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Original Research Article

Beyond The Skin: Prolactin in Psoriasis as a Biomarker of Disease Severity

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Abstract

Background: Psoriasis is a long-term immune-mediated skin condition marked by keratinocyte hyperproliferation. The anterior pituitary secretes the multifunctional neuropeptide hormone prolactin, which has been linked to immunological regulation and keratinocyte proliferation, indicating a possible role in the pathogenesis of psoriasis. There are still few data from the Indian subcontinent despite several studies conducted worldwide.

Objectives: to compare psoriasis patients' blood prolactin levels to those of controls and to establish a correlation between prolactin levels and clinical metrics such as disease severity indicators (PASI and BSA).

Methods: A case-control study was conducted at Navodaya Medical College, Hospital & Research Centre, Raichur, from February 2024 to June 2025. A total of 140 participants, 70 psoriasis patients and 70 age- and sex-matched controls were enrolled. Serum prolactin levels were measured and correlated with epidemiological variables, BSA, and PASI scores using independent t-test, Chi-square test, ANOVA, and Pearson/Spearman correlation analysis.

Results: The mean serum prolactin level was significantly higher in cases (13.24 ± 9.12 ng/mL) compared to controls (6.84 ± 2.91 ng/mL; $p < 0.001$). Elevated prolactin was observed in 15 cases (21.4%) compared to only 4 controls (5.7%). Female patients exhibited significantly higher prolactin levels (18.62 ± 13.07 ng/mL) than males (11.03 ± 6.24 ng/mL; $p = 0.003$). Prolactin levels were associated significantly with PASI score ($p = 0.001$) and BSA ($p = 0.041$). The highest mean prolactin was noted in the PASI 41–55 group (19.84 ± 12.43 ng/mL).

Conclusion: Psoriasis patients have significantly higher serum prolactin levels, which are correlated with the severity of the condition as determined by PASI and BSA. Prolactin should be taken into account in the immunopathogenesis of psoriasis and may be a clinically available biomarker for tracking its severity.

Keywords: Psoriasis; Prolactin; PASI; BSA; Disease severity; Biomarker; Immunopathogenesis; Keratinocyte proliferation.

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Introduction

About 2-3% of people worldwide suffer from psoriasis, a chronic immune-mediated skin condition with a wide range of clinical manifestations. Due to hyperproliferation of keratinocytes and altered differentiation patterns, it causes an eight-fold increase in epidermal cell turnover, which primarily appears as well-defined, erythematous, scaly papules and plaques. [1]

The disease presents with diverse morphological variants and involves not only the skin but also the nails and joints, conferring significant morbidity and psychosocial burden on affected individuals. [2] The anterior pituitary glands lactotroph cells secrete the neuropeptide hormone prolactin. Prolactin is involved in calcium metabolism and osmoregulation in addition to its traditional functions in lactation, reproduction, and maternal behavior [3,4]. A "Prolactin circuit" between the skin and the central nervous system is a bidirectional neuroendocrine axis that links emotional and physiological stressors to cutaneous inflammation. Prolactin is particularly relevant to dermatology since it regulates both cutaneous growth and immune function.

Prolactin mechanistically causes keratinocytes, epithelial cells, and lymphocytes to proliferate. It causes keratinocytes to produce more of the interferon-gamma (IFN-γ)-induced chemokines

CXCL9, CXCL10, and CXCL11. These chemokines then attract Th1 and Th17 lymphocytes to psoriatic plaques, intensifying the inflammatory cascade that defines the condition [3].This molecular crosstalk underscores the potential for prolactin to function not merely as a bystander but as an active participant in psoriasis immunopathogenesis. Psychoemotional stress is a recognised trigger of psoriasis exacerbations, and emerging evidence suggests that stress-induced variations in prolactin a key neuroendocrine modulator of stress responses may mediate at least some of these cutaneous effects. [5] This positions hyperprolactinaemia not only as a consequence of chronic inflammatory states but potentially as a driver of disease flares.

