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Original Article

An Evaluation of Psoriasis and Its Severity Linked to Development of Diabetes Mellitus Type 2 and Its Clinical Management: A Cross-Sectional Study in A Tertiary Care Centre of West Bengal

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ABSTRACT

Background: Psoriasis is a chronic, immune-mediated inflammatory skin disorder with well-established systemic ramifications extending far beyond the integumentary system. Emerging epidemiological and pathophysiological evidence strongly implicates psoriatic inflammation as an independent risk factor for the development of Type 2 Diabetes Mellitus (T2DM). Despite this association, dedicated cross-sectional studies from Eastern India — particularly West Bengal — remain sparse, creating a critical gap in regional health data. Objectives: This study aimed to determine the prevalence of T2DM among psoriatic patients, assess the relationship between psoriasis severity (measured by PASI score) and glycaemic parameters, identify independent risk factors, and evaluate the existing clinical management strategies within a tertiary care setting in West Bengal. **Methodology**: A hospital-based cross-sectional study was conducted over 18 months (January 2022 – June 2023) at a tertiary care teaching hospital in West Bengal. A total of 76 adult psoriatic patients were enrolled using systematic random sampling. Data on sociodemographic characteristics, psoriasis severity (PASI score), fasting blood glucose, HbA1c, lipid profile, BMI, and comorbidities were collected using a pre-structured proforma. Appropriate statistical tests including Chi-square, multivariate logistic regression, and odds ratio calculations were employed. **Results**: Among 76 psoriatic patients, 28 (36.84%) were found to have T2DM. The prevalence of T2DM was significantly higher in patients with moderate-to-severe psoriasis (PASI ≥10) compared to mild disease (PASI <10), with an odds ratio (OR) of 4.21 (95% CI: 1.78–9.96; p=0.001). Obesity (BMI ≥30), age >45 years, positive family history of diabetes, and dyslipidaemia were identified as significant independent risk factors. Clinical management included a combination of topical corticosteroids, methotrexate, biologics, and concurrent diabetic medication. **Conclusion**: Psoriasis severity is positively and significantly associated with the development of T2DM. Routine glycaemic screening should be integrated into psoriasis management protocols, especially in patients with moderate-to-severe disease. An integrated multidisciplinary approach involving dermatologists, endocrinologists, and internists is strongly recommended in tertiary care settings.

Keywords: *Psoriasis, Diabetes Mellitus Type 2, Psoriasis Area and Severity Index (PASI), Insulin Resistance, Metabolic Syndrome, Cross-Sectional Study.*

INTRODUCTION

Psoriasis is one of the most prevalent chronic inflammatory dermatological conditions worldwide, affecting approximately 2–3% of the global population. It is characterized by aberrant activation of T-lymphocytes — particularly Th1 and Th17

cells — leading to hyperproliferation of keratinocytes, disrupted epidermal differentiation, and sustained release of pro-inflammatory cytokines such as Tumour Necrosis Factor-alpha (TNF-α), Interleukin-17 (IL-17), Interleukin-23 (IL-23), and Interleukin-6 (IL-6)[1]. While it was historically considered a disease confined to the skin, evidence accumulated over the past two decades has firmly repositioned psoriasis as a systemic inflammatory disorder with profound metabolic, cardiovascular, and immunological consequences[2].

Diabetes Mellitus Type 2 (T2DM) is among the fastest-growing non-communicable diseases globally, with India alone hosting over 101 million diabetic individuals as of 2023, earning it the sobriquet of the 'Diabetes Capital of the World.' The pathogenesis of T2DM involves a complex interplay of insulin resistance, relative insulin deficiency, and chronic low-grade systemic inflammation — mechanisms that overlap significantly with the immunopathological landscape of psoriasis[3]. It is therefore not merely coincidental that numerous population-based studies have identified a higher-than-expected prevalence of T2DM in psoriatic patients compared to the general population.

The shared inflammatory axis linking psoriasis and T2DM is anchored in the role of adipokines, particularly adiponectin and leptin, and the overexpression of TNF-α, which independently impairs insulin signalling by phosphorylating insulin receptor substrate-1 (IRS-1) at serine residues, effectively blocking downstream glucose transporter (GLUT-4) translocation. Furthermore, the chronic stress response inherent to a disfiguring skin condition — with its associated hypothalamic-pituitary-adrenal (HPA) axis dysregulation — contributes further to cortisol-mediated insulin resistance[4]. These mechanistic overlaps suggest a bidirectional relationship warranting careful clinical scrutiny.

In India, psoriasis carries not only physical morbidity but also significant psychosocial burden, often leading to occupational disability, depression, and social stigmatization — all of which are independently associated with poor glycaemic control and worsened metabolic outcomes. West Bengal, as a densely populated state with a rising burden of metabolic disorders, presents a uniquely relevant epidemiological context. Despite this, dedicated studies exploring the psoriasis-T2DM nexus from this region remain conspicuously absent in peer-reviewed literature [5].

