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A correlative study of CRP and C3 complement element in women with polycystic ovarian syndrome

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ABSTRACT

Background: Polycystic ovarian syndrome (PCOS) is a frequently occurring hormonal disorder affecting women of reproductive age. It is marked by symptoms such as infrequent or absent menstrual cycles (oligomenorrhea or amenorrhea), lack of ovulation, excessive body weight, increased body hair (hirsutism), elevated levels of androgens, and the presence of multiple small cysts within enlarged ovaries. The global prevalence of PCOS is estimated at around 7%, while in India, it ranges from approximately 4% to 11%. Only a limited number of studies have evaluated the levels of complement component 3 (C3) in individuals with PCOS. The relationship between C3, insulin resistance, and obesity in the context of PCOS remains unclear. However, some researchers reported a positive association between elevated levels of C3 and C-reactive protein (CRP) with insulin resistance and the presence of PCOS.

Aims and Objective: To evaluate the role of CRP and C3 in women with polycystic ovarian syndrome and factors related to pathogenesis of PCOS.

Materials and Methods: This hospital-based prospective cross-sectional study was conducted in the Department of Obstetrics & Gynecology at Index Medical College Hospital, Indore, from January 1, 2018, to December 31, 2019. It included 260 participants aged 15–45 years, divided into two groups: confirmed PCOS cases and healthy controls.

Results: Among the 260 participants, 130 were confirmed cases of PCOS, and the other 130 were healthy controls, determined through clinical evaluation and biochemical testing. In the PCOS group, the mean ± SD values were: body mass index (BMI) 32.97 ± 8.46, total cholesterol (TC) 188.42 ± 31.12, triglycerides (TG) 134.43 ± 50.01, high-density lipoprotein (HDL) 36.29 ± 9.55, TC/HDL ratio 5.54 ± 1.86, serum CRP 3.41 ± 0.94, and complement component C3 160.66 ± 29.15. In comparison, the control group recorded BMI 22.87 ± 2.47, TC 155.42 ± 26.33, TG 110.00 ± 42.19, HDL 41.22 ± 10.91, TC/HDL ratio 4.08 ± 1.39, serum CRP 2.25 ± 0.8, and C3 level 127.48 ± 35.60.

Conclusion: This study examined the role of CRP, C3, and various biochemical markers in PCOS patients and controls. Findings revealed that most PCOS patients exhibited elevated levels of CRP, C3, and other biochemical parameters. Both CRP and C3, indicators of chronic low-grade inflammation, were significantly higher in newly diagnosed PCOS cases compared to controls. Additionally, C3 levels showed a statistically significant correlation with increased BMI, age, marital status, and hirsutism.

Keywords: *Polycystic ovary syndrome, PCOS, CRP, C3, BMI, total cholesterol, triglyceride.*

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1. INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder in women of reproductive age, defined by persistent oligomenorrhea (less than nine menstrual cycles per year), amenorrhea (absence of menstruation for three or more months), anovulation, obesity, clinical or biochemical hyperandrogenism, and the presence of numerous follicular cysts within enlarged ovaries on imaging.Globally and in India the prevalence rate of polycystic ovarian syndrome (PCOS) is 7% and approximately 4-11% respectively, in women with reproductive age group. Stein and Leventhal noticed the association between amenorrhea, hirsutism, and enlarged polycystic ovaries, and hence was also named as the Stein Leventhal syndrome, first described PCOS in 1935. [1,2]

Polycystic ovarian syndrome is characterized by an increased number of small antral follicles with disrupted folliculogenesis and hypertrophy of the ovarian theca cell layer, with ovulatory dysfunction frequently presenting as persistent anovulation. Moreover, women with PCOS often demonstrate a spectrum of metabolic perturbations, including insulin resistance, compensatory hyperinsulinemia, dyslipidemia, and obesity. [2,3]