The connection between prolactin and psoriasis has been investigated in a number of worldwide research, with varying degrees of success. Some studies have found a favorable correlation between the severity of the disease and considerably high serum prolactin in psoriatic individuals; however, other studies have not found such an association. Crucially, there is still a dearth of Indian data on this topic, which restricts generalizability to the local genetic and demographic setting. Therefore, the current study was conducted to assess serum prolactin levels in psoriasis patients in a tertiary care facility and to compare results with recognized indicators of disease severity.

Objectives:

The specific objectives of this study were:

1. To assess the clinical and epidemiological characteristics of psoriasis patients.
2. To assess the differences in serum prolactin levels between individuals with psoriasis and age and sex-matched controls
3. To establish a correlation between clinical disease severity metrics, such as Body Surface Area (BSA) and Psoriasis Area and Severity Index (PASI), and blood prolactin levels.

Materials and Methods

A hospital-based case-control study was conducted in the Outpatient Department of Dermatology at Navodaya Medical College, Raichur, over a period of 18 months. Patients aged 18 to 70 years with a confirmed clinical diagnosis of psoriasis vulgaris attending the Dermatology OPD were recruited. Written informed consent was given by each subject.

Patients with conditions such as prolactinoma, thyroid illness, and acromegaly, renal failure, and hepatic failure, tumors of the central nervous system, cancer, post-transplant status, or cardiovascular events that are known to affect prolactin levels were not included. Patients on medications known to change prolactin levels, such as antipsychotics, antidepressants, dopaminergic blockers, opioids, cimetidine, ranitidine, verapamil, or oestrogens, were also excluded. The same exclusion criteria used for cases also applied to controls, who were matched for age and sex and did not have psoriasis and attended the outpatient clinic.

Sample size: Sample size was calculated using the formula: $n = 2SD^2 (Z^{1-\alpha/2} + Z^{1-\beta})^2 / d^2$. Using a standard deviation of 6.74 for serum prolactin, a 95% confidence level (Z = 1.96), 80% power (Z = 0.84), and a clinically significant difference (d) of 4 ng/mL, the required sample size was 45 per group. Accounting for potential dropouts, 70 cases and 70 age- and sex-matched controls were enrolled.

Assessment: Every individual who was enrolled had a comprehensive dermatological examination and a full clinical history. Body Surface Area (BSA) involvement and the Psoriasis Area and Severity Index (PASI) were used to measure the severity of the disease.

Comorbidities, lifestyle factors (smoking, alcohol consumption), joint engagement, nail alterations, and family history were all methodically documented. Each participant had a 5 mL random venous blood sample taken in the Navodaya Medical College, Raichur laboratory under aseptic circumstances. The conventional enzyme-linked immunosorbent test (ELISA) was used to evaluate serum prolactin levels. For males and non-pregnant females, the reference range for normal serum prolactin was 2–25 ng/mL and 2–29 ng/mL, respectively; readings beyond the top limit were considered high.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using SPSS version 22.0. Categorical variables were expressed as frequencies and percentages and compared using the Chi-square test or Fisher's exact test. Continuous variables were presented as mean ± SD. Normality was assessed using the Shapiro–Wilk test. Independent samples t-test was used to compare mean serum prolactin levels between cases and controls, while one-way ANOVA was used for multiple group comparisons. Correlation between serum prolactin levels and disease severity indices (PASI and BSA) was assessed using Pearson's or Spearman's correlation analysis, as appropriate. A p-value <0.05 was considered statistically significant.

