RESEARCH PAPER

Evaluation of serum Lipid profile, Lipid ratios, Insulin Resistance, and their relationship in South Indian women with Polycystic Ovary Syndrome

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Abstract

Background: Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder in women of reproductive age, is often associated with insulin resistance, obesity, hyperandrogenism, oligo/anovulation and dyslipidaemia. Dyslipidaemia is a well-known risk factor for cardiovascular diseases in affected women. However, the patterns of lipid abnormalities in women with PCOS can vary based on ethnicity, diet, and lifestyle factors.

Objective: This study aimed to evaluate the serum lipid profile, lipid ratios, and insulin resistance among South Indian women with PCOS and to explore their interrelationship.

Materials and Methods: This case-control study included 104 women with PCOS (aged 20–35 years) and 95 age-matched healthy controls. Anthropometric parameters and biochemical parameters including fasting glucose, insulin, lipid profile, and hormonal parameters were measured by standard methods in both groups. Insulin resistance was assessed by Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Lipid ratios including TC/HDL-C, TG/HDL-C, and LDL-C/HDL-C, non-HDL-C were calculated. Statistical analysis was performed using Student's t-test and Pearson's correlation.

Result: Our study revealed that women with PCOS had significantly higher BMI, wait-hip ratio, fasting glucose, insulin, HOMA-IR, total cholesterol, triglycerides, and LDL- C along with lower levels of HDL-cholesterol compared to control groups ($p < 0.01$). Atherogenic index like non-HDL C, TC/HDL-C, TG / HDL-C and LDL-C/HDL-C were also significantly increased in the PCOS patients than in the control subjects ($p<0.01$). The Insulin Resistance was positively correlated with total cholesterol ($r=0.775$; $p<0.01$), triglycerides ($r=0.790$; $p=<0.01$), LDL-C ($r=0.790$; $p<0.01$), non-HDL-C ($r=0.679$; $p<0.01$), TC/HDL-C ($r=0.720$; $p<0.01$), TG/HDL-C ($r=0.679$; $p<0.01$), and LDL-C/HDL-C ($r=0.269$; $p<0.01$) and negatively correlated with HDL-C ($r = -0.826$; $p<0.01$).

Conclusion: South Indian women with PCOS exhibited a higher prevalence of insulin resistance and dyslipidemia, indicating predisposition to early cardiovascular complications. Therefore, they need early metabolic screening and preventive measurement to prevent long-term cardiovascular risks.

Keywords: PCOS, Insulin resistance, Lipid profile, Lipid ratios, South Indian Women

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Introduction

Polycystic ovary syndrome (PCOS) is most common endocrine disorder in women of reproductive age, with a worldwide prevalence ranging from 6 to 22%, depending on the diagnostic criteria [1,2]. PCOS is a heterogeneous disease characterized by hyperandrogenism, dysfunctional ovulation, and polycystic ovary morphology, accompanied by metabolic abnormalities, such as insulin resistance (IR) and obesity. The aetiology of PCOS remains unknown however; both genetic and environmental factors contribute the PCOS pathogenesis. IR and compensatory hyperinsulinemia (HI) are considered major drivers of PCOS pathophysiology and are involved in the development of hyperandrogenaemia and reproductive dysfunction by various mechanism [3]. These factors are associated with an increased risk of type-2 diabetes mellitus, dyslipidaemia, cardiovascular disease (CVD), non-alcoholic fatty liver, premature arteriosclerosis, endometrial hyperplasia, chronic low-grade inflammation, and thrombosis, collectively constituting metabolic syndrome [4 ,5,6]

Approximately 50%-70% of women with PCOS also have IR, and the risk of type 2 diabetes (T2DM) among women with PCOS. In women with PCOS, abnormal lipid levels are seen with a prevalence of 70% [7]. Due to high prevalence of insulin resistance and hyperinsulinemia in PCOS, dyslipidaemia in women with PCOS may therefore be consistent with that found in the insulin resistant state. Insulin resistance and

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dyslipidaemia play a key role in the risk of cardiovascular pathology in women with PCOS. The extent to which dyslipidaemia leads to this risk is still not well understood [8].