Insulin resistance is closely associated with diabetes, metabolic syndrome (MS), and obesity, and also serves as an independent risk factor for cardiovascular disease development. It is now well established that insulin resistance is accompanied by chronic low-grade inflammation, evidenced by elevated concentrations of high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), complement component 3 (C3), and other inflammatory mediators. Obesity represents a significant risk factor for metabolic disorders, including type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, and coronary artery disease. Approximately 40% of patients with PCOS present with obesity, most commonly demonstrating an androgenic distribution pattern characterized by an elevated waist-to-hip ratio.[2]

All these characteristics constitute components of metabolic syndrome (MS), in which low-grade inflammation serves as a key hallmark in its pathogenesis. Consequently, women with PCOS face an elevated risk of developing type 2 diabetes mellitus (T2DM), which further contributes to their increased susceptibility to cardiovascular disease, underscoring the importance of identifying reliable biomarkers of low-grade inflammation prior to the onset of associated complications in this population.[6] Most woman with PCOS produces large quantity of male sex hormones (androgens), namely hyperandrogenism leading to overgrowth of body, hair growth (hirsutism), acne, and male pattern baldness. Hyperandrogenism and dysregulated levels of various sex hormones disrupt normal ovulatory processes and menstrual cyclicity, resulting in subfertility or, in severe cases, complete infertility. Among women who achieve conception, there is an elevated risk of metabolic syndrome (MS) and pregnancy loss. Furthermore, chronic anovulation and persistent hormonal imbalances predispose affected individuals to endometrial hyperplasia and an increased risk of endometrial carcinoma.

CRP: C-reactive protein (CRP) is an acute-phase protein present in blood plasma, synthesized primarily by hepatocytes, and markedly elevated during inflammatory responses. Its primary function is to bind lysophosphatidylcholine on the membranes of apoptotic or necrotic cells, as well as certain bacteria, thereby activating the complement cascade through interaction with complement component 1q (C1q). CRP is highly conserved across vertebrate species and present in many invertebrates, reflecting its central role in systemic inflammatory responses. Plasma CRP concentrations rise rapidly following tissue injury or infection, making it a widely utilized biomarker for diagnosing inflammatory conditions. Elevated CRP levels signify an active inflammatory process, which, although a component of the innate immune defence, often indicates underlying pathology such as infection, autoimmune disorders, renal injury, or pancreatitis. Persistently high CRP levels are also associated with an increased risk of atherosclerosis and coronary artery disease, contributing to heightened susceptibility to cardiovascular events.

Complement component 3 (C3) is an acute phase protein and secretion of cytokine from liver and adipose tissue is strongly associated with dyslipidaemia, obesity, and hypertension and insulin resistance.[2,6] Very few studies have determined complement component 3 levels in PCOS patients. [7,8]The association between complement component 3, insulin resistance and obesity in PCOS is still not clear. Some researchers investigated the affiliation among C3 and CRP with insulin resistance and PCOS in their studies, also mentioned the positive correlation between rise in C3 and CRP with insulin resistance and PCOS while the strong association between C3 and PCOS was observed. [9,5] The aim of this is to evaluate the serum C3 level and CRP in PCOS in comparison with healthy controls matched for age and BMI. In different words, we have to find the changes in CRP and C3 in non-obesity patients. In addition, we wanted to find out the association between CRP and C3 with insulin resistance (As defined according to the homeostasis model assessment [HOMA] in patients with PCOS.) Only few studies have shown the correlation of complement C3 levels in PCOS patients and its association with Insulin resistance and hyperandrogenism.[7,8] This study was undertaken to investigate the relationship between complement component C3, insulin resistance, and obesity in polycystic ovary syndrome (PCOS), as this association remains inadequately elucidated.

AIMS:

Assess the role of C-reactive protein (CRP) and complement component C3 in women with polycystic ovary syndrome (PCOS) and to examine factors contributing to its pathogenesis.