Results

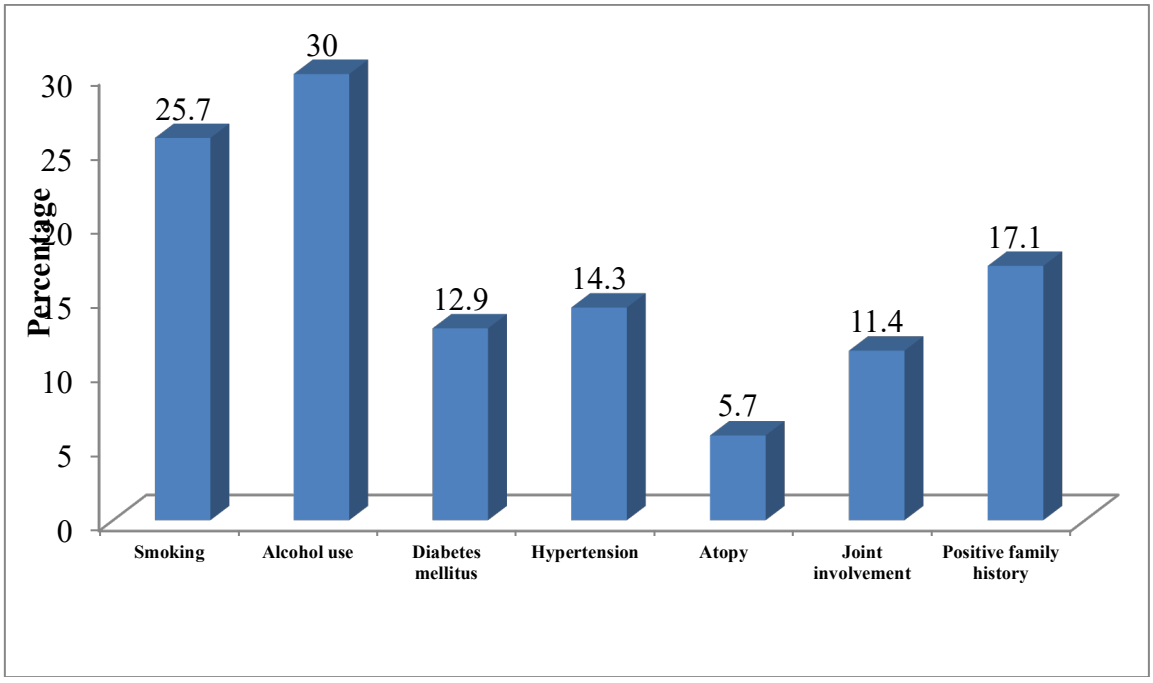
A total of 140 participants were enrolled: 70 psoriasis cases and 70 age- and sex-matched healthy controls. The majority of cases (38.6%) belonged to the 18–30 year age group, followed by 27.1% in the 31–40 year group, reflecting a

predominantly younger cohort. Male preponderance was noted, with 50 males (71.4%) and 20 females (28.6%), yielding a male-to-female ratio of 2.5:1. Age and sex distribution did not differ significantly between cases and controls (p = 0.512 and p = 0.163, respectively), confirming adequate matching. Frequency matching for age and sex was performed.

Table 1: Comparison of age and sex distribution between cases and controls

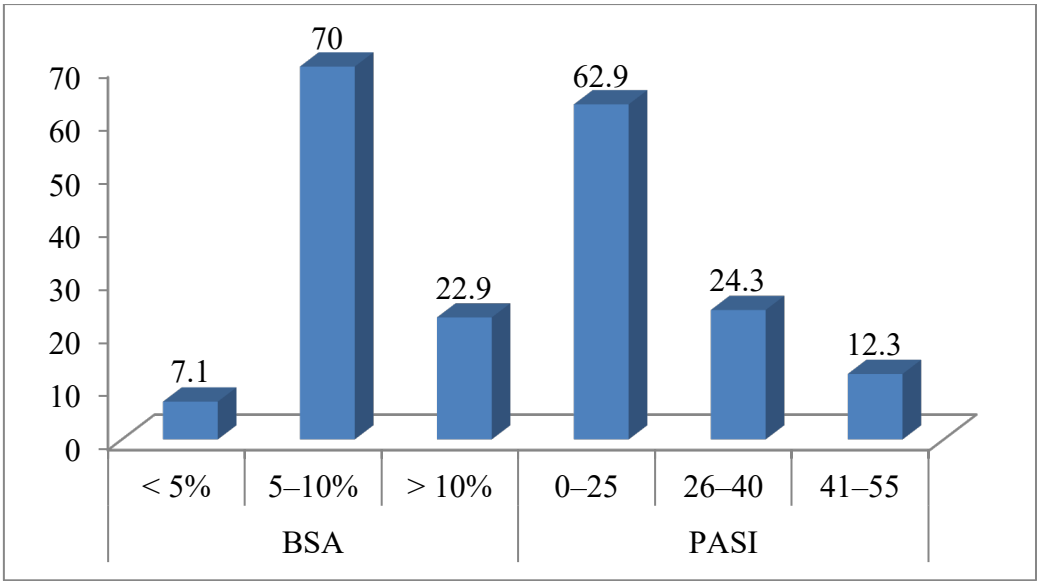
Variable	Cases (n=70)	%	Controls (n=70)	%	p-value
Age 18–30	27	38.6%	33	47.1%	0.512
Age 31–40	19	27.1%	17	24.3%	
Age 41–50	11	15.7%	9	12.9%	
Age 51–60	7	10.0%	6	8.6%	
Age 61–70	6	8.6%	5	7.1%	
Male	50	71.4%	42	60.0%	0.163
Female	20	28.6%	28	40.0%	

