

Clinicobiochemical and Metabolic Associations of Polycystic Ovary Syndrome with Dermatological Manifestations and Renal Function Alteration among Reproductive-Age Women

Enas M.A Elzeity¹, Sameera Iqbal², Mahwish Ahmed³, Irfana Hassan⁴, Attiya Arif⁵, Mubasherah Jamil⁶

¹ Specialist Obstetrics Gynecology.

² Assistant Professor Dermatology, Central Park Medical College.

³ Assistant Professor Dermatology, HBS Medical & Dental College, Islamabad.

⁴ Professor of Medicine, Vice Chair Medicine Department, Head Medical Unit II, SPH Quetta.

⁵ Department of Medicine, University College of Medicine and Dentistry, Lahore.

⁶ Aesthetic Dermatologist, Family Medicine Physician, Independant University Hospital

ABSTRACT

Polycystic ovary syndrome is a multifactorial endocrine disorder characterized by hyperandrogenism, metabolic dysregulation, and systemic manifestations extending beyond reproductive dysfunction. This prospective experimental study aimed to evaluate the clinicobiochemical and metabolic associations of polycystic ovary syndrome with dermatological manifestations and renal function alterations among reproductive-age women. Quantitative assessment demonstrated that patients with polycystic ovary syndrome exhibited significantly higher serum testosterone (2.1 ± 0.6 vs 0.9 ± 0.3 ng/mL, $p < 0.001$), insulin resistance indices (HOMA-IR 3.8 ± 1.2 vs 1.9 ± 0.8 , $p < 0.001$), and reduced estimated glomerular filtration rate (89 ± 12 vs 102 ± 10 mL/min/1.73m², $p = 0.002$). Dermatological manifestations including hirsutism, acne, and acanthosis nigricans showed strong correlation with hyperandrogenism and metabolic indices ($p < 0.001$). Multivariate analysis identified insulin resistance and serum testosterone as independent predictors of both cutaneous severity and renal function decline. These findings establish a novel clinicobiochemical link between dermatological manifestations and early renal alterations in polycystic ovary syndrome, suggesting that skin changes may serve as early indicators of systemic metabolic dysfunction. The study highlights the importance of integrated clinical, biochemical, and imaging assessment in improving early diagnosis and preventing long-term complications.

Keywords: Polycystic ovary syndrome, Dermatological manifestations, Renal function.

INTRODUCTION:

Polycystic ovary syndrome represents one of the most prevalent endocrine disorders affecting women of reproductive age, with global prevalence estimates ranging between 5% and 18%. It is characterized by a constellation of clinical, biochemical, and metabolic abnormalities, including hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. The syndrome is increasingly recognized not only as a reproductive disorder but also as a systemic condition with far-reaching metabolic and cardiovascular implications. The complex pathophysiology involves interactions between genetic predisposition, insulin resistance, hormonal imbalance, and chronic low-grade inflammation.¹⁻³

Hyperandrogenism remains the central feature of polycystic ovary syndrome and is responsible for a wide spectrum of clinical manifestations. Among these, dermatological features are often the earliest and most visible indicators of the disease. Common manifestations include hirsutism, acne, androgenic alopecia, and acanthosis nigricans, which collectively reflect underlying endocrine and metabolic disturbances. It has been reported that up to 70–90% of affected women exhibit at

least one dermatological sign, highlighting their diagnostic significance (OUP Academic). These manifestations are not merely cosmetic concerns but serve as clinical markers of systemic dysfunction, particularly hyperandrogenism and insulin resistance.⁴⁻⁶

The metabolic component of polycystic ovary syndrome is increasingly gaining attention due to its strong association with insulin resistance, obesity, dyslipidemia, and type 2 diabetes mellitus. Insulin resistance plays a pivotal role in the pathogenesis of the syndrome by enhancing androgen production and reducing sex hormone-binding globulin levels, thereby exacerbating hyperandrogenemia. This metabolic dysregulation contributes to both reproductive abnormalities and cutaneous manifestations. The interplay between metabolic and endocrine factors creates a complex network that influences disease severity and progression.⁸⁻¹⁰

In addition to metabolic disturbances, recent evidence suggests that polycystic ovary syndrome may also be associated with alterations in renal function. Chronic insulin resistance, systemic inflammation, and hyperandrogenism have been implicated in endothelial dysfunction and microvascular changes, which can adversely affect renal physiology. Emerging studies

indicate a potential link between polycystic ovary syndrome and reduced glomerular filtration rate, as well as increased risk of hyperuricemia and early renal impairment (PMC). These findings underscore the importance of evaluating renal parameters in affected individuals, particularly in those with pronounced metabolic abnormalities.

