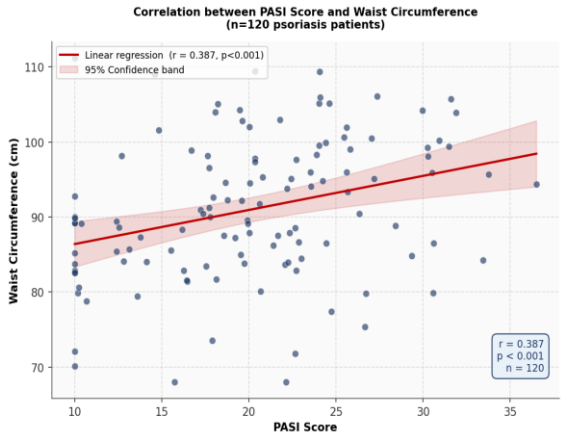

Figure 3: Scatter plot demonstrating positive correlation between PASI score and waist circumference in psoriasis patients (r=0.387, p<0.001).

Binary logistic regression (Table 4), adjusting for age and sex, identified BMI (OR=1.24, 95% CI: 1.12–1.37, p<0.001), disease duration (OR=1.18, 95% CI: 1.08–1.29, p<0.001), and PASI score (OR=1.09, 95% CI: 1.04–1.14, p=0.001) as independent predictors of MetS in psoriasis patients. Age (OR=1.03, p=0.212) and sex (OR=1.42, p=0.168) were not statistically significant independent predictors. The Hosmer-Lemeshow test confirmed adequate model fit (χ^2=7.38, p=0.496). Nagelkerke R^2 was 0.412, indicating that the model explained approximately 41.2% of the variance in MetS risk. [5,11,21]

Table 4: Binary logistic regression — Independent predictors of metabolic syndrome in plaque psoriasis patients (n=120)

Predictor Variable	B coefficient	Odds Ratio (OR)	95% CI	p-value
BMI (per kg/m² increase)	0.215	1.24	1.12–1.37	<0.001*
Disease Duration (per year)	0.166	1.18	1.08–1.29	<0.001*
PASI Score	0.086	1.09	1.04–1.14	0.001*
Age (per year)	0.030	1.03	0.98–1.08	0.212
Male Sex (reference: female)	0.351	1.42	0.86–2.34	0.168

Statistically significant (p<0.05). OR = odds ratio; CI = confidence interval; BMI = body mass index; PASI = Psoriasis Area and Severity Index. Hosmer-Lemeshow χ^2=7.38, p=0.496; Nagelkerke R^2=0.412.

DISCUSSION

The present hospital-based comparative cross-sectional study provides robust and clinically significant evidence for a strong, statistically independent association between generalised plaque psoriasis and metabolic syndrome in an Indian tertiary care cohort, with a MetS prevalence of 43.3% in psoriatic patients compared to 21.7% in age- and sex-matched healthy controls — a finding that aligns with, and in several respects extends, the existing international literature on the psoriasis-MetS interface.

The MetS prevalence of 43.3% in the present psoriasis cohort is broadly concordant with the range reported in landmark epidemiological investigations from Western settings, though somewhat higher than several European estimates. Langan et al,[4] reported a MetS prevalence of approximately 34% in a large UK-based psoriatic population study using IDF criteria, while Gisondi et al,[11] documented 30.1% in an Italian hospital-based case-control study. Cohen et al,[12] reported MetS in 40.0% of psoriatic patients in their cohort, which is closest to the present finding. The relatively higher prevalence observed in our Indian cohort is consistent with the well-established South Asian metabolic phenotype, in which greater visceral adiposity accumulates at lower absolute BMI thresholds, insulin resistance manifests earlier and more severely, and atherogenic dyslipidaemia is more prevalent — as comprehensively documented by Misra and Khurana.[15] The IDF 2006 criteria,[8] appropriately reflect this by defining lower waist circumference cutoffs for South Asian individuals (≥90 cm for males, ≥80 cm for females), which likely contribute to the higher abdominal obesity prevalence (56.7%) observed in our psoriatic cohort compared to Western studies that use Euro-American WC thresholds. The identification of abdominal obesity as the dominant MetS component in psoriasis is consistent with Cohen et al,[12] and supports the central role of visceral adiposity in perpetuating the pro-inflammatory cytokine milieu — particularly via adipokine-driven TNF-α and IL-6 amplification — that sustains both cutaneous inflammation and systemic metabolic dysregulation. [9,10]