Most patients (65.7%) had age of onset below 40 years. In terms of disease duration, 38.6% had disease for 0–6 months, 25.7% for 6–12 months, and 22.9% for 1–3 years; thus, 87.1% of patients had disease duration of less than 3 years. Farmers (30.0%) and students (27.1%) constituted the predominant occupational groups. Seasonal aggravation predominantly during winter was reported by 52.9% of cases. Stress as a triggering factor was identified in 18.6% of patients. Among documented risk factors, alcohol use was the most prevalent 21(30.0%), followed by smoking 18(25.7%). Hypertension was present in 10(14.3%) and diabetes mellitus in 9(12.9%) of cases. A positive family history was elicited in 12(17.1%). Joint involvement was observed in 8(11.4%) cases. Atopy was uncommon, noted in only 4(5.7%) of patients.



Graph 1: Risk factor and comorbidity distribution in psoriasis cases (n=70)

The most common primary site of involvement was the knees (67.1%), followed by scalp (57.1%), elbows (54.3%), trunk (14.3%), and palms and soles (4.3%). On progression, lower limbs were involved in 97.1% of patients, trunk in 90.0%, upper limbs in 74.3%, and scalp in 58.6%. Nail changes were noted in 52.9% of cases, of which pitting the most common abnormality was (91.9%), followed by subungual hyperkeratosis (67.6%), onycholysis (62.2%), and salmon patch (29.7%). The majority of patients (70.0%) had moderate psoriasis (BSA 5–10%), 22.9% had severe psoriasis (BSA > 10%), and 7.1% had mild disease (BSA < 5%).



Graph 2: BSA and PASI distribution among cases (n=70)

The mean serum prolactin level in cases was 13.24 ± 9.12 ng/mL, ranging from 3.84 to 42.67 ng/mL. Controls demonstrated a substantially lower mean of 6.84 ± 2.91 ng/mL (range: 3.11 to 18.43 ng/mL). The mean serum prolactin level was significantly higher in psoriasis patients compared to controls, with a mean difference of 6.40 ng/mL. The difference was highly statistically significant ($p < 0.001$). Elevated prolactin levels were identified in 15 cases (21.4%) compared to only 4 controls (5.7%), further underscoring the differential burden in the psoriatic cohort.

Table 2: Comparison of mean serum prolactin between cases and controls

Group	Mean± SD (ng/mL)	Range	p-value
Cases (n=70)	13.24 ± 9.12	3.84–42.67	**< 0.001**
Controls (n=70)	6.84 ± 2.91	3.11–18.43	

Prolactin correlation with demographic and clinical variables:

Gender: Female patients demonstrated significantly higher serum prolactin levels (18.62 ± 13.07 ng/mL) compared to males (11.03 ± 6.24 ng/mL; $p = 0.003$), consistent with known physiological differences in prolactin secretion and oestrogenic modulation of lactotroph activity.

Age: Mean prolactin varied across age groups — 11.82 ± 7.21 (18–30 yrs), 14.17 ± 9.83 (31–40 yrs), 13.44 ± 8.56 (41–50 yrs), 11.96 ± 6.74 (51–60 yrs), and 16.03 ± 9.12 ng/mL (61–70 yrs) but no statistically significant inter-group difference was noted ($p = 0.842$).