2. METHODOLOGY:

STUDY POPULATION: The present investigation, titled "A Correlative Study of CRP and Complement Component C3 in Women with Polycystic Ovary Syndrome," was conducted over a two-year period, from January 1, 2023, to December 31, 2024. The study enrolled a total of 260 participants, categorized into two groups: cases and controls. Of these, 130 women were clinically diagnosed with PCOS, while the remaining 130 were healthy controls with no history of the disorder. Participants, aged 15 to 45 years, were recruited from the outpatient department of Obstetrics and Gynecology at SKS Hospital, Medical College, and Research Center, Mathura, Uttar Pradesh. The study followed a prospectively designed, hospital-based, case–control methodology.

Inclusion criteria: All patients who had a history of irregular periods (oligomenorrhea) and/or not ovulating (anovulation)were checked for signs of high androgen levels. This included both physical signs like excessive hair growth (hirsutism with a Ferriman-Gallwey score of 8 or higher) and blood test results showing high testosterone levels (2. 5nmol/Lor more) or high DHEAS levels (8. 5µmol/Lor more). These patients were also examined with an ultrasound to check for polycystic ovaries and ruled out other possible causes such as congenital adrenal hyperplasia, androgen-producing tumour, or Cushing's syndrome.

Exclusion criteria: Patients who had a history of connective tissue diseases, immune disorders, heart muscle disease, cancer, inherited high cholesterol, long term liver or kidney problems, or were under- going chemotherapy were not included. Women who were taking oral contraceptives or corticosteroids were included. However, women with a history of high prolactin levels, type 2 diabetes, type 1 diabetes, late-onset adrenal disorders, thyroid issues, Cushing's syndrome, or those on medicines that change hormone or chemical levels in the body were not part of this study.

Ethical consideration- The study was initiated only after receiving approval from the Institutional Ethics Committee, and all participant information was maintained with strict confidentiality.

Collection of blood samples: Approximately 5 mL of overnight fasting venous blood was obtained from each participant, with samples collected in plain vials for the evaluation of serum biochemical parameters and in citrated vials for the measurement of CRP and complement component C3 levels.

Estimation of C3, CRP, and lipid profile-It was done by collecting fasting blood sample and auto analyser machine (Erba EM 200) is used to perform the test based upon the spectro photometric principle.

Statistical Analysis: Statistical analysis was performed using SPSS software version 20 (IBM Corp., Chicago, IL, USA). Quantitative data were expressed as mean ± standard deviation (SD) and analyzed using analysis of variance (ANOVA, F-test) where appropriate. Demographic variables and laboratory findings were compiled in a master chart, systematically calculated, and subjected to statistical evaluation.The qualitative results were provided as numbers, percentages and contracted, if possible, with the Chi-square (X2) method. The value of P was considered statistically significant, when it came <0.05.

3. STUDY DESIGN

This was hospital based cross-sectional prospective study carried out among non-duplicate confirm PCOS patients along with healthy controls, conducted in the Department of Obstetrics & Gynecology,Index Medical College Hospital, Indore. The different laboratory and clinical parameters were analysed and tabulated as per the master chart. Laboratory and clinical parameters were systematically analyzed and recorded in a master chart. Results were presented as mean ± standard deviation (SD), with statistical significance reported where applicable.

Age wise distribution among the study subject:

The mean ± SD age of participants was 25.19 ± 3.54 years in the case group and 27.49 ± 5.16 years in the control group (Table 1). The majority of patients (46.1%) were within the 26–30-year age range.

Table1 Age wise distribution among the study subject

Age group	Case	Control	Total	c2(p-value)
15-20	12(9.2%)	11 (8.5%)	23	
21-25	53(40.8%)	38 (29.2%)	91	

26-30	60(46.1%)	46(35.4%)	106	27.12 (P< 0.0001)
31-35	4(3.1%)	22 (16.9%)	26	
>35	1(0.8%)	13(10%)	14	
Total	130(100%)	130 (100.0%)	260	
Mean±-std	25.19±3.54	27.49±5.158		

Marital status of the study subjects:

In our study, out of the 130 cases, 119 (86.9%) were married and 11(13.1%) patients were unmarried. Similarly, among control group (130), 107(82.3%) married and 23(17.7%) were unmarried.

