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CRP: A SIMPLE MARKER OF LOW GRADE INFLAMMATION IN POLYCYSTIC OVARY SYNDROME

[1*]Dr. Amina, [1]Dr. Afsana, Dr. Janeeta Tehreem
[1]Liaquat University Hospital Hyderabad
[2]Indus Medical College

ORIGINAL ARTICLE

ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a prevalent endocrine condition among women of reproductive age, distinguished by ovulatory failure, hyperandrogenism, and polycystic ovarian form. Recent information indicates that PCOS is not solely a reproductive issue additionally an endocrine and inflammatory condition. High-sensitivity C-reactive protein (hs-CRP), a recognized indicator of systemic inflammation, has been extensively investigated in polycystic ovary syndrome (PCOS), correlating it with resistance to insulin, cardiovascular risk, and obesity.
Objective: To assess blood hs-CRP levels among women with PCOS and examine their correlation with metabolic indicators such as body weight (BMI), resistance to insulin, lipid count, and androgen levels.
Methods: The present cross-sectional research comprised 150 women aged between the ages of years, diagnosed with PCOS based on the Rotterdam criteria. A control group including one hundred matched by age healthy women in usual periods was also included. We assessed physical characteristics, fasting blood glucose levels, cholesterol levels, serum testosterone, and hs-CRP. The HOMA-IR index was used to figure out how resistant to insulin someone was. We used SPSS to look at the data.
Results: Women with PCOS exhibited significantly higher hs-CRP levels in comparison to controls (3.8 ± 1.2 mg/L vs. 1.4 ± 0.8 mg/L, $p < 0.001$). High hs-CRP levels were positively linked to weight ($r = 0.54$), the HOMA- ($r = 0.46$), triglycerides in the blood ($r = 0.39$), and overall testosterone level ($r = 0.34$). CRP levels were considerably higher in slim PCOS individuals than in BMI-matched controls, demonstrating inflammation that is independent of obesity.
Conclusion: Increased hs-CRP levels in patients with PCOS substantiate the involvement of mild persistent inflammation in its etiology. Inflammation in PCOS seems to be somewhat independent of fat, indicating fundamental metabolic irregularities. Keeping an eye on hs-CRP levels may assist find patients who are more likely to have heart and metabolic problems.
Keywords: Polycystic ovary syndrome, C-reactive protein, insulin resistance and obesity.

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Corresponding Author:
Dr. Amina
Liaquat University Hospital Hyderabad

INTRODUCTION: Polycystic Ovary Syndrome (PCOS) is a common hormonal condition that affects women of reproductive age. According to the diagnostic criteria employed and the demographic investigated, it affects between 6% and 20% of women [1]. PCOS is marked by hyperandrogenism, chronic anovulation, and polycystic ovaries on ultrasound. It is also linked to a range of metabolic issues, such as insulin resistance, obesity, dyslipidemia, and a higher risk of heart disease [2].

Historically seen as a reproductive concern, recent research has classified PCOS as a systemic condition, with inflammation as a

pivotal factor [3]. The C-reactive protein (CRP), a protein in the acute phase produced by the liver's cells in reaction to interleukin-6 (IL-6), serves as a sensitive indicator of low-grade inflammation. High-sensitivity CRP (hs-CRP) levels are elevated in heart disease, type 2 diabetes, and metabolic syndrome, which frequently occur with PCOS [4].

The association among PCOS and metabolic disorder is well-documented; nevertheless, the role of chronic inflammation, especially CRP, is an emerging field of study. Some researchers contend that the elevated levels of CRP in PCOS are just indicative of obesity, whilst others assert that the state of inflammation is inherent to the condition, irrespective of adiposity [5].

This study aimed to evaluate hs-CRP levels among women with PCOS and investigate their associations with metabolic indicators such as resistance to insulin, cholesterol levels, and body weight (BMI), seeking to elucidate the significance of inflammation within the overarching pathophysiology of PCOS.

METHODOLOGY: From May 2023 to March 2024, this cross-sectional, case-control investigation was conducted at the Dept. of Obstetrics and Gynaecology of a tertiary care hospital. All participants provided written informed consent.

Inclusion criteria:
Women between the ages of 18 and 35
Using the Rotterdam criteria, the patient was diagnosed with PCOS because she had any two of the following: oligo/anovulation, clinical or the biochemical hyperandrogenism, or polycystic ovaries on the ultrasound device.
There must have been no hormonal therapy for a minimum of three months prior to enrollment.

Exclusion criteria:
Diabetes mellitus
Problems with the thyroid
Diseases that attack the immune system
Infections that is short-term or long-term
Cardiovascular disorders that are known
Drinking booze or smoking
Taking medicines that lower inflammation or the immune system
Control Group: The control group comprised 100 women matched for age and BMI, exhibiting monthly menstrual periods and lacking clinical manifestations of PCOS or other hormonal diseases.