Disease Duration: A progressive trend toward higher prolactin was observed with increasing disease duration: 10.42 ± 7.13 ng/mL (0–6 months), 12.87 ± 8.94 (6–12 months), 14.23 ± 9.67 (1–3 years), 16.11 ± 10.42 (3–5 years), and 18.74 ± 11.38 ng/mL (> 5 years), though this did not reach statistical significance ($p = 0.418$).

Comorbidities and Lifestyle Factors: No statistically significant differences in serum prolactin were observed across smoking, alcohol use, diabetes mellitus, hypertension, atopy, or BMI subgroups. However, diabetics (16.23 ± 7.84 ng/mL) and hypertensives (15.47 ± 8.12 ng/mL) trended toward higher values compared to their respective counterparts.

Table 3: Serum prolactin levels across clinical subgroups

Variable	Subgroup	Mean ± SD (ng/mL)	n	p-value
Sex	Male	11.03 ± 6.24	50	**0.003**
	Female	18.62 ± 13.07	20	
BMI (kg/m²)	18.5–24.9 (Normal)	11.47 ± 6.18	38	0.214
	25–29.9 (Overweight)	15.83 ± 11.37	24	
	≥ 30 (Obese)	14.92 ± 10.61	8	
Smoking	Smokers	13.84 ± 7.12	18	0.731
	Non-smokers	13.02 ± 9.74	52	
Alcohol	Users	13.94 ± 6.84	21	0.647

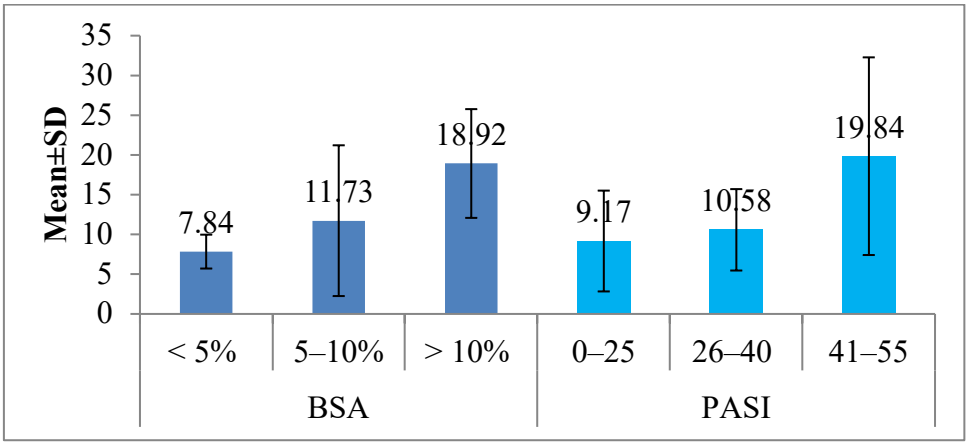
	Non-users	12.97 ± 9.83	49	
Diabetes	Yes	16.23 ± 7.84	9	0.318
	No	12.87 ± 9.26	61	
Hypertension	Yes	15.47 ± 8.12	10	0.412
	No	12.93 ± 9.18	60	
Joint Involvement	Yes	17.24 ± 7.43	8	0.197
	No	12.77 ± 9.24	62	
Nail Changes	Yes	15.18 ± 10.84	37	0.184
	No	11.08 ± 6.73	33	

Prolactin correlation with disease severity (BSA and PASI): A progressive and statistically significant increase in mean serum prolactin was observed across BSA categories: 7.84 ± 2.13 ng/mL (BSA < 5%, n=5), 11.73 ± 9.48 ng/mL (BSA 5–10%, n=49), and 18.92 ± 6.84 ng/mL (BSA > 10%, n=16; overall p = 0.041), suggesting a strong positive association between prolactin level and extent of cutaneous involvement.