BMI of the study subjects:

The mean±SD of body mass index below 20 kg/m2 was 33.98±10.71 and 22.17±1.87 and above 20kg/m2 was 32.95±8.3 and 22.98±2.51in case and control respectively, and this difference was statistically significant (P<0.0001).

Lipid parameters of study subjects:

Comparison of lipid profile parameters among study participants (Table 2) revealed a statistically significant difference (P < 0.001) in serum total cholesterol, triglycerides, high-density lipoprotein (HDL), and the total cholesterol/HDL ratio between cases and controls. The mean ± SD levels of total cholesterol were 188.42 ± 31.13 mg/dL in cases and 155.42 ± 26.33 mg/dL in controls, while triglyceride levels were 134.43 ± 50.01 mg/dL and 110.00 ± 42.19 mg/dL, respectively. The mean ± SD HDL concentration was 36.29 ± 9.58 mg/dL in cases compared with 41.22 ± 10.91 mg/dL in controls. The total cholesterol/HDL ratio was 5.54 ± 1.87 in cases and 4.08 ± 1.39 in controls (Table 2).

Table2 Lipid parameters of study subjects:

Lipid parameters	Cases	Controls	t-test	P value
Total cholesterol mg/dl	188.42±31.126	155.42±26.333	-9.22	<0.0001
Triglyceride mg/dl	134.43±50.01	110.00±42.19	-4.257	<0.0001
High density lipoprotein mg/dl	36.29±9.55	41.22±10.912	3.876	<0.0001
Total cholesterol /HDL Ratio	5.54±1.865	4.08±1.39	-7.157	<0.0001

Complement component 3 & CRP of the study subjects:

The mean ± SD values of serum complement component C3 and C-reactive protein (CRP), both markers of inflammation, were compared between cases and controls. Serum C3 levels were 160.66 ± 29.16 mg/dL in cases and 127.48 ± 35.60 mg/dL in controls, while CRP levels were 2.41 ± 0.94 mg/L and 2.25 ± 0.83 mg/L, respectively. A statistically significant difference was observed in serum C3 concentrations, with PCOS patients exhibiting markedly elevated C3 levels, as summarized in Table 3.

Table 3: Complement component 3 & CRP of the study subjects:

Paramete r	**Case**	**Control**	**t-test**	**P value**
serum C3	160.66±29.155	127.48±35.60	-8.221	P < 0.0001

CRP	2.41±0.94	2.25±0.83	-1.455	P = 0.1469

Hirsutism in the study subjects:

Hirsutism, an indicator of hyperandrogenism, was present in 83.1 % of clients. There was a significant difference (P<0.0001) between cases and controls in regards to hirsutism as shown in table 4.

Table 4: Hirsutism in the study subjects:

Hirsutism	Cases	Controls	Total	P-value
Absent	22(16.9%)	130(100.0%)	152(58.5%)	<0.0001
Present	108(83.1%)	0(0.0%)	108(41.5%)	
Total	**130(100.0%)**	**130(100.0%)**	**260(100%)**	

Pearson's correlation between various clinical parameters with the C3:

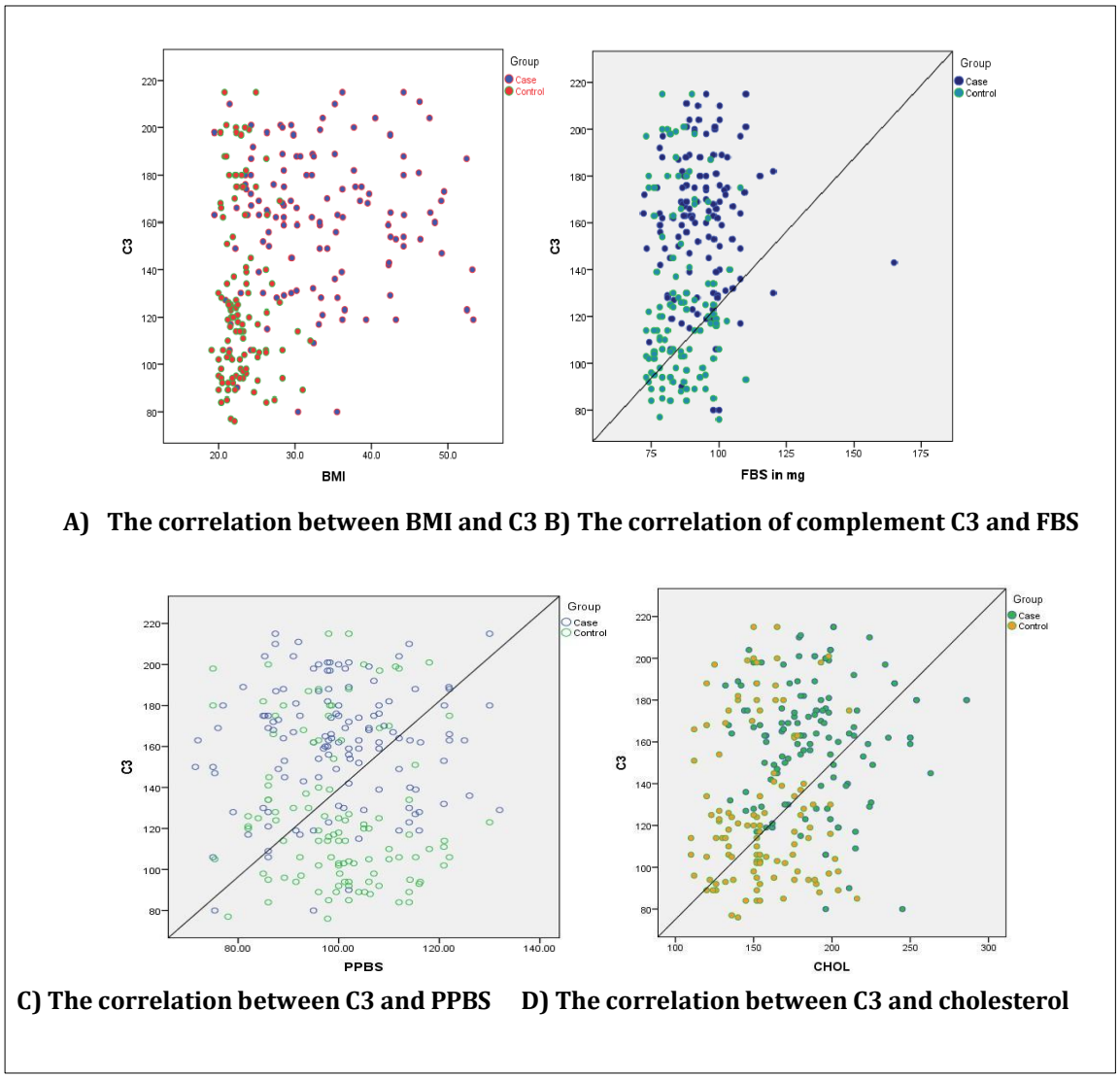
C3 shows positive correlation with FBS, total cholesterol, triglyceride, total cholesterol HDL ratio, BMI, systolic blood pressure (BP). There was a positive correlation between diastolic BP with C3 but the difference was statistically insignificant with p value 0.619.(Table 5).

High-density lipoprotein (HDL) levels demonstrated a negative correlation with serum C3, which was statistically significant (p = 0.001). In contrast, comparison of C3 levels with postprandial blood sugar (PPBS) yielded a p-value of 0.850, indicating no statistically significant association (Table 5; Figures 1A–D and 2).