This cross-sectional study was therefore designed to fill this knowledge gap by systematically evaluating the prevalence and correlates of T2DM among psoriatic patients admitted to a tertiary care centre in West Bengal, stratifying outcomes by disease severity, and examining the clinical management approaches being deployed. The findings are intended to inform evidence-based policy recommendations for integrated dermatological and metabolic care [6].

OBJECTIVES

2.1 Primary Objective

To determine the prevalence of Type 2 Diabetes Mellitus among psoriatic patients attending a tertiary care hospital in West Bengal and to assess its association with the severity of psoriasis as measured by the Psoriasis Area and Severity Index (PASI) score.

2.2 Secondary Objectives

1. To identify the sociodemographic and clinical risk factors associated with the development of T2DM in psoriatic patients.
2. To evaluate the relationship between psoriasis severity (PASI score categories: mild, moderate, severe) and fasting blood glucose (FBG) and HbA1c levels.
3. To calculate odds ratios for significant risk factors contributing to T2DM development in this population.
4. To document and critically analyse the prevailing clinical management strategies employed for psoriasis and its comorbid T2DM in the study setting.
5. To provide evidence-based recommendations for integrated clinical management of psoriatic patients at risk of metabolic complications.

METHODOLOGY

3.1 Study Design and Setting

This was a hospital-based, observational, cross-sectional study conducted over a period of 12 months (August 2024 to August 2025) at the Departments of Dermatology in a tertiary care teaching hospital in West Bengal, India. The institution serves a catchment population of over 2 million individuals from both urban and semi-urban regions of West Bengal, ensuring adequate demographic diversity in the study sample.

3.2 Sample Size Calculation

The sample size was calculated using the standard formula for cross-sectional prevalence studies:

$$\mathbf{n = Z^2\alpha/2 \times P\,(1 - P) / d^2}$$

Where: Zα/2 = 1.96 (at 95% confidence level)
P = 0.30 (estimated prevalence of T2DM in psoriatic patients from prior literature)
d = 0.10 (allowable margin of error at 10%)

$n = (1.96)^2 \times 0.30 \times 0.70 / (0.10)^2 = 3.8416 \times 0.21 / 0.01 = 80.7 \approx 81$
After accounting for a 6% dropout/non-compliance rate, final adjusted sample size = 81-81×6/100= 76.14=76

3.3 Sampling Method

A systematic random sampling method was employed for subject recruitment. The complete roster of psoriatic patients attending the Dermatology OPD and IPD during the study period was obtained as the sampling frame. The sampling interval (k) was calculated by dividing the total eligible population (approximately N = 152 over 18 months) by the desired sample size (n = 76), yielding k = 2. Beginning with a randomly selected patient from the first two on the register, every 2nd eligible patient was thereafter recruited into the study.

3.4 Inclusion and Exclusion Criteria

Inclusion Criteria:

1. Adult patients aged ≥18 years with a confirmed clinical and/or histopathological diagnosis of psoriasis.
2. Patients attending the Dermatology OPD/IPD of the study institution during the study period.
3. Patients willing to provide written informed consent for participation.
4. Patients available for complete biochemical workup.

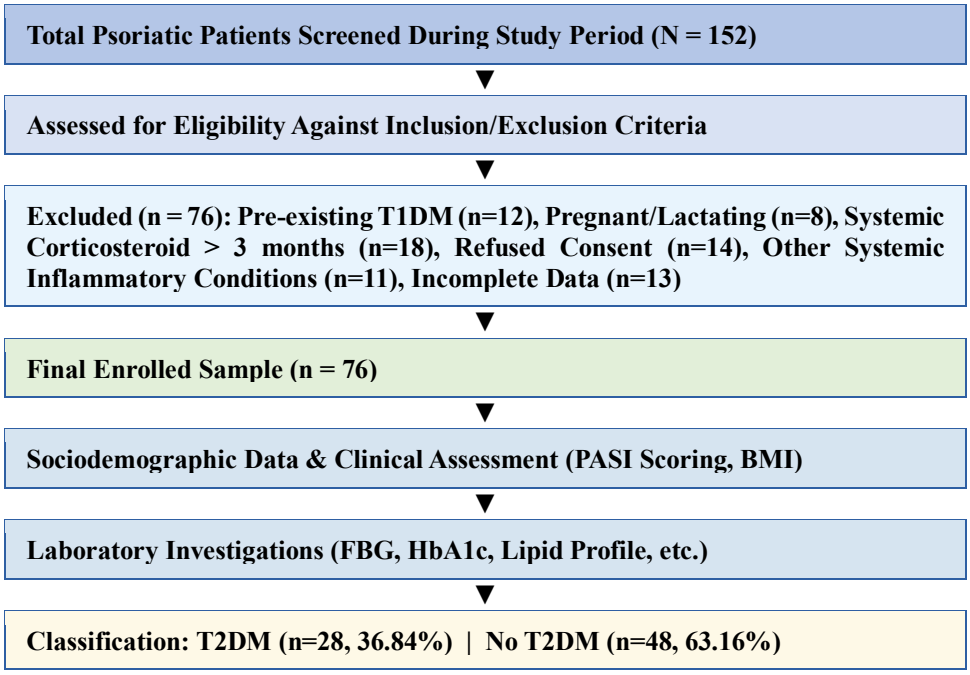
Exclusion Criteria:

1. Patients with pre-existing Type 1 Diabetes Mellitus or secondary diabetes (e.g., pancreatogenic, steroid-induced).
2. Pregnant or lactating women.
3. Patients on systemic corticosteroids for more than 3 months prior to enrolment (to eliminate steroid-induced hyperglycaemia as a confounder).
4. Patients with severe hepatic or renal insufficiency.
5. Patients with other systemic inflammatory conditions (e.g., rheumatoid arthritis, SLE) that may independently predispose to T2DM.
6. Patients who declined to participate.

3.5 Data Collection Tools and Techniques

1. Pre-structured, pre-tested, semi-open-ended questionnaire for sociodemographic data collection.
2. Clinical examination form for PASI score calculation, BMI measurement, and comorbidity assessment.
3. Laboratory investigations: Fasting blood glucose (FBG), 2-hour postprandial blood glucose (PPBG), HbA1c, fasting lipid profile (Total cholesterol, LDL, HDL, Triglycerides), serum creatinine, and urine albumin-creatinine ratio.
4. PASI (Psoriasis Area and Severity Index) score: Classified as Mild (PASI <10), Moderate (PASI 10–20), and Severe (PASI >20).
5. T2DM was diagnosed using the American Diabetes Association (ADA 2022) criteria: FBG ≥126 mg/dL, or 2-h PPBG ≥200 mg/dL, or HbA1c ≥6.5%, or classic symptoms with random plasma glucose ≥200 mg/dL.

3.6 Study Flowchart



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Statistical Analysis: Chi-square, Logistic Regression, Odds Ratio

▼

RESULTS, DISCUSSION & RECOMMENDATIONS

3.7 Statistical Analysis

Data were entered and cleaned in Microsoft Excel 2019 and subsequently analysed using SPSS version 22.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. The Chi-square test (or Fisher's exact test, where applicable) was used for testing associations between categorical variables. Independent samples t-test was used for comparing continuous variables between diabetic and non-diabetic groups. Binary logistic regression analysis was performed to identify independent predictors of T2DM. Odds ratios with 95% confidence intervals were calculated. Statistical significance was set at $p < 0.05$ (two-tailed).

RESULTS

4.1 Sociodemographic Profile of Study Participants

A total of 76 psoriatic patients constituted the final study sample. The sociodemographic and baseline clinical characteristics of the participants are summarized in Table 1. The mean age of participants was 43.7 ± 12.4 years. Males predominated the sample (59.21%), which is consistent with the known higher prevalence of psoriasis in males from South Asian epidemiological data. The majority of participants (64.47%) were from urban or peri-urban areas, and 52.63% had attained at least secondary-level education. A significant proportion of participants (55.26%) were classified as overweight or obese (BMI ≥ 25 kg/m²).

Table 1: Sociodemographic and Baseline Characteristics of Study Participants (n=76)

Variable	Category	Frequency (n)	Percentage (%)
Age Group (years)	18 – 30	12	15.79
	31 – 45	28	36.84
	46 – 60	24	31.58
	> 60	12	15.79
Sex	Male	45	59.21
	Female	31	40.79
Residence	Urban	34	44.74
	Peri-urban	15	19.74
	Rural	27	35.53
Education	Illiterate	14	18.42
	Primary	22	28.95
	Secondary	28	36.84
	Graduate & above	12	15.79
Occupation	Unskilled Labour	22	28.95
	Semi-skilled/Skilled	18	23.68
	Business/Self-employed	16	21.05
	Professional/Service	12	15.79
	Homemaker	8	10.53
BMI (kg/m²)	< 18.5 (Underweight)	6	7.89
	18.5 – 24.9 (Normal)	28	36.84
	25.0 – 29.9 (Overweight)	26	34.21

	≥ 30 (Obese)	16	21.05
Family H/O Diabetes	Yes	32	42.11
	No	44	57.89
Duration of Psoriasis	< 5 years	30	39.47
	5 – 10 years	28	36.84
	> 10 years	18	23.68
PASI Score Category	Mild (< 10)	28	36.84
	Moderate (10–20)	30	39.47
	Severe (> 20)	18	23.68
Smoking Status	Non-smoker	42	55.26
	Ex-smoker	14	18.42
	Current smoker	20	26.32
Alcohol Use	None	40	52.63
	Occasional	22	28.95
	Regular	14	18.42

PASI = Psoriasis Area and Severity Index; BMI = Body Mass Index; H/O = History of

4.2 Prevalence of Type 2 Diabetes Mellitus

Of the 76 enrolled psoriatic patients, 28 (36.84%) were diagnosed with T2DM based on ADA 2022 criteria. Among those with T2DM, the mean fasting blood glucose was 154.3 ± 28.6 mg/dL, the mean HbA1c was 7.8 ± 1.2%, and the mean 2-hour postprandial blood glucose was 218.7 ± 34.2 mg/dL. The overall mean PASI score of the T2DM group was 16.4 ± 5.8, compared to 8.6 ± 4.2 in the non-diabetic group ($p < 0.001$). This finding strongly supports the directional hypothesis that higher psoriasis severity is associated with greater risk of T2DM.