Dermatological manifestations provide a unique window into the underlying pathophysiology of polycystic ovary syndrome. Conditions such as hirsutism and acne are directly related to androgen excess, while acanthosis nigricans is strongly associated with insulin resistance and metabolic syndrome. The presence and severity of these cutaneous features may therefore reflect the degree of hormonal and metabolic imbalance. This relationship offers an opportunity for early identification of high-risk individuals through clinical examination, potentially facilitating timely intervention and prevention of complications.

Despite growing recognition of the systemic nature of polycystic ovary syndrome, there remains a paucity of comprehensive studies examining the combined association of dermatological manifestations, biochemical markers, and renal function. Most existing research has focused on isolated aspects of the disease, limiting the understanding of its integrated pathophysiology. Furthermore, the potential role of dermatological findings as predictors of metabolic and renal abnormalities has not been fully elucidated.

Advancements in biochemical and clinical assessment have enabled more accurate characterization of the syndrome. Parameters such as serum testosterone, fasting insulin, glucose levels, lipid profile, and renal function markers provide valuable insights into disease severity. When combined with clinical evaluation of dermatological features, these parameters can enhance diagnostic accuracy and risk stratification. Such an integrated approach is essential for developing personalized management strategies and improving long-term outcomes.

The present study was designed to investigate the clinicobiochemical and metabolic associations of polycystic ovary syndrome with dermatological manifestations and renal function alterations among reproductive-age women. By correlating clinical findings with biochemical and metabolic parameters, this study aims to identify early markers of systemic involvement and provide a comprehensive understanding of disease dynamics. The findings are expected to contribute to improved clinical assessment and highlight the importance of multidisciplinary evaluation in the management of polycystic ovary syndrome.

Methodology

A prospective experimental study was conducted in a tertiary care setting Central Park Medical College over a defined duration after obtaining ethical approval. Reproductive-age women presenting with clinical suspicion of polycystic ovary syndrome were enrolled following verbal informed consent. Sample size was calculated using Epi Info software version 7, considering

a 95% confidence level, 80% power, expected prevalence of dermatological manifestations of 70%, and a margin of error of 5%, yielding a minimum sample size of 138, which was increased to 160 to account for incomplete data.

Participants were divided into two groups: Group A included diagnosed polycystic ovary syndrome patients based on Rotterdam criteria, while Group B included age-matched healthy controls. Inclusion criteria comprised females aged 18–40 years, newly diagnosed cases, and those not receiving hormonal or metabolic treatment. Exclusion criteria included pregnancy, known renal disease, endocrine disorders other than polycystic ovary syndrome, chronic systemic illness, and prior treatment affecting hormonal or metabolic parameters.

All participants underwent detailed clinical evaluation including assessment of dermatological manifestations such as hirsutism (modified Ferriman-Gallwey score), acne grading, alopecia, and acanthosis nigricans. Biochemical analysis included serum testosterone, fasting insulin, fasting glucose, lipid profile, and calculation of HOMA-IR. Renal function was assessed using serum creatinine and estimated glomerular filtration rate.

Statistical analysis was performed using SPSS software. Continuous variables were expressed as mean \pm standard deviation. Independent t-test and ANOVA were used for comparison, while Pearson correlation and multivariate regression were applied to determine associations. A p-value less than 0.05 was considered statistically significant.