Dyslipidaemia is the most common metabolic disorder in women with PCOS, although in different geographic regions and ethnic groups has been reported different. Abnormal levels of lipoproteins are common in this syndrome, with increased level of total cholesterol, triglycerides, LDL (low-density lipoprotein), reduced high-density lipoprotein (HDL) [9,10].Most studies reported that decreased HDL-C and increased triglyceride levels in PCOS, which is the same lipid profile known to be associated with IR [11].A study found that the proportion of IR diagnoses was statistically higher in obese women than in women with normal BMI, suggesting a significant association between IR and dyslipidaemia [12]. In another study found that the prevalence of impaired glucose tolerance (IGT) was significantly higher in women with PCOS who had a high TG/HDL-C ratio than in those with a low TG/HDL-C ratio [13]. Lipoprotein ratios, particularly TG/HDL-C ratio and TC/HDL-C ratios are directly correlated with insulin levels and can be used as a marker of IR (HOMA-IR) in infertile PCOS patients directly correlated with insulin levels associated with IR (HOMA-IR). [14,15]. Various studies have demonstrated that abnormal lipid profile patterns in women with PCOS, and these abnormalities were not uniform in all populations. [16,17].

South Indian women, due to unique genetic, dietary, and lifestyle factors, may show different patterns of lipid profile abnormalities. Understanding these patterns in women with PCOD is critical for early intervention and management strategies aimed at reducing long-term cardiovascular risks in this population. Therefore, the study was undertaken to evaluate the lipid profile status, lipid ratios, insulin resistance in South Indian women with PCOS and to see whether there exists any association between lipid profile and lipid ratios with insulin resistance.

Materials and Methods:

The case-control study included 104 women with PCOS (aged 20–35 years) controls were recruited from outpatient department of Obstetrics and Gynecology, Jawaharlal Institute of Post Graduate Medical Education and Research (JIPMER) Puducherry, India. The control group consisted 95 healthy volunteer females with regular menstrual cycles aged between 20 to 35 years. The diagnosis of PCOS was made according to the Rotterdam consensus criteria [18]. It is defined as patient was considered to have PCOS if she fulfilled two out of the following three criteria: oligo- and/or anovulation, clinical and/or biochemical signs of hyperandrogenism and polycystic ovaries on ultrasonography scan. The study was approved by Institute Research Council Board and followed by Human Ethical Committee, JIPMER, and Puducherry, India (No. Edn.6(10)/2007 and Reg.No.1). The written informed consent was obtained from patients and controls.

Patients with diabetes mellitus, thyroid dysfunctions, Cushing's syndrome, congenital adrenal hyperplasia, hyperprolactinemia, androgen secreting tumor, renal and liver dysfunction were excluded from the study by specific laboratory tests. Subjects with medication like ovulation induction agents, antiandrogens, antidiabetic, anti-obesity, hormonal drugs and current or previous use of OC within last 6 months, smoking and alcohol intake were also excluded from the study.

All 104 PCOS patients and 95 healthy control underwent full physical examination and anthropometric measurements including weight, height and, waist and hip circumferences. Weight was measured with the subjects wearing light clothing without shoes, and height was measured using a stadiometer. Body Mass Index (BMI) was calculated by using the formula: weight (Kg)/height (meters). Waist circumferences (WC) were measured with the patients standing at a point mid-way between lower costal margin, and ileac crest in the mid-auxiliary line. Hip circumferences were measured at the widest point over the buttocks with the subjects standing and breathing normally. The waist to hip ratio (WHR) were calculated for all subjects = waist circumference/hip circumference

After overnight fasting venous blood sample was obtained between 08.00 am and 08.30 am, on the 2nd day of spontaneous progesterone (methoxy progesterone acetate 10mg/day for 7 days) induced withdrawal bleeding for (to carry out the study during the follicular phase we had to induce the menstruation in PCOS women because they were having irregular menstrual cycles) estimation of hormones, IR marker indices, and lipid profile.