More strikingly, a highly significant graded elevation of prolactin was observed across PASI categories: 9.17 ± 6.34 ng/mL (PASI 0–25, n=44), 10.58 ± 5.12 ng/mL (PASI 26–40, n=17), and 19.84 ± 12.43 ng/mL (PASI 41–55, n=9; overall p = 0.001). Serum prolactin levels showed a significant positive correlation with PASI score (r = 0.52, p = 0.001) and BSA involvement (r = 0.34, p = 0.041), suggesting an association between elevated prolactin levels and increasing psoriasis severity. The most severe psoriasis subgroup exhibited prolactin levels more than twice those of the milder disease group, highlighting the potential utility of prolactin as a clinically trackable severity biomarker.

Table 4: Serum prolactin levels stratified by BSA and PASI

Severity Index	Category	Mean ± SD (ng/mL)	p-value
BSA	< 5% (Mild)	7.84 ± 2.13	**0.041**
	5–10% (Moderate)	11.73 ± 9.48	
	> 10% (Severe)	18.92 ± 6.84	
PASI	0–25 (Mild)	9.17 ± 6.34	**0.001**
	26–40 (Moderate)	10.58 ± 5.12	
	41–55 (Severe)	19.84 ± 12.43	



Graph 3: Mean serum prolactin levels across by BSA and PASI categories

Discussion

This case-control study provides robust evidence that serum prolactin is significantly elevated in patients with psoriasis vulgaris compared to healthy controls, with a strong graded correlation with disease severity as measured by PASI.

The mean serum prolactin in cases (13.24 ± 9.12 ng/mL) was nearly twice that of controls (6.84 ± 2.91 ng/mL), a difference of high statistical significance (p < 0.001). This mirrors findings reported by Almohamady et al [6] (cases: 12.41 ± 6.05 ng/mL; controls: 5.53 ± 2.97 ng/mL) and Keen MA et al [7] (cases: 11.29 ± 8.05 ng/mL; controls: 7.90 ± 3.93 ng/mL), both of whom demonstrated comparable case-control differentials. Giasuddin et al [8] and Dilme-Carreras et al [9] similarly reported significantly elevated prolactin in psoriatic patients. Elsherif et al. [10], in a multi-disease dermatological case-control study (n=25 psoriasis patients; Egypt, 2015), reported a mean of 16.90 ± 6.80 ng/mL versus 7.10 ± 3.20 ng/mL in

controls ($p < 0.001$). Talukdar C et al [11] reported the highest case mean of 19.20 ± 6.70 ng/mL versus controls at 8.60 ± 3.40 ng/mL ($p < 0.05$), confirming that prolactin elevation is reproducible across Indian geographic and genetic cohorts.

Elevated prolactin was detected in 15 cases (21.4%) versus 4 controls (5.7%) a substantially higher proportion. The biological rationale for hyperprolactinaemia in psoriasis is compelling. Prolactin receptors are expressed on keratinocytes, T lymphocytes, and antigen-presenting cells. Prolactin stimulates keratinocyte proliferation directly and indirectly via upregulation of IFN-γ-induced chemokines (CXCL9, CXCL10, CXCL11), which orchestrate the recruitment of Th1 and Th17 cells into dermal and epidermal layers. The net effect is amplification of the inflammatory milieu that drives the psoriatic phenotype. This immunostimulatory mechanism may explain not only elevated baseline levels but also the stress-induced flares observed clinically.

Dissenting findings by Gorpelioglu et al [12], Priestley et al [13], and Handjani et al [14] who found no significant case-control difference may reflect differences in sample size, patient selection criteria, prolactin assay methodology, and the timing of blood collection relative to disease activity. Notably, stress-induced surge in prolactin is transient; studies failing to capture active disease phases may underestimate true differences.

Female patients in our study had substantially higher prolactin levels than males (18.62 ± 13.07 vs 11.03 ± 6.24 ng/mL; $p = 0.003$). This is consistent with the well-recognised oestrogen-mediated upregulation of prolactin secretion in women. Oestrogen directly stimulates lactotroph proliferation and increases prolactin gene expression, establishing a physiological baseline elevation in females. Almohamady et al [6] reported similar sex-related differences. In contrast, Gupta et al [15] and Robati et al [16] found male-predominant elevations, possibly reflecting cohort-specific hormonal or lifestyle confounders. The clinical implication is that sex-stratified reference ranges should be considered when using prolactin as a disease marker in psoriasis.