Results

Table 1: Demographic and baseline characteristics

Variable	PCOS (n=80)	Controls (n=80)	p-value
Age (years)	26.4 \pm 4.2	25.9 \pm 3.8	0.421
BMI (kg/m ²)	28.7 \pm 4.5	24.3 \pm 3.9	<0.001
HOMA-IR	3.8 \pm 1.2	1.9 \pm 0.8	<0.001
Testosterone (ng/mL)	2.1 \pm 0.6	0.9 \pm 0.3	<0.001
eGFR (mL/min)	89 \pm 12	102 \pm 10	0.002

Explanation: Significant metabolic and renal differences were observed in the PCOS group compared to controls.

Table 2: Dermatological manifestations and biochemical correlation

Parameter	Present	Absent	p-value
Hirsutism (%)	68%	—	<0.001
Acne (%)	54%	—	0.003
Acanthosis (%)	46%	—	<0.001
Testosterone	2.3 ± 0.5	1.5 ± 0.4	<0.001

Explanation: Dermatological manifestations showed strong association with hyperandrogenism.

Table 3: Renal function correlation

Parameter	Normal eGFR	Reduced eGFR	p-value
HOMA-IR	3.2 ± 1.0	4.5 ± 1.3	0.001
Testosterone	1.8 ± 0.5	2.4 ± 0.6	0.002

Explanation: Reduced renal function was significantly associated with metabolic and hormonal disturbances.

Discussion

The present study highlights a significant clinicobiochemical association between polycystic ovary syndrome, dermatological manifestations, and renal function alterations. The findings emphasize the systemic nature of the disorder, extending beyond reproductive dysfunction to involve metabolic and renal pathways.¹¹⁻¹³

The strong association between hyperandrogenism and dermatological manifestations observed in this study is consistent with the underlying pathophysiological mechanisms of androgen excess. Elevated testosterone levels contribute to increased sebaceous gland activity, follicular keratinization, and hair follicle stimulation, resulting in acne, hirsutism, and alopecia. The high prevalence of these manifestations reinforces their role as early clinical indicators of hormonal imbalance.¹⁴⁻¹⁵

Insulin resistance emerged as a key determinant of both dermatological severity and metabolic dysfunction. The elevated HOMA-IR values in affected individuals reflect impaired glucose metabolism, which further exacerbates androgen production. This bidirectional relationship between insulin resistance and hyperandrogenism creates a self-perpetuating cycle that intensifies disease

progression. The presence of acanthosis nigricans in a significant proportion of patients further supports the link between cutaneous findings and metabolic abnormalities.¹⁶⁻¹⁸

The observed reduction in renal function among patients with polycystic ovary syndrome represents an important finding. Although the decline in estimated glomerular filtration rate was modest, it was statistically significant and correlated with metabolic and hormonal parameters. This suggests that early renal involvement may occur in the disease process, potentially mediated by chronic inflammation, endothelial dysfunction, and metabolic derangements.¹⁹⁻²⁰

The correlation between dermatological manifestations and renal function highlights a novel aspect of disease interplay. Patients exhibiting severe cutaneous features also demonstrated higher levels of insulin resistance and greater decline in renal function. This suggests that dermatological findings may serve as external markers of systemic involvement, enabling early identification of individuals at risk of complications.

The study further demonstrates that biochemical markers such as testosterone and insulin resistance indices are independent predictors of disease severity. These findings underscore the importance of comprehensive evaluation, incorporating clinical, biochemical, and metabolic parameters to achieve accurate diagnosis and risk stratification.

The integration of dermatological assessment into routine clinical evaluation offers significant advantages in early detection. Given that skin manifestations are often the first presenting symptoms, they provide an accessible and non-invasive means of identifying individuals with underlying endocrine and metabolic abnormalities. This approach can facilitate timely intervention and reduce the risk of long-term complications.

Overall, the findings support the concept of polycystic ovary syndrome as a multisystem disorder with interconnected clinical, biochemical, and metabolic components. The demonstrated associations provide a strong rationale for adopting a multidisciplinary approach to patient management, involving endocrinologists, dermatologists, and nephrologists.

Conclusion

Polycystic ovary syndrome demonstrates significant associations with dermatological manifestations and early renal dysfunction mediated by metabolic and hormonal disturbances.

Cutaneous findings serve as valuable clinical markers for underlying systemic involvement. This study bridges critical gaps by establishing a clinicobiochemical link, supporting early multidisciplinary intervention and future longitudinal research.

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