Table 5: Pearson's correlation between various clinical parameters with the C3

Correlation of C3 with different clinical variables	**r value**	**P value**
Serum complement component 3vsBMI < 20kg/m2	0.069	0.773
Serum complement component 3vsBMI >20kg/m2	0.287	**<0.001**
Serum complement component 3 vs Age <25 Years	-0.115	0.224
Serum complement component 3 vs Age>25 Years	**-0.215**	**0.009**
Serum complement component 3vs fasting blood sugar	**0.188**	**0.002**
Serum complements component 3vs postprandial blood sugar	-0.012	0.850
Serum complement component 3vs Total Cholesterol	**0.238**	**<0.001**
Serum complement component 3vs Triacylglycerol	**0.177**	**0.004**
Serum complements component 3vs high density lipoprotein	**-0.253**	**<0.001**
Serum complement component 3vs Cholesterol/high density lipoprotein ratio	**0.276**	**<0.001**
Serum complement component 3vs Systolic BP	**0.267**	**<0.001**
Serum complement component 3vs Diastolic BP	0.031	0.619

Figure 1: The correlation between C3 with various clinical parameters (A,B,C,D)



A) The correlation between BMI and C3 B) The correlation of complement C3 and FBS

C) The correlation between C3 and PPBS D) The correlation between C3 and cholesterol

Pearson's correlation between various clinical parameters with CRP

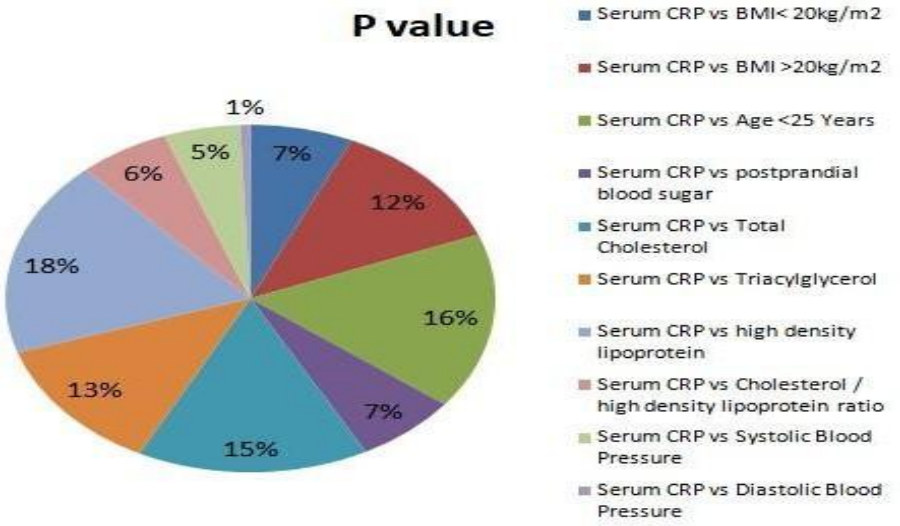
CRP showed a variable correlation with all clinical parameters. In case of FBS and HDL it was positively correlated but statically non-significant and HDL/total cholesterol ratio and PPBS was negatively correlated with r value -0.074 and r-value was -0.07, but it was statistically non-significant (Table 6) (Figure 2).

Table 6 Pearson's correlation between various clinical parameters with CRP

Correlation between serum CRP with different clinical variables	r value	P value
Serum CRP *vs* BMI< 20kg/m2	0.264	0.262
Serum CRP *vs* BMI >20kg/m2	0.047	0.473
Serum CRP *vs* Age <25 Years	-0.045	0.634
Serum CRP *vs* Age >25 Years	-0.130	0.118
Serum CRP *vs* fasting blood sugar	0.001	0.983

Serum CRP *vs* postprandial blood sugar	-0.07	0.262
Serum CRP *vs* Total Cholesterol	-0.033	0.591
Serum CRP *vs* Triacylglycerol	0.044	0.482
Serum CRP *vs* high density lipoprotein	0.023	0.706
Serum CRP *vs* Cholesterol / high density lipoprotein ratio	-0.074	0.235
Serum CRP *vs* Systolic Blood Pressure	0.081	0.194
Serum CRP *vs* Diastolic Blood Pressure	0.138	0.026

Figure 2 Pearson's correlation between various clinical parameters with CRP



4. DISCUSSION

Polycystic ovary syndrome (PCOS) is a disorder closely associated with metabolic syndrome features, including insulin resistance, central obesity, and cardiovascular disease, and is often accompanied by alterations in inflammatory markers such as C-reactive protein (CRP), complement component 3 (C3), interleukin-6 (IL-6), tumor necrosis factor (TNF), as well as dyslipidemia.[10,11] We hypothesized that PCOS represents a state of low-grade chronic inflammation, which may activate the immune response and lead to elevated levels of inflammatory mediators, including C-reactive protein (CRP) and complement component 3 (C3).