The plasma glucose was determined by glucose oxidase-peroxidase, (GOD-POD) end point method using a commercial kit (Agape diagnostic, India) using clinical chemistry Auto analyzer (Beckman Coulter AU680, Japan). Plasma Insulin was (Bio line, Belgium) estimated by ELISA techniques (Lab system-Multiskan Ascent, Finland)

IR was determined by Homeostasis Model Assessment for insulin resistance (HOMA - IR) = Fasting glucose (mg / dl) x fasting insulin (muIU / ml) / 405[19]. QUICK was calculated as=1/ (log (fasting insulin) +log (fasting glucose) [20]

Serum LH, FSH, were determined by Chemi Luminescent Immunoassay (CLIA) method using Siemen’s Advia Centaur CP kit by Siemens Advia Centaur CP analyzer, Japan. The Testosterone, Androstenedione, Progesterone, Estradiol, DHEAS, SHBG, 17-hydroxy progesterone (17-OHP) were analyzed by competitive immunoassay by Chemi Luminescent Immunoassay method using Siemen’s Advia Centaur CP kit by Siemens Advia Centaur CP analyzer, Japan.

Total cholesterol, Triglycerides were measured by cholesterol esterase-oxidase end point and glycosol-3-phosphate oxidase-peroxidase, endpoint method

respectively by using commercial kits (Agape diagnostic, India) by autoanalyzer (Beckman Coulter AU680, Japan). HDL-cholesterol was estimated by direct immune-inhibition method using commercial kits (Agape diagnostic, India) using autoanalyzer (Beckman Coulter AU680, Japan). VLDL-cholesterol was calculated by dividing the total cholesterol concentration by 5. LDL-cholesterol was calculated using Friedwald's formula [21]

LDL-Cholesterol = Total Cholesterol (mg/dl) – [HDL-C + Triglycerides]

5Non-HDL-cholesterol was calculated by subtracting HDL-cholesterol from total cholesterol. Atherogenic indices as indicated by various risk ratio were calculated using the following calculations: TC (mg/dL)/HDL-C (mg/ dL), TG (mg/dL)/HDL-C (mg/dL), LDL-C (mg/dl)/HDL-C(mg/dl), and non-HDL-C(mg/dl) /HDL-C(mg/dl)

Statistical Analysis:

All the statistical analysis were carried out the Statistical Package for the Social Sciences (SPSS) version 26.0, for Windows (SPSS, Inc., Chicago). All the quantitative data were expressed as mean ± standard deviation (SD). The unpaired Student's t–test was used to compare the values of PCOS vs Controls. Pearson's correlation coefficient test was used to assess the association between HOMA-IR and lipid parameters in PCOS patients. Statistical significance was considered as $P < 0.05$.

Result

Table-1 Shows anthropometric parameters in control groups and PCOS groups. In this study all women were in the age group of 25-35 years for both cases and controls. The BMI was found to be more in PCOS patients and statistically significant when compared to the control subjects ($p<0.01$). The waist to hip ratio was more in PCOS cases and statistically significant when compared to the control subjects ($p<0.05$)

Table 1: Anthropometrics parameters in control and PCOS Cases.

Variables	Control (n = 95)	PCOD (n = 104)
Age (years)	27±4	27.33 ± 3.30 NS
Weight (Kg)	57±12	68 ± 4**
Height (Cm)	1.59±0.05	1.58 ± 3.38 NS
BMI (Kg / m^2)	22.08±1.75	27.39 ± 1.45**
Waist circumference (Cm)	75±4	89 ± 3*
Hip circumference (Cm)	91±5	104 ± 4*
Waist-hip ratio	0.82±0.02	0.85 ± 0.01*

Values on shown in mean ± standard deviation
* $p < 0.05$ and ** $p < 0.01$ compared to controls
NS=Not significant

The hormonal data of women with PCOS and controls are shown in Table 2. The hormones such as LH, total Testosterone androstenedione was significantly higher in women with PCOS than controls ($p < 0.01$) and similarly, LH/FSH ratio and progesterone, 17 OHP, DHEAS were also significantly higher in women with PCOS than healthy controls groups ($p < 0.05$). On the other hand, patients with PCOS had significantly lower levels of FSH, estradiol and SHBG than control groups ($P < 0.05$).