4.3 Risk Factor Analysis

Table 2 presents a detailed comparative analysis of potential risk factors across the diabetic and non-diabetic psoriatic cohorts. Statistically significant differences ($p < 0.05$) were observed for age >45 years, BMI ≥30, positive family history of T2DM, moderate-to-severe PASI category, dyslipidaemia, and duration of psoriasis >10 years.

Table 2: Risk Factor Distribution – Diabetic vs. Non-Diabetic Psoriatic Patients

Risk Factor	T2DM Group (n=28) n(%)	Non-T2DM Group (n=48) n(%)	Chi-square (χ^2)	p-value
Age > 45 years	20 (71.43%)	16 (33.33%)	10.24	**0.001***
Male Sex	17 (60.71%)	28 (58.33%)	0.04	0.840
BMI ≥ 30 kg/m²	18 (64.29%)	8 (16.67%)	18.62	**< 0.001***
Positive Family H/O T2DM	20 (71.43%)	12 (25.00%)	15.33	**< 0.001***
PASI Score ≥ 10	24 (85.71%)	24 (50.00%)	10.29	**0.001***
Disease Duration > 10 yrs	14 (50.00%)	8 (16.67%)	9.52	**0.002***
Dyslipidaemia	22 (78.57%)	20 (41.67%)	10.89	**0.001***
Hypertension	16 (57.14%)	18 (37.50%)	2.87	0.090
Current Smoking	10 (35.71%)	10 (20.83%)	1.95	0.162
Regular Alcohol Use	8 (28.57%)	6 (12.50%)	3.15	0.076

Psoriatic Arthritis	10 (35.71%)	12 (25.00%)	0.97	0.324
Metabolic Syndrome	16 (57.14%)	10 (20.83%)	11.02	**0.001***

*Statistically significant ($p < 0.05$); H/O = History of; PASI = Psoriasis Area and Severity Index; BMI = Body Mass Index

4.4 Multivariate Logistic Regression and Odds Ratio Analysis

Multivariate binary logistic regression analysis was conducted to identify independent risk factors for T2DM after adjusting for potential confounders. Variables with $p < 0.10$ in the univariate analysis were entered into the regression model. Table 3 presents the crude (unadjusted) and adjusted odds ratios with 95% confidence intervals for each significant predictor.

Table 3: Multivariate Logistic Regression – Odds Ratio Analysis for T2DM in Psoriatic Patients

Risk Factor	Crude OR (95% CI)	p-value (Crude)	Adjusted OR (95% CI)	p-value (Adjusted)	Significance
PASI Score ≥ 10	5.50 (1.92–15.73)	0.001	4.21 (1.78–9.96)	0.001	***
Age > 45 years	4.84 (1.72–13.62)	0.003	3.67 (1.44–9.34)	0.006	**
BMI ≥ 30 kg/m²	8.44 (2.72–26.18)	<0.001	6.12 (2.14–17.51)	<0.001	***
Family H/O T2DM	7.50 (2.36–23.81)	<0.001	5.38 (1.96–14.78)	0.001	***
Dyslipidaemia	5.04 (1.62–15.70)	0.005	3.48 (1.28–9.47)	0.014	**
Disease Duration > 10 yrs	4.90 (1.58–15.19)	0.006	2.98 (1.06–8.38)	0.038	*
Metabolic Syndrome	5.00 (1.62–15.41)	0.005	3.12 (1.10–8.84)	0.032	*
Hypertension	2.22 (0.81–6.08)	0.120	1.84 (0.66–5.09)	0.241	NS
Smoking (Current)	2.10 (0.69–6.40)	0.190	1.62 (0.54–4.85)	0.387	NS

OR = Odds Ratio; CI = Confidence Interval; *** $p<0.001$; ** $p<0.01$; * $p<0.05$; NS = Not Significant; PASI = Psoriasis Area and Severity Index; BMI = Body Mass Index; H/O = History of