In this observational study, we have assessed the role of CRP and C3 in newly diagnosed PCOS and healthy women. In this present study approximately half of the number of the patients belongs to the age group between 26 to 30 (46.1%) which is significant and similar with the study conducted by Ramanand et al. which indicated that the prevalence of PCOS increased with age and is peaked up in the early and mid-twenties of life. [2]

In our study, marital status was also considered out of the 130 positive PCOS cases, 119 (91.53%) were married and 11(8.47%) clients were unmarried. Similarly, out of 130 controls 107 (82.30%) were married followed by 23 (17.70%) unmarried. This study was similar to the study conduct by the Shanet al. this study includes that 10 (1.7%) unmarried and 570 (98.3%) married women.[12] The mean ± SD body mass index (BMI) was 32.97 ± 8.47 in women with PCOS and 22.87 ± 2.47 in controls. The mean BMI was significantly higher in the PCOS group compared to controls ($P < 0.001$). These findings are consistent with the study by Dehdashtihaghighat et al., which reported a strong association between elevated BMI and PCOS, suggesting that this relationship likely results from complex interactions between genetic predisposition, lifestyle, and environmental factors..[13] In this study, participants were categorized as obese or non-obese using a BMI cutoff of ≥20 kg/m² to define obesity. Among the 260 subjects, 240 (92.3%) were classified as obese, while 20 (7.7%) were non-obese. The high prevalence of obesity in this cohort underscores an elevated risk for complications such as insulin resistance, metabolic syndrome, and type 2 diabetes mellitus (T2DM).[3,5] Our findings also demonstrated that menstrual irregularities were more prevalent among obese PCOS patients compared to their non-obese counterparts, suggesting that obesity significantly contributes to menstrual dysfunction. Similarly, studies by Majumdar et al. and Wei et al. reported