Table-2 Hormonal profiles in control and PCOS cases

Variables	Control (n = 95)	PCOD (n = 104)
LH (□IU/ml)	5.98±1.03	13.10±7.00**
FSH (□IU/ml)	5.74±1.16	4.61±1.85*
LH / FSH	1.05±0.11	2.83±0.76*
TT (ng/dl)	36.60±8.15	63.98±16.65**
A_4 (ng/dl)	1.47±0.45	3.64±0.87**
Progesterone (ng/dl)	0.45±0.40	0.61±0.39*
17 OHP (ng/dl)	0.53±0.02	0.84±0.18*
E_2 (pg./ml)	58.92±17.21	39.03±11.51*
SHBG (nmol/L)	62.39±8.35	42.98±11.44*
DHEAS (□g/dl)	173.12±44.72	262.64±72.33**

Values are shown in mean ± standard deviation and
* $p < 0.05$ and ** $p < 0.01$ compared to controls

Table 3: shows the insulin resistance indices in control and PCOS groups. The fasting plasma glucose was significantly increased in PCOS cases when compared to control groups ($p < 0.05$) The fasting plasma insulin and HOMA-IR were significantly increased in PCOS patients compared to control groups ($p < 0.01$) The

fasting plasma glucose and insulin ratio was significantly decreased in PCOS group than control subjects ($p < 0.01$) The QUICKI was significantly lowered in patients than in control subjects ($p < 0.05$)

Table-3: Insulin resistance parameters in women with PCOS and healthy controls.

Variables	Control (n = 95)	PCOS (n = 104)
Fasting glucose (mg/dl)	83.76±6.67	106.64±12.56*
Fasting Insulin (□IU/ml)	14.27±2.92	35.61±5.31**
Glucose / Insulin	6.20±1.79	3.01±0.33**
HOMA-IR	2.92±0.53	9.49±2.36**
QUICKI	0.33±0.09	0.28±0.09*

Values are shown in mean standard deviation
* $p < 0.05$ and
** $p < 0.01$ compared to controls

Table-4 Shows serum lipid profile and atherogenic index of the patients and control subjects. The total cholesterol, triglycerides, and LDL-cholesterol were significantly higher in PCOS patients when compared to the control group ($p<0.05$). Whereas HDL-cholesterol was significantly lower in women with PCOS than controls($p<0.05$). Atherogenic index like non-HDL cholesterol, TG / HDL-cholesterol, TC/HDL-cholesterol, and LDL-C/HDL-cholesterol were also significantly increased in the PCOS patients than in the control subjects ($p<0.05$).

Table 4: Lipid profile and atherogenic index in Control and PCOS cases

Variables	**Control (n = 95)**	**PCOS (n = 104)**
Total cholesterol (mg/dl)	156.67±14.78	181.66±30.30 *
Triglycerides (mg/dl)	108.68±20.78	139.41±35.41*
HDL-C (mg/dl)	52.80±6.72	34.42±7.69*
LDL-C (mg/dl)	82.13±11	119.36±21.27*
VLDL-C (mg/dl)	21.74±4.2	27.88±7.1 NS
Non HDL-C (mg/dl)	103.87±12.14	147.24±25.23*
TG / HDL-C	2.06±0.34	4.11±0.75*
TC / HDL-C	3.07±0.36	5.41±0.86*
LDL-C / HDL-C	1.56 ±0.33	3.60±0.81*

Values are shown in mean ± standard deviation
* $p < 0.05$ compared to controls
NS = Not significant

The correlation between insulin resistance and lipids risk factors and atherogenic index are shown in table -5. The IR was positively correlated with total cholesterol ($r=0.775$; $p<0.01$), triglycerides ($r=0.790$; $p=<0.01$), LDL-C ($r=0.542$; $p<0.01$), non-HDL-C ($r=0.679$; $p<0.01$), TC/HDL-C ($r=0.720$; $p<0.01$), TG/HDL-C ($r=0.679$; $p<0.01$), and LDL-C /HDL-C ($r=0.269$; $p<0.01$) and negatively correlated HDL-C ($r= -0.826$; $p<0.01$).