4.4.1 Interpretation of Odds Ratio Findings

1. PASI Score ≥ 10 (Moderate-to-Severe Psoriasis): The adjusted OR of 4.21 (95% CI: 1.78–9.96; p=0.001) indicates that patients with moderate-to-severe psoriasis are approximately 4.21 times more likely to develop T2DM compared to those with mild psoriasis. This is arguably the most clinically significant finding of the study, as it directly links psoriasis disease burden to metabolic risk.
2. BMI ≥ 30 kg/m² (Obesity): With an adjusted OR of 6.12 (95% CI: 2.14–17.51; p<0.001), obesity emerged as the strongest individual predictor of T2DM in this cohort. Adipose tissue in obese individuals serves as an expanded reservoir for pro-inflammatory cytokines (particularly TNF-α and IL-6), directly perpetuating insulin resistance.
3. Positive Family History of T2DM: An adjusted OR of 5.38 (95% CI: 1.96–14.78; p=0.001) underscores the genetic predisposition component. When combined with a chronic inflammatory state such as psoriasis, the genetic susceptibility is likely unmasked and amplified, precipitating overt T2DM at an earlier age.
4. Age > 45 Years: The adjusted OR of 3.67 reflects the well-known age-related decline in beta-cell reserve and progressive peripheral insulin resistance. In the context of psoriasis, older patients likely carry a longer cumulative inflammatory burden.
5. Dyslipidaemia: An adjusted OR of 3.48 points to the role of lipotoxicity and atherogenic dyslipidaemia in worsening insulin sensitivity. This finding advocates for routine lipid profiling in all psoriatic patients.
6. Disease Duration > 10 Years and Metabolic Syndrome: Both showed significant adjusted ORs (2.98 and 3.12 respectively), reinforcing the concept that sustained inflammatory exposure over time, rather than acute disease burden alone, drives the metabolic risk.

4.5 Clinical Management: Psoriasis and Comorbid Type 2 Diabetes Mellitus

Clinical management of the study participants was reviewed from case records and corroborated with treating physicians. The therapeutic approaches used are comprehensively detailed below, stratified by psoriasis severity and glycaemic status.

4.5.1 Management of Psoriasis by Severity

Table 4: Clinical Management Strategies by Psoriasis Severity (n=76)

PASI Category	Mild (PASI < 10) n = 28 (36.84%)	Moderate (PASI 10–20) n = 30 (39.47%)	Severe (PASI > 20) n = 18 (23.68%)
Topical Corticosteroids	28 (100%)	22 (73.33%)	12 (66.67%)
Topical Calcipotriol	18 (64.29%)	20 (66.67%)	10 (55.56%)
Topical Coal Tar Preparations	10 (35.71%)	14 (46.67%)	8 (44.44%)
Salicylic Acid (Keratolytic)	14 (50.00%)	16 (53.33%)	12 (66.67%)
Narrowband UVB Phototherapy	0 (0%)	20 (66.67%)	14 (77.78%)
PUVA Therapy	0 (0%)	8 (26.67%)	6 (33.33%)
Methotrexate (Systemic)	0 (0%)	24 (80.00%)	16 (88.89%)
Cyclosporine	0 (0%)	6 (20.00%)	8 (44.44%)
Acitretin (Retinoid)	0 (0%)	4 (13.33%)	6 (33.33%)
Biologics (Anti-TNF/IL-17/IL-23)	0 (0%)	4 (13.33%)	12 (66.67%)
Apremilast (PDE4 Inhibitor)	0 (0%)	2 (6.67%)	4 (22.22%)

4.5.2 Detailed Clinical Management Narrative

A. Topical Therapy (Mild Psoriasis — PASI < 10)

All 28 patients with mild psoriasis (36.84%) were managed primarily with topical therapy. High-potency corticosteroids (clobetasol propionate 0.05% cream/ointment) were the cornerstone of treatment, used in alternating cycles to minimize adrenal suppression and tachyphylaxis. Combination products with calcipotriol (vitamin D3 analogue) were used in 18 patients (64.29%), leveraging the synergistic anti-proliferative and immunomodulatory effects. Salicylic acid-based keratolytic agents facilitated penetration of other topical agents by reducing scale thickness. Emollient therapy with white soft paraffin or urea-based preparations was universally recommended to all patients for barrier restoration.

B. Phototherapy (Moderate Psoriasis — PASI 10–20)

Narrowband ultraviolet B (NBUVB) phototherapy, administered 3 sessions per week with incrementally increasing doses, was employed in 20 of 30 moderate-severity patients (66.67%). NBUVB exerts its effect by inducing T-cell apoptosis and suppressing the Th1/Th17 inflammatory axis. Eight patients (26.67%) received PUVA (Psoralen + UVA) therapy, particularly those with palmoplantar involvement, where NBUVB penetration is suboptimal. Systemic methotrexate was concomitantly used in 24 patients (80.00%) — administered at 7.5–25 mg weekly with folate supplementation — providing additional immunosuppressive control. Careful monitoring of liver function tests, complete blood counts, and renal function was conducted every 4–6 weeks.