that obese women with PCOS exhibit more pronounced hyperandrogenism and associated clinical manifestations, including hirsutism, menstrual disturbances, and anovulation, than non-obese women with PCOS.[14,15]It was observed that, the obesity as well low high-density lipoprotein (HDL) was more prevalent in obese PCOS women by many researchers.[1,2,16] The mean HDL level was significantly higher in healthy controls compared to women with PCOS, whereas triglyceride concentrations were elevated in the PCOS group relative to controls, a difference largely attributable to obesity in affected women. The decreased HDL levels in PCOS patients indicate a heightened risk of cardiovascular disease, metabolic syndrome, and type 2 diabetes mellitus (T2DM). These findings align with observations by Kiranmayee et al., who reported that the majority of women with PCOS exhibited dyslipidemia, with over 70% demonstrating lipid abnormalities such as reduced HDL, elevated triglycerides, and increased low-density lipoprotein (LDL) cholesterol. A significant positive correlation between triglyceride and cholesterol levels was also noted[17]. Halasawadekar et al. similarly reported that triglyceride levels and the total cholesterol/HDL ratio were significantly elevated in women with PCOS compared to healthy controls.[18]In our study, the mean ± SD serum C3 levels in PCOS cases and controls were 160.66 ± 29.16 and 127.48 ± 35.60 mg/dL, respectively, demonstrating a statistically significant elevation of C3 in women with PCOS. This finding contrasts with the study by Wu et al., who reported higher C3 levels in premenopausal women with PCOS compared to controls; however, their results did not reach statistical significance. [11] This variation may be attributed to certain limitations of our study, including a relatively small sample size and a restricted study population, which reduce the generalizability of the findings. Additionally, parameters such as visceral fat distribution and waist-to-hip ratio were not evaluated, which could have provided further insights into adiposity and insulin resistance in women with PCOS compared to controls. Pearson correlation analysis of serum complement component 3 (C3) with various clinical variables in PCOS patients demonstrated consistently higher associations compared to the prevalence rates of metabolic syndrome observed in the control group. These findings align with the study by Kelly et al. (2001), which similarly reported that the prevalence of metabolic syndrome in women with PCOS was comparable to that of control women approximately 30 years older.[19]The underlying mechanisms linking polycystic ovary syndrome (PCOS) with metabolic syndrome and cardiovascular morbidity and mortality remain incompletely elucidated. Emerging evidence increasingly highlights low-grade chronic inflammation as a potential contributor to the long-term complications associated with PCOS. [20,21]The mean level of C3 was greater in women with PCOS versus controls. The serum C3 levels of patients with PCOS were positively correlated with FBS, cholesterol, triglyceride, CHO/HDL ratio, systolic BP, diastolic BP and BMI (BMI <20kg/m2 and BMI>20kg/m2, and negatively correlated with the age (Age<25 year and >25 year), PPBS, HDL cholesterol, but this finding was quiet similar with the study conducted by an author Snyderet al., and their findings suggests that the C3 levels was positively correlated with BMI, fasting glucose, triglycerides, total cholesterol, HDL-C and LDL-C in women with PCOS and controls (p<0.05). [22]The mean CRP in the PCOS group was 2.41 mg/dl, and in control group was 2.25 mg/dl, showed non- significant difference between these two groups. Serum CRP levels of patients with PCOS were positively correlated with BMI (BMI< 20kg/m2 and BMI>20kg/m2, pathological investigations and negatively correlated with the insulin sensitivity index, which was low as compared to the study conducted by Boulman et al., found high CRP levels in some PCOS .women.[23] Pearson correlation analysis showed that, both C3 and CRP demonstrate that the C3 level had a stronger association with PCOS as supported with the study conducted by Yang et al.. [7]When the level of complement 3 and CRP was compared in patients with hirsutism, the patients with hirsutism showed statistically significant increase in C3 levels and CRP. Similar study conducts by Adams et, al. this study finds, maximum number of women with hirsutism and PCOS, and possibly also idiopathic hirsutism, a large proportion of whom (>90%) have PCOS.[24] Thus, the complement component 3 levels and CRP were significantly correlated with clinical severity of polycystic ovarian syndrome. Hence, present study demonstrates that the level of inflammation is higher in women with PCOS with an ovulation and infertility, one of the major reproductive inabilities in women with PCOS shows significant increase in chronic low-grade inflammation. Although exact mechanisms of PCOS are not fully understood yet, there are numerous studies that underline pathological impact of obesity and insulin resistance in increased inflammation, suggesting or modulating influence of these states on PCOS pathogenesis. There are also studies indicating a strong relationship between higher concentration of androgen and white blood cells count, so that chronic low-grade inflammation may be mediated not only through adiposity, but also through androgen concentration.

5. CONCLUSION:

In the present study, the role of inflammation in PCOS was considered. It was found that a majority of the PCOS patients were obese and insulin resistant in our study. The levels of CRP and C3, marker of chronic low-grade inflammation, were higher in freshly diagnosed patients as compared to the controls. The complement component 3 values correlated well with increased in BMI and age. This could probably imply a state of insulin resistance, low grade inflammation, abdominal obesity and activation of the complement cascade in a majority of women with PCOS. Thus, in the current study, the state of low-grade inflammation seen in cases of PCOS could strongly influence their risk for adverse polycystic ovarian syndrome and metabolic complications. Further studies are needed to establish relationships, especially the association of new polymorphisms of genes which play significant roles in the pathophysiologic inflammatory mechanism in PCOS.

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Authors' contributions:

Dr. Indra Prasad Adhikari has designed the study, conducted various experiments, and written the manuscript. Dr. Devendra Sharma was responsible for patient recruitment and experiment guidance. Dr Sandeep Kumar Sharma is the research head and has contributed to manuscript preparation, editing the journal article. Dr. Diwakar dutta Tripathi & Ms. Sanghapriya Mukherjee has contributed to the writing of the journal article

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