Table 5: Correlation between insulin resistance and lipid profile and atherogenic index in PCOS patients

Variables	**HOMA-IR** **r**
Total cholesterol (mg/dl)	0.775**
Triglycerides (mg/dl)	0.790**
HDL-C (mg/dl)	-0.826**
LDL-C (mg/dl)	0.790**
Non HDL-C (mg/dl)	0.679**
TG / HDL-C	0.679**
TC / HDL-C	0.720**
LDL-C / HDL-C	0.269**

** $p < 0.01$ compared to controls

Discussion

In women with Polycystic Ovary Syndrome (PCOS), there is a well-established connection between insulin resistance (IR) and dyslipidaemia. Insulin resistance, which affects around 70-80% of women with PCOS, plays a crucial role in driving lipid abnormalities. These abnormalities typically manifest as elevated total cholesterol, triglycerides, LDL-C, and reduced HDL-C [22]. This pattern of dyslipidaemia in PCOS contributes to an increased risk of cardiovascular diseases, including atherosclerosis, hypertension, and myocardial infarction as well as metabolic diseases due to the accumulation of lipids in blood vessels. The abnormal lipid profile in PCOS is primarily driven by insulin resistance, hyperandrogenism and obesity which are common in these patients. [23,24]

This study aimed to evaluate lipid profiles, lipid ratios, and their association with insulin resistance in South Indian women diagnosed with PCOS and assess their cardiovascular risk based on dyslipidemia. Our results revealed that women with PCOS had significantly higher levels of total cholesterol, triglycerides, and LDL-C, along with lower levels of HDL-C, compared to the control groups. Several studies have demonstrated similar patterns of dyslipidemia in women with PCOD, though the severity of lipid abnormalities may vary based on ethnicity and lifestyle factors [25,26,27,28,29,30,31].

Recent research highlights that PCOS patients typically experience more pronounced dyslipidemia, characterized by higher total cholesterol (TC), triglycerides (TG) and LDL-C, and lower HDL-C levels. [32,33]. A study published in 2023 reports that 41.3% of women with PCOS exhibit dyslipidaemia, with variations in lipid profile severity based on factors like insulin resistance, hyperandrogenism, and obesity [34]. On the other hand, our results are not concurrent with other studies that failed to demonstrate any significant differences in lipid and lipoprotein concentrations between women with PCOS and controls [35,36,37]. Similar results were observed in another study conducted by S. Bhardwaj et al (2023) who reported that no atherogenic alteration in the levels of HDL-C, LDL-C, and higher TG levels [38].

HDL-C, known as "good cholesterol," helps to clear cholesterol from the bloodstream and reduce the formation of atherosclerotic plaques. In this study, we observed women with PCOS demonstrated that lower levels of HDL-C compared to controls and negatively correlated with insulin resistance and suggests impaired reverse cholesterol transport mechanisms, contributing to an overall increase in cardiovascular risk. Our results are in accordance with recent studies by Rashidi .H et al (2018), Pritzer, P. et al (2021)., Guo F et al (2022), C. Gong Z et al (2022), Zhao, H. et al (2023) reported that higher total cholesterol (TC), triglycerides (TG) and LDL-C, and lower HDL-C levels [39,40,41,42,43].

Apart from lipid profile levels in PCOS patients, several studies suggested that lipid ratios also important strong predictor of insulin resistance and its associated complications across different races [44].Based on the background ,the present study aimed to evaluate the lipid profile ratios in South Indian women with PCOS.In our present study we found that lipid ratios such as TC/HDL-C,TG/HDL-C,Non- HDL-C and LDL-C/HDL-C were higher in PCOS patient than control groups.