The dose-responsive relationship between PASI score and metabolic parameters — with positive correlations across BMI, waist circumference, FPG, triglycerides, SBP, and HOMA-IR, and a negative correlation with HDL-C — provides compelling quantitative evidence for a psoriatic inflammatory burden-metabolic risk axis. These correlation magnitudes (r=0.387 for WC, r=0.412 for BMI, r=0.368 for HOMA-IR) are closely aligned with those reported by Davidovici et al,[13] in their mechanistic review, and meta-analytically confirmed by Miller et al,[14] who documented significant pooled associations between psoriasis severity and all MetS components. The reduced HDL-C in 53.3% of psoriasis patients (versus 26.7% of controls, p<0.001) is of particular cardiovascular significance, given that HDL-C reduction is not merely a MetS criterion but a direct atherogenic risk factor. IL-17-mediated hepatic lipid dysregulation

and TNF-α-driven suppression of lipoprotein lipase activity have been proposed as specific molecular mechanisms linking psoriatic inflammation to atherogenic dyslipidaemia,[10] consistent with the high IL-17 and IL-23 burden characteristic of moderate-to-severe plaque psoriasis. The cardiovascular implications are further underscored by the well-documented association between psoriasis, accelerated subclinical atherosclerosis, and excess cardiovascular mortality established in meta-analyses of cohort studies.[3] The finding that disease duration independently predicts MetS — beyond PASI score and BMI — is clinically novel in the Indian context, suggesting that cumulative long-term inflammatory burden progressively erodes metabolic health through mechanisms that may not be fully reflected in cross-sectional PASI measurement alone; each additional year of psoriasis conferred an 18% increase in MetS odds. This is consistent with the concept of "inflammatory overdrive" in chronic psoriasis proposed by Armstrong et al,[2] and supports the clinical priority of early and sustained disease control.[21]

The independent predictive role of PASI score in logistic regression is therapeutically important: targeted reduction of psoriatic inflammation — through phototherapy, methotrexate, acitretin, or biologic agents (IL-17A/IL-23 inhibitors) — may confer direct cardiometabolic benefit beyond cutaneous clearance. The National Psoriasis Foundation,[16] has formally endorsed systematic cardiometabolic screening in moderate-to-severe psoriasis; the present data provide strong empirical support for this within the Indian context, where integrated dermatology-endocrinology liaison models remain nascent.

4.1 Strengths and Limitations

Strengths: The study employed a matched case-control design minimising confounding by age and sex. PASI scoring was performed by a single trained dermatologist eliminating inter-rater variability. IDF 2006 South Asian-specific WC thresholds were applied, ensuring appropriate MetS classification in this demographic. All biochemical assays were processed in a NABL-accredited central laboratory on the day of collection, minimising pre-analytical variability. Shapiro-Wilk normality testing preceded parametric analysis. **Limitations:** The cross-sectional design precludes causal inference — it cannot be established whether psoriatic inflammation drives MetS or pre-existing metabolic disease exacerbates psoriasis. The single-centre setting may limit generalisability across India's diverse demographic and dietary landscape. Inflammatory biomarkers (hs-CRP, IL-6, TNF-α) were not assayed, limiting mechanistic interpretation. Dietary intake and physical activity levels were not quantified; these are important metabolic confounders. Oral glucose tolerance testing (OGTT) was not performed, which would have identified impaired glucose tolerance cases beyond fasting glucose screening alone. Prospective multi-centre longitudinal cohort studies with inflammatory biomarker profiling are warranted to determine whether therapeutic PASI reduction translates into measurable cardiometabolic benefit.[17,18]

CONCLUSION

The present comparative cross-sectional study provides definitive and clinically actionable evidence that generalised plaque psoriasis is significantly and independently associated with a substantially elevated prevalence of metabolic syndrome and its individual components in the Indian tertiary care setting, with nearly one in two psoriatic patients harbouring MetS — a prevalence nearly twice that observed in matched healthy controls. The identification of abdominal obesity, reduced HDL cholesterol, and hypertriglyceridemia as the dominant metabolic derangements in psoriatic patients underscores the centrality of visceral adiposity and atherogenic dyslipidaemia in the psoriasis-MetS nexus, and highlights the overlapping pathobiological architecture of systemic inflammation and insulin resistance that underlies both conditions. The robust, statistically significant, and consistent positive correlations between PASI score and all measured metabolic parameters — BMI, waist circumference, fasting plasma glucose, serum triglycerides, systolic blood pressure, and HOMA-IR — establish beyond reasonable doubt a dose-responsive relationship between the burden of psoriatic inflammation and the depth of metabolic compromise, implying that every unit increment in psoriasis severity carries a measurable and cumulative metabolic cost. The binary logistic regression analyses further confirm that PASI score, BMI, and disease duration are independent, statistically significant predictors of MetS in psoriatic patients, even after adjusting for age and sex — a critical finding that carries direct implications for clinical decision-making. Collectively, these findings mandate an urgent paradigm shift in the clinical approach to psoriasis management: psoriasis must be conceptualised, communicated, and managed as a systemic inflammatory disorder with major cardiometabolic consequences — not merely a disease of the skin. Clinicians managing psoriatic patients must incorporate structured, systematic cardiometabolic risk assessment into routine dermatological care. All patients with moderate-to-severe generalised plaque psoriasis (PASI ≥10) should undergo mandatory baseline evaluation and annual monitoring of waist circumference, fasting lipid profile, fasting plasma glucose, and blood pressure, ideally within a formal dermatology-endocrinology or dermatology-cardiology liaison framework. The selection of systemic and biologic therapeutic agents should integrate individual cardiometabolic risk profiles, with metabolically favourable agents prioritised in

