

Serum lipid profile of polycystic ovary syndrome in Sudanese women

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Abstract

Background: Polycystic ovarian syndrome (PCOS) has been one of the major public health problems in Sudan, which leads to medical consequences. It is caused by several factors such as menstrual dysfunction, hyperandrogenism, hirsutism, dyslipidemia, and obesity, which elevate the risk of diabetes mellitus and cardiovascular disease.

Objective: To know the lipid profile variation in Sudanese women with PCOS.

Materials and Methods: This descriptive, analytic, cross-sectional, and hospital-based study was conducted in Khartoum state, Sudan; it was carried out from March 2013 to May 2014. The subjects enrolled for the study included 200 women with PCOS who were compared with 100 healthy women of the control group; all of them were age and weight matched. Samples were taken after overnight fasting, and then serum lipid levels were analyzed using enzymatic colorimetric methods. Data management and analysis were done by using Microsoft Excel and SPSS software, version 20.00.

Result: The mean age of patients with PCOS was 29.61 ± 5.4 years, and the mean age of subjects in the control group was 31.23 ± 4.93 years. Lipid profile parameters in PCOS group were as follows: the (mean \pm SD) of serum triglyceride, total cholesterol, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) were 89.89 ± 33.36 mg/dL, 166.95 ± 46.94 mg/dL, 43.83 ± 20.75 mg/dL, and 104.55 ± 52.209 mg/dL, respectively, while those of control group were 82.13 ± 26.51 mg/dL, 145.14 ± 31.019 mg/dL, 51.02 ± 14.81 mg/dL, and 72.62 ± 30.04 mg/dL, respectively. There were statistically significant increased levels of triglyceride, total cholesterol, and LDL cholesterol (LDL-C) in PCOS group when compared with the control group ($P < 0.05$) and decreased level of HDL cholesterol (HDL-C).

Conclusion: The altered lipid profile in PCOS patients showed a sign of cardiovascular disease.

KEY WORDS: Polycystic ovary syndrome, triglyceride, total cholesterol, HDL-C, LDL-C

Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine disease and metabolic disorder in adolescent and reproductive women, which is the predominant reason for female infertility, with the incidence in 5%–10% of women of

reproductive age.^[1,2] Chronic anovulation, hyperandrogenism, and ovarian polycystic changes observed by ultrasound examination in clinic are the conditions, which confirm the prevalence of PCOS. Although the causative factors of PCOS are not well known, previous studies have shown the close association of PCOS with lipid metabolism disorder and insulin resistance.^[3,4] Over 50% of patients with PCOS showed obesity (android-type), which indicated an elevated risk of diabetes mellitus and cardiovascular disease (CVD).^[5] Approximately 40%–50% of patients, especially obese women, exhibit insulin resistance.^[6] The PCOS that entitles a unique attention was first described in 1935 by Stein and Leventhal, and this condition is also known as polycentric ovaries, sclerocystic ovarian disease, Stein–Leventhal syndrome, chronic anovulatory syndrome, and polycystic ovarian disease (PCOD).^[7] Earlier, the clinical diagnosis was based on

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the trio of hirsutism, amenorrhea, and obesity. The diagnosis of PCOS is based on the Rotterdam criteria for the presence of any two of the following conditions: (i) chronic anovulation, (ii) clinical/biochemical parameters for hyperandrogenism, and (iii) polycystic ovaries on ultrasonography.^[6] Insulin resistance, hyperandrogenism, and dyslipidemia are presumed to be the major risk factors for CVD in women with PCOS.^[9,10] Insulin resistance and dyslipidemia allegedly play a key role on the risk of cardiovascular pathology in women with PCOS. The extent to which dyslipidemia leads to this risk is still not well understood.^[11] Dyslipidemia is the most common abnormality in PCOS,^[12] with elevated levels of total cholesterol, triglycerides (TGs), and low-density lipoprotein cholesterol (LDL-C) and with low levels of high-density lipoprotein cholesterol (HDL-C).^[5,13] Talbott *et al.*^[14] reported increased level of LDL-C in patients with PCOS, and Conway *et al.*^[15] reported that the most characteristic lipid alteration is decreased levels of HDL-C. There are few studies done to know the alteration in serum lipid profile in PCOS patients; thus, this study was done to know the lipid profile variation in women with PCOS.