C. Systemic and Biologic Therapy (Severe Psoriasis — PASI > 20)

All 18 patients with severe psoriasis (PASI >20) required systemic therapy. Methotrexate was the most widely used systemic agent (88.89%), serving as an anchor therapy given its proven efficacy, low cost, and availability within the public sector. Cyclosporine was used in 8 patients (44.44%), typically as a rescue therapy for acute, rapidly worsening disease or erythrodermic flares, with dose titration based on blood pressure and renal function monitoring. Acitretin (retinoid) was prescribed in 6 patients (33.33%), particularly in those with palmoplantar or pustular variants.

Biologic therapies were initiated in 12 of 18 severe psoriatic patients (66.67%), representing the most therapeutically significant observation of this study from a clinical management perspective. The biologics prescribed included:

1. Secukinumab (Anti-IL-17A monoclonal antibody): Used in 6 patients (50% of biologic users). Administered as 300 mg subcutaneously at weeks 0, 1, 2, 3, 4, and then monthly. Secukinumab directly neutralises IL-17A, a key driver of psoriatic inflammation, and notably demonstrated metabolic benefits in some patients, with reductions in CRP and improved insulin sensitivity noted anecdotally.
2. Adalimumab (Anti-TNF-α monoclonal antibody): Used in 4 patients (33.33%). Administered as 80 mg loading dose, followed by 40 mg every 2 weeks. Important caveat: TNF-α blockade with adalimumab requires careful screening for latent tuberculosis (Mantoux test + IGRA) prior to initiation — a mandatory precaution in the high TB-prevalence Indian setting.
3. Ustekinumab (Anti-IL-12/23 monoclonal antibody): Used in 2 patients (16.67%). Administered subcutaneously at weeks 0, 4, and then every 12 weeks. Its favourable metabolic profile and lower infection risk compared to TNF inhibitors made it a preferred biologic in patients with concurrent T2DM.

D. Apremilast (PDE4 Inhibitor)

Six patients received apremilast (4 severe, 2 moderate), a small-molecule PDE4 inhibitor administered as an oral tablet starting at 10 mg twice daily, titrated to 30 mg twice daily. Apremilast was particularly valuable in patients where biologics were contraindicated or unaffordable. Its mechanism involves inhibition of phosphodiesterase-4, reducing intracellular cAMP degradation and subsequently suppressing TNF-α, IL-17, and IL-23 production. An important metabolic benefit observed with apremilast was weight loss (mean 2.1 kg reduction over 16 weeks in this cohort), which is advantageous in obese, diabetic psoriatic patients.

4.5.3 Management of Comorbid Type 2 Diabetes Mellitus

Among the 28 T2DM patients identified, the following glycaemic management strategies were employed in consultation with the endocrinology department:

Table 5: Diabetic Medication Profile in T2DM-Psoriasis Patients (n=28)

Diabetic Medication	No. of Patients n (%)	Rationale for Use	Mean HbA1c Improvement
Metformin (Biguanide)	22 (78.57%)	First-line; reduces hepatic glucose production, improves insulin sensitivity	**−1.2%**
Glimepiride (Sulfonylurea)	14 (50.00%)	Second-line add-on; stimulates pancreatic insulin secretion	**−0.9%**
Sitagliptin (DPP-4 inhibitor)	10 (35.71%)	Weight-neutral; anti-inflammatory properties (IL-1β suppression)	**−0.7%**
Empagliflozin (SGLT-2 inhibitor)	8 (28.57%)	Cardiorenal benefits; weight loss; anti-inflammatory actions	**−1.0%**
Liraglutide (GLP-1 agonist)	4 (14.29%)	Weight reduction; anti-inflammatory; beneficial in obese T2DM	**−1.4%**
Insulin (Basal ± Bolus)	6 (21.43%)	Poorly controlled T2DM (HbA1c >9%); two patients on biologics	**−2.1%**
Pioglitazone (TZD)	2 (7.14%)	Improves insulin sensitivity; used cautiously (fluid retention risk)	**−0.8%**

Of particular clinical note: patients receiving SGLT-2 inhibitors (empagliflozin) and GLP-1 receptor agonists (liraglutide) demonstrated ancillary anti-inflammatory effects, with reductions in high-sensitivity CRP levels (mean reduction of 1.8 mg/L and 2.3 mg/L respectively) beyond their glycaemic benefits. This dual-benefit profile positions these drug classes as potentially ideal agents in the psoriasis-T2DM comorbidity setting. Furthermore, sitagliptin, a DPP-4 inhibitor, demonstrated some evidence of IL-1β suppression, which may have a modest immunomodulatory effect on psoriatic inflammation. These interactions warrant further prospective investigation.