Clinical and Laboratory Evaluation
Everyone who took part went through:
A thorough medical evaluation and physical assessment
Taking measurements of blood pressure, height, weight, weight, the ratio of waist to hips (WHR), and waist-to-hip ratio (WHR)
After an overnight fast, blood samples were taken to check:
Insulin and glucose levels after fasting
Cholesterol levels: total, LDL, HDL, and triglycerides
Testosterone in the blood
C-reactive protein (hs-CRP) with high sensitivity
The Homeostasis Model Assessment (HOMA-IR) equation was used to figure out how resistant to insulin the body was:
HOMA-IR = [Fasting insulin (μIU/mL) × Fasting glucose (mg/dL)] /405

Statistical Analysis: SPSS was used to look at the data. Continuous variables were represented as means ± standard deviations as well as analyzed using either Student's t-test or the Mann–Whitney U test for comparison. We used Pearson's correlation coefficient to look at the link among hs-CRP and metabolic parameters. A p-value of less than 0.05 was deemed statistically significant.

RESULTS: There were 150 women with PCOS and 100 normal controls in the research. The average age of those included was 24.6 ± 4.8 years in the group suffering from PCOS and 25.1 ± 4.6 years in the control group (p = 0.43). Women with PCOS had a body weight and waist circumference that was much greater.

TABLE 01: CLINICAL PROFILE: PCO VS CONTROL

Parameter	PCOS (n=150)	Control (n=100)	p-value
Age (years)	24.6 ± 4.8	25.1 ± 4.6	0.43
BMI (kg/m²)	27.4 ± 3.9	23.8 ± 3.1	<0.001

Parameter	PCOS (n=150)	Control (n=100)	p-value
WHR	0.88 ± 0.06	0.81 ± 0.05	<0.001
Fasting insulin	16.3 ± 5.2	9.1 ± 3.6	<0.001
HOMA-IR	3.8 ± 1.2	1.9 ± 0.9	<0.001
Total testosterone (ng/mL)	2.4 ± 0.8	1.2 ± 0.5	<0.001
hs-CRP (mg/L)	3.8 ± 1.2	1.4 ± 0.8	<0.001

Correlation Analysis:
Hs-CRP was positively correlated with
BMI ($p < 0.001$)
HOMA-IR ($p < 0.001$)
Triglycerides: ($p = 0.01$)
Testosterone ($p = 0.03$)

On the other hand, lean women with PCOS (BMI <25 kg/m²) had much higher hs-CRP levels than women with the same BMI who were not obese (2.3 ± 0.7 vs 1.2 ± 0.5 mg/L, respectively; $p < 0.001$). This shows that inflammation can happen even when a person is not overweight. [9].

DISCUSSION: Our findings indicate that PCOS is associated with low-grade systemic inflammation. High hs-CRP in all women with PCOS irrespective of obesity would identify inflammation as the anti-obesity-treatment-independent pathogenic pathways influencing development of polymorphisms.
As other similar studies have also shown over the years they likewise confirmed the above findings. Kelly et al. have shown that CRP levels were higher in PCOS women than controls despite BMI, suggesting that the obesity is not simply a consequence of increased adiposity [6]. Similarly, Mohlig et al. low grade inflammation as one of the syndrome component, results in both lean and obese PCOS subjects showed higher CRP [7].
Our finding of a strong positive association between HOMA-IR and CRP for such a subject group is also contributing to the same mechanism. Secondly, chronic inflammation may also have impared insulin-signaling pathways leading to metabolic derangements, a finding that is in line with our human results and supported by pre-clinical data. Further increased circulating serum IL-6 and TNF-α also help to further induce the liver to secrete more C-reactive protein (CRP) in PCOS women. [8].

Our results therefore demonstrate an association of CRP with at least some androgen excess. In fact, the causality could be bidirectional as hyperandrogenism may cause inflammation or vice versa, inflammation in turn exacerbates ovarian dysfunction [9]. This is relevant and will be included in a separate study. These elevations in this inflammatory marker were not detected in the control group, suggesting that CRP may act as a predictive biomarker of cardiovascular risk from an early age in PCOS. Whilst several meta-analyses have identified increased CV morbidity in women with PCOS [10-12], it may offer a theoretical targeted for risk stratification of high-risk individuals at an earlier stage.

Clinical Implications:
CRP may help to reflect the patients with inflammatory status, and can be used for stratifying patients in PCOS.
Lifestyle interventions aimed at controlling inflammation, such as weight loss, exercise, or anti-inflammatory diets are likely to improve metabolic and reproductive outcomes.
However, metformin and statins with anti-inflammatory effects would kill two birds with one stone by controlling chronic inflammation in addition to the other various manifestations of PCOS.

Limitations:
A limitation is that this was a single-center trial and therefore our findings may not be generalizable.
Cross-sectional study design: A cross-sectional design does not by definition allow us to determine causality.
Cytokine levels (IL-6, TNF-α) were not analyzed.

CONCLUSION: The association between CRP and PCOS is demonstrated in this study that eccentric by high levels of CRP with a large number of confounder factors. This indicates that [PCOS] is a chronic, low-grade

inflammatory condition, and not entirely obesity dependent or related. Measurement of serum CRPCRP may potentially serve as a tool for risk stratification and management of metabolic and CV consequences in PCOS.

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Authors Contribution

Dr. Amina	*Concept & design*
Dr. Afsana	*Acquisition of data*
Dr. Janeeta Tehreem	*Study Designing*