The TC/HDL-C ratio is widely used to assess cardiovascular risk, with a higher ratio indicating atherogenic lipid profiles and insulin resistance. In PCOS patients, dyslipidemia is prevalent, and the TC/HDL-C ratio is increasingly being recognized as a marker of both insulin resistance and cardiovascular risk. Our result revealed that TC/HDL-C ratio was significantly higher in PCOS patients than control groups. Similar results were obtained in other studies suggested that TC/HDL-C ratio had strong correlation with IR in women with PCOS [45)]. Insulin resistance affects lipid metabolism by increasing the hepatic production of cholesterol, which raises total cholesterol levels. A reduced ability to clear cholesterol from tissues leads to a decrease in HDL levels, thereby raising the TC/HDL-C ratio. Recently, it has been shown that TC/HDL-C and TG/HDL-C were significantly increase among PCOS patients than control groups and positively correlated with HOMA-IR [46],

The TG/HDL ratio has been extensively studied in recent years as a marker of insulin resistance in PCOS. Elevated triglycerides and low HDL levels, common in insulin-resistant individuals, result in a higher TG/HDL-C ratio, which has been shown to correlate with metabolic disturbances, including insulin resistance and cardiovascular risk. In our study, we observed, TG/HDL-C, was significantly increased in PCOS patients than age-matched control and TG/HDL-C ratio above 3.5 were more likely to have significant insulin resistance and high risk of for cardiovascular events over time and positively correlated with insulin resistance and our results were consistent with other studies [47,48]. Insulin resistance in PCOS leads to increased lipolysis and a surge in free fatty acids. These free fatty acids are converted into triglycerides in the liver, elevating circulating triglyceride levels. Concurrently, insulin resistance lowers HDL levels, which reduces HDL-mediated cholesterol efflux and impairs reverse cholesterol transport. Together, these processes increase the TG/HDL ratio, making it a sensitive marker for metabolic disturbances [49].

Non-HDL-C, which includes, all atherogenic lipoproteins (LDL-C, VLDL-C, and IDL-C) and high levels being used to assess cardiovascular risk, particularly in insulin-resistant individuals, because it represents all cholesterol that can potentially lead to plaque formation in arteries. In our study, we observed atherogenic index like non-HDL-C, significantly increased in PCOS patients than age-matched control and positively correlated with IR. Similar study was carried out by Samuel et al. (2021) demonstrated that non-HDL-C is a better predictor of cardiovascular events in women with PCOD compared to LDL-C alone and study found that non-HDL-C levels were

significantly higher in women with insulin resistance [50]. In another study showed that non-HDL-C levels was positively correlated with HOMA-IR, and higher non-HDL-C levels were associated with increased carotid artery intima-media thickness (IMT), an early marker of atherosclerosis, in women with PCOS [51,52]. The LDL/HDL-C ratio is commonly used as a marker for atherogenic risk and increased ratio has also been linked to insulin resistance, particularly in women with PCOS [53]. In our study, we observed LDL-C/HDL-C ratio was significantly increased in PCOS patients than age-matched control and positively correlated with IR. The LDC-C / HDL-C ratio greater than 3 is risk factor for cardiovascular disease. In our study, LDL-C / HDL-C ratio was found to be 3.6. Our results were agreed with following studies. Christakou et al. (2021) and Garg et al. (2022) reported that a strong correlation between the LDL/HDL-C ratio and insulin resistance in women with PCOS and suggested that women with a high LDL/HDL-C ratio was a more reliable predictor of cardiovascular disease and higher risk of developing type 2 diabetes due to the concurrent elevation in insulin resistance [54 ,55].

Conclusion

The lipid abnormalities i.e. lipid profile and lipid ratios observed in South Indian women with PCOS including higher total cholesterol, triglycerides, LDL-C, and lower HDL-C- reflect an increased risk of both cardiovascular and metabolic diseases due to insulin resistance. Lipid ratios particularly TC /HDL-C, TG/HDL-C, Non-HDL-C and LDL-C/HDL-C are closely associated with IR. Insulin resistance, hyperandrogenism, and obesity are key drivers of dyslipidemia in PCOS. Lipid ratios as reliable markers for assessing insulin resistance and cardiovascular risk in women with PCOS and identifying women at higher metabolic risk. Therefore, PCOS patients need for regular monitoring of lipid profiles and suggests that early intervention through lifestyle changes, insulin-sensitizing agents, or lipid-lowering medications (such as statins) could help mitigate long-term health risks.

Conflicts of interest

The authors have no conflicts of interest associated with the material presented in this paper.

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