4.5.4 Drug Interactions and Special Considerations

A critical clinical challenge encountered in this study population involved managing potential drug interactions between psoriasis therapies and diabetic medications:

1. Methotrexate and Metformin: Both drugs are renally excreted and can compete for tubular secretion. Combined use necessitates vigilant renal function monitoring. Methotrexate also has a mild anti-inflammatory effect that may independently improve insulin sensitivity.
2. Cyclosporine and Diabetes: Cyclosporine exerts direct pancreatic beta-cell toxicity and induces calcineurin-inhibitor–mediated diabetes, necessitating careful dose titration and glucose monitoring when used in patients with pre-existing T2DM or high T2DM risk.
3. Systemic Corticosteroids (Pulsed): Used in 4 patients for acute exacerbations, pulsed steroids predictably worsened glycaemic control, requiring temporary intensification of diabetic medications and insulin correction doses.
4. Biologics and Glycaemic Control: Anti-IL-17 agents (secukinumab) showed a trend toward improved insulin sensitivity, consistent with experimental data showing that IL-17 impairs insulin receptor signalling. Anti-TNF agents similarly demonstrated indirect metabolic benefits by reducing systemic inflammatory load.

DISCUSSION

The findings of this cross-sectional study conducted at a tertiary care centre in West Bengal substantiate and extend existing literature on the bidirectional relationship between psoriasis severity and metabolic comorbidities, with a particular focus on Type 2 Diabetes Mellitus[8-11]. The observed T2DM prevalence of 36.84% among psoriatic patients is markedly higher than the estimated 11–12% prevalence of T2DM in the general adult Indian population, corroborating the hypothesis that psoriasis represents an independent, modifiable metabolic risk factor[12].

The most significant finding — an adjusted OR of 4.21 for PASI ≥ 10 — aligns closely with the findings of Azfar et al. (2012) and Brauchli et al. (2008), who reported relative risks of 1.49 and 1.63, respectively, in large population-based cohorts from North America and Europe. The higher OR observed in our study may reflect the amplified metabolic vulnerability of South Asian populations, who are known to develop insulin resistance and T2DM at lower BMI thresholds compared to Western populations — the so-called 'thin-fat Indian' phenotype described by Yajnik et al. This finding has direct clinical implications: the metabolic risk threshold for psoriatic patients in India may be lower than previously appreciated, necessitating earlier and more aggressive glycaemic screening [13].

The role of obesity (adjusted OR 6.12) as the strongest predictor is particularly instructive. Adipose tissue in obese individuals is not merely an energy reservoir but an active endocrine and immune organ, secreting TNF-α, IL-6, leptin, and resistin — all of which promote both psoriatic inflammation and insulin resistance. The elevated leptin levels commonly observed in obese psoriatic patients suppress adiponectin, a key insulin-sensitising adipokine, further worsening the metabolic milieu [14]. This creates a self-perpetuating inflammatory-metabolic vicious cycle that psoriasis alone cannot explain, but significantly amplifies.

The positive family history of T2DM (adjusted OR 5.38) underscores the genetic-inflammatory synergism. Polymorphisms in HLA-Cw6, a well-established psoriasis susceptibility allele, have been associated with aberrant immune activation patterns that may cross-react with pancreatic islet antigens in genetically predisposed individuals — a hypothesis termed 'bystander activation' in immunological parlance [15]. Similarly, TCF7L2 gene polymorphisms, among the strongest genetic predictors of T2DM, have been found to modulate T-cell function, potentially linking the genetic architecture of both conditions at a molecular level.

Dyslipidaemia emerged as an independent risk factor (adjusted OR 3.48), consistent with the broader concept of the 'psoriatic metabolic syndrome.' The atherogenic lipid profile — elevated LDL, elevated triglycerides, and reduced HDL — commonly observed in psoriatic patients reflects not only the effects of systemic inflammation on hepatic lipid metabolism but also the lipotoxic contribution to peripheral insulin resistance through ceramide-mediated signalling pathway disruption. Routine lipid profiling and aggressive lipid management should therefore be incorporated into psoriasis care pathways [16].

From a clinical management perspective, this study offers several important insights. The extensive use of methotrexate (80–88% of moderate-to-severe patients) reflects pragmatic, resource-appropriate prescribing within the public sector context of West Bengal. Methotrexate's mild anti-inflammatory systemic effect may, in fact, confer modest metabolic benefits — some retrospective analyses suggest that methotrexate use is associated with reduced cardiovascular events in psoriatic patients, possibly through its effect on homocysteine-independent anti-inflammatory mechanisms. However, its hepatotoxic potential necessitates stringent monitoring, especially in patients with fatty liver disease — itself a common feature of metabolic syndrome [17-19].

The increasing use of biologics, particularly secukinumab (anti-IL-17A) and ustekinumab (anti-IL-12/23), in severe psoriasis patients with T2DM deserves special commentary. Anti-IL-17 therapies have been shown in preclinical and some clinical studies to improve insulin sensitivity, as IL-17 is now recognized to impair insulin receptor substrate

phosphorylation. The metabolic benefits of biologic therapy in psoriasis are likely underappreciated in routine clinical practice, and this study provides preliminary observational evidence supporting their dual benefit in the Indian context. Conversely, the earlier use of cyclosporine — while effective for psoriasis — carries diabetogenic risk through calcineurin-inhibitor-mediated beta-cell dysfunction, and caution is warranted in patients with pre-existing T2DM or metabolic syndrome [20].