The predominance of cases in the 18–30 and 31–40 year age groups (65.7%) aligns with similar findings by Keen MA et al [7] and Aalemi AK et al [17] who identified the second and third decades as peak susceptibility periods. Psychosocial stressors, obesity, and genetic predisposition are likely contributors to early-onset disease in this demographic. While mean prolactin showed a numerical trend across age groups notably higher in the 61–70 year group (16.03 ± 9.12 ng/mL) no statistically significant age-prolactin association was identified ($p = 0.842$), consistent with Ghiasi et al. [18]

Disease duration showed a numerically ascending prolactin trend (10.42 ng/mL at 0–6 months to 18.74 ng/mL at > 5 years), without reaching statistical significance ($p = 0.418$). Almohamady et al [6] and Elsherif et al [10] did demonstrate a significant positive correlation in their larger cohorts, suggesting that duration-prolactin correlations may emerge with greater statistical power.

The most clinically significant finding of this study is the graded elevation of serum prolactin across increasing PASI categories (9.17 → 10.58 → 19.84 ng/mL; $p = 0.001$). The near-doubling of prolactin in the severe PASI group (41–55) compared to mild disease is a compelling signal that prolactin tracks disease burden. A parallel and statistically significant trend was observed with BSA (7.84 → 11.73 → 18.92 ng/mL; $p = 0.041$). Nasim et al [19] reported a statistically significant prolactin-PASI correlation in their 50-patient cohort. Saini PK et al [20] demonstrated significant differences between both case-control groups and disease severity subgroups. Almohamady ASA et al [6], Dilme-Carreras E et al [9], Elsherif NA et al [10] and Gupta M et al [15]. collectively provide a convergent evidence base that prolactin elevation is not merely an epiphenomenon but may actively amplify disease severity.

This clinical utility is further underscored by the practical advantages of prolactin assay: it is inexpensive, widely available in resource-limited settings, and interpretable alongside standard biochemical panels. As psoriasis management increasingly moves toward targeted monitoring, a simple neuroendocrine marker that tracks with PASI could complement or supplement existing tools. Joint involvement, present in 11.4% of cases, was associated with a numerically higher mean prolactin (17.24 ± 7.43 vs 12.77 ± 9.24 ng/mL), though the difference did not reach statistical significance ($p = 0.197$). Hedman et al [21] found no significant prolactin elevation in synovial fluid of psoriatic arthritis patients, while Botezatu et al [22] and Husakova et al [23] raised the possibility that prolactin may serve as a biomarker in psoriatic arthritis independent of disease activity.

The limited sample size ($n = 8$) in the joint involvement subgroup in our study restricts definitive conclusions; a larger cohort would be needed to determine if prolactin can help distinguish psoriatic arthritis from uncomplicated psoriasis vulgaris. Nail changes, found in 52.9% of cases, trended toward higher prolactin (15.18 ± 10.84 ng/mL) compared to those without nail involvement (11.08 ± 6.73ng/mL), without statistical significance. Male predominance

(71.4%; M:F ratio 2.5:1) is consistent with many Indian series including Nasim et al [19] (2.6:1) and Nayak PB et al. (3.36:1) [24]. Seasonal aggravation in winter (50%) was attributed to dry skin, occlusive clothing, and reduced ultraviolet exposure factors well-established in dermatological literature.

Conclusion

This study shows that blood prolactin levels are meaningfully correlated with objective measures of disease severity, specifically the PASI score, and are considerably higher in psoriasis patients than in age- and sex-matched controls. Higher prolactin is independently predicted by female sex, which is consistent with oestrogen-mediated control of lactotroph function. Serum prolactin shows promise as a biomarker of the severity of psoriasis, especially in healthcare settings with limited resources where advanced biomarker panels might not be regularly available. To determine its function in therapeutic response monitoring, longitudinal studies assessing prolactin dynamics before and after treatment with concurrent PASI monitoring are necessary.

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