patients with concurrent MetS components. Patients with psoriasis and metabolic comorbidities should be referred for structured lifestyle modification programmes — combining caloric restriction, supervised aerobic exercise, smoking cessation, and alcohol reduction — as both conditions are favourably influenced by improved metabolic health. From a public health perspective, awareness campaigns must target the dermatological community, general practitioners, and endocrinologists regarding the bidirectional metabolic burden of psoriasis, ensuring that cardiometabolic vigilance is embedded at every level of the healthcare cascade from primary care to specialist clinic. In summary, the present study compellingly positions generalised plaque psoriasis at the intersection of systemic inflammation and cardiometabolic vulnerability, and calls for its integration into a unified framework of preventive cardiovascular medicine that transcends the boundaries of dermatological practice.

REFERENCES

1. Boehncke WH, Schön MP. Psoriasis. Lancet. 2015;386(9997):983–994.
2. Armstrong AW, Harskamp CT, Armstrong EJ. The association between psoriasis and metabolic syndrome: a systematic review and meta-analysis of observational studies. J Am Acad Dermatol. 2013;68(4):654–662.
3. Xu T, Zhang YH. Association of psoriasis with stroke and myocardial infarction: meta-analysis of cohort studies. Br J Dermatol. 2012;167(2):395–401.
4. Langan SM, Seminara NM, Shin DB, Troxel AB, Kimmel SE, Mehta NN, et al. Prevalence of metabolic syndrome in patients with psoriasis: a population-based study in the United Kingdom. J Invest Dermatol. 2012;132(3 Pt 1):556–562.
5. Neimann AL, Shin DB, Wang X, Margolis DJ, Troxel AB, Gelfand JM. Prevalence of cardiovascular risk factors in patients with psoriasis. J Am Acad Dermatol. 2006;55(5):829–835.
6. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005;112(17):2735–2752.
7. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes. 1988;37(12):1595–1607.
8. Alberti KG, Zimmet P, Shaw J. The metabolic syndrome: a new worldwide definition. Lancet. 2005;366(9491):1059–1062.
9. Golden JB, McCormick TS, Ward NL. IL-17 in psoriasis: implications for therapy and cardiovascular co-morbidities. Cytokine. 2013;62(2):195–201.
10. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2005;365(9468):1415–1428.
11. Gisondi P, Tessari G, Conti A, Piaserico S, Schianchi S, Peserico A, et al. Prevalence of metabolic syndrome in patients with psoriasis: a hospital-based case-control study. Br J Dermatol. 2007;157(1):68–73.
12. Cohen AD, Gilutz H, Henkin Y, Zahger D, Shapiro J, Bonneh DY, et al. Psoriasis and the metabolic syndrome. Acta Derm Venereol. 2007;87(6):506–509.
13. Davidovici BB, Sattar N, Prinz J, Nestle FO, Schön MP, Ischaiah D, et al. Psoriasis and systemic inflammatory diseases: potential mechanistic links between skin disease and co-morbid conditions. J Invest Dermatol. 2010;130(7):1785–1796.
14. Miller IM, Ellervik C, Yazdanyar S, Jemec GB. Meta-analysis of psoriasis, cardiovascular disease, and associated risk factors. J Am Acad Dermatol. 2013;69(6):1014–1024.
15. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. J Clin Endocrinol Metab. 2008;93(11 Suppl 1):S9–30.
16. Kimball AB, Gladman D, Gelfand JM, Gordon K, Horn EJ, Korman NJ, et al. National Psoriasis Foundation clinical consensus on psoriasis comorbidities and recommendations for screening. J Am Acad Dermatol. 2008;58(6):1031–1042.
17. Sterry W, Strober BE, Menter A; International Psoriasis Council. Obesity in psoriasis: the metabolic, clinical and therapeutic implications. Report of an interdisciplinary conference and review. Br J Dermatol. 2007;157(4):649–655.
18. World Medical Association. Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191–2194.
19. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO Consultation. World Health Organ Tech Rep Ser. 2000;894:i–xii, 1–253.
20. Feldman SR, Zhao Y, Navaratnam P, Herrera V, Lu J, Tian H. Patterns of medication utilization and costs associated with the use of etanercept, adalimumab, and ustekinumab in the management of moderate-to-severe psoriasis. J Dermatol Treat. 2014;25(2):174–178.
21. Biswas A. Clinical implications of generalised plaque psoriasis in association with metabolic syndrome: a hospital-based comparative cross-sectional study. AkiNik J Dermatol Clin Res. 2025 [In Press].
22. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412–419.