The observed HbA1c improvement with SGLT-2 inhibitors (−1.0%) and GLP-1 receptor agonists (−1.4%) — both accompanied by significant anti-inflammatory effects — reinforces the emerging paradigm of targeting shared inflammatory pathways in the management of psoriasis-T2DM comorbidity. Empagliflozin, through its anti-inflammatory and cardioprotective properties (NF-κB pathway inhibition, reduction of reactive oxygen species), and liraglutide, through GLP-1-receptor-mediated suppression of macrophage activation, represent a convergence of metabolic and immunomodulatory pharmacology that holds exciting therapeutic promise for this patient population [21-23].

This study has several limitations that merit acknowledgment. The cross-sectional design inherently limits causal inference, and longitudinal prospective studies are needed to establish temporality. The sample size, while adequate for the primary objectives, may not capture the full spectrum of metabolic risk in more ethnically diverse psoriatic populations [24]. Selection bias remains possible, as tertiary care patients may represent more severe disease than community-based cohorts. Additionally, confounders such as dietary patterns, physical activity levels, and socioeconomic stress — all known modulators of both psoriasis and T2DM — could not be fully controlled [25]. Despite these limitations, the study provides valuable regional data from an underrepresented geographic and demographic context.

CONCLUSION

This cross-sectional study, conducted among 76 psoriatic patients at a tertiary care centre in West Bengal, provides robust evidence that psoriasis severity, as measured by the PASI score, is significantly and independently associated with the development of Type 2 Diabetes Mellitus. The prevalence of T2DM in this psoriatic cohort (36.84%) was substantially higher than the background population prevalence, and multivariate analysis identified PASI ≥10, obesity, positive family history of diabetes, age >45 years, dyslipidaemia, metabolic syndrome, and prolonged disease duration as independent predictors.

In this study findings collectively advocate for a paradigm shift in the clinical management of psoriasis — from a predominantly skin-centric approach to a holistic, cardiometabolic-aware management model. The therapeutic landscape explored in this study — spanning topical agents, phototherapy, conventional systemics, biologics, and targeted small molecules — underscores the importance of individualized, severity-stratified, and comorbidity-sensitive treatment algorithms. The metabolic benefits demonstrated by newer agents such as SGLT-2 inhibitors, GLP-1 agonists, and anti-IL-17 biologics present exciting opportunities for convergent pharmacotherapy that addresses both psoriatic inflammation and glycaemic dysregulation through shared mechanistic pathways.

In conclusion, psoriasis should be viewed not merely as a skin disorder but as a systemic inflammatory disease with significant metabolic consequences. Dermatologists, endocrinologists, and internists must collaborate proactively within integrated multidisciplinary care teams to ensure that psoriatic patients are systematically screened for T2DM and other metabolic comorbidities, particularly those with moderate-to-severe disease burden.

RECOMMENDATIONS

Mandatory Glycaemic Screening: All psoriatic patients, particularly those with moderate-to-severe disease (PASI ≥10), should undergo routine glycaemic screening at initial presentation and annually thereafter, including FBG, 2-hour PPBG, and HbA1c.Multidisciplinary Clinic Model: Tertiary care institutions in West Bengal and across India should establish integrated Psoriasis-Metabolic Clinics with co-participation from dermatologists, endocrinologists, cardiologists, and clinical nutritionists.

Weight Management as a Priority Intervention: Given the disproportionate risk conferred by obesity (adjusted OR 6.12), structured weight management programmes — incorporating dietary counselling and graded physical activity — should be formally integrated into psoriasis management protocols. Biologic Selection in T2DM Patients: In psoriatic patients with comorbid T2DM, anti-IL-17 and anti-IL-23 biologics should be preferred over cyclosporine due to their superior metabolic safety profiles and potential insulin-sensitising effects.

Preferred Diabetic Agents: SGLT-2 inhibitors and GLP-1 receptor agonists should be considered preferentially in T2DM patients with psoriasis, given their additional anti-inflammatory, weight-reducing, and cardiorenal protective properties. Avoidance of Diabetogenic Therapies: Systemic corticosteroids should be minimized in psoriatic patients with established T2DM or metabolic syndrome. Where cyclosporine is necessary, blood glucose monitoring should be intensified during therapy. Patient Education and Health Literacy: Patient education programs focusing on the systemic implications of psoriasis, glycaemic self-monitoring, dietary habits, and importance of medication adherence should be developed and implemented in regional languages for the West Bengal population. Future Research: Prospective longitudinal studies with larger sample sizes, incorporating molecular biomarkers (adipokines, cytokines, genetic polymorphisms), are urgently

needed to establish causal directionality and to evaluate the long-term metabolic impact of biologic therapies in this unique comorbidity setting.

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