Materials and Methods

This descriptive, analytic, cross-sectional, and hospital-based study was carried out in educational hospital of Khartoum state, Sudan, from March 2013 to May 2014; all patients selected showed a history of PCOS. Subjects enrolled for the study included 200 patients diagnosed with PCOS and 100 healthy women (control group) who showed no history of drugs affecting lipid metabolism (with normal menstrual cycle and sex hormone level and no evidence of polycystic ovary). A written informed consent was obtained from all the participants; all of them were age and weight matched. The blood samples were obtained to determine the levels of lipids parameters. The blood samples were drawn after overnight fasting in the morning (between 8 a.m. and 11 a.m.). Blood samples of 5 mL from each individual of the study population were collected from both cases and control. The blood samples were centrifuged at 3,000 rpm for 10 min and sera obtained; samples were stored in +4°C until analyzed during the same day. Enzymatic colorimetric methods were used to determine total cholesterol, TGs, HDL-C, and LDL-C levels. All these parameters were investigated and recorded, and then, a comparison was made between PCOS patients and control participants. Later, the results were collected and

presented in graphs and tables, showing the values as mean \pm standard deviation. Data were analyzed by SPSS, version 20 (IBM). Student *t*-test was used for the calculation. $P \leq 0.05$ was considered significant.

All the chemical reagents were purchased from Bio Systems company (Spine Company for analytical material and chemical reagents).

Result

A total of 200 patients with PCOS were included in the study with a mean age of 29.61 ± 5.41 years and 100 healthy women with a mean age of 31.23 ± 4.93 years. Other basic characteristics of the study participants such as body mass index (BMI) of the PCOS patients was statistically significantly increased when compared with control group [Table 1].

The mean \pm SD of the lipid profile parameters in PCOS group of serum TG, total cholesterol, HDL, and LDL were 89.89 ± 33.36 mg/dL, 166.95 ± 46.94 mg/dL, 43.83 ± 20.75 mg/dL, and 104.55 ± 52.209 mg/dL, respectively, while those of control group were 82.13 ± 26.51 mg/dL, 145.14 ± 31.019 mg/dL, 51.02 ± 14.81 mg/dL, and 72.62 ± 30.04 mg/dL, respectively [Table 2]. There were statistically significant increased levels of TG, total cholesterol, and LDL-C in PCOS group and decreased levels of HDL-C when compared with the control group ($P < 0.05$) [Table 2]. In our study, the correlations between BMI and all the parameters in the study were found.

Figure 1 shows no correlation between BMI and the serum levels of TGs ($r = 0.00$, $P = 0.001$). Figure 2 shows insignificant and very weak positive correlation between BMI and the serum levels of cholesterol ($r = 0.04$, $p = 0.55$).

Figure 3 shows insignificant, very weak positive correlation between BMI and the serum levels of HDL ($r = 0.01$, $P = 0.84$).

Figure 4 shows insignificant, very weak positive correlation between BMI and the serum levels of LDL ($r = 0.04$, $P = 0.56$).

Table 1: Mean \pm SD of baseline characteristics of patients with PCOS and control group

Variable	PCOS group	Control group	P
Age (years)	29.61 ± 5.41	31.23 ± 4.93	0.060
Weight (kg)	72.83 ± 10.88	68.03 ± 11.31	0.030
Height (cm)	160.00 ± 6.00	162.60 ± 5.52	0.210
Height (cm)	29.76 ± 4.24	24.14 ± 3.76	0.001

$P < 0.05$ is statistically significant.

Table 2: Mean \pm SD of lipid parameters in the PCOS group and control group

Parameters	PCOS group	Control group	P
Serum triglyceride (mg/dL)	89.89 ± 33.36	82.13 ± 26.51	0.037
Serum cholesterol (mg/dL)	166.95 ± 46.94	145.14 ± 31.19	0.001
Serum HDL-C (mg/dL)	43.83 ± 20.75	51.02 ± 14.81	0.001
Serum LDL-C (mg/dL)	104.55 ± 52.29	72.62 ± 30.04	0.001

$P < 0.05$ is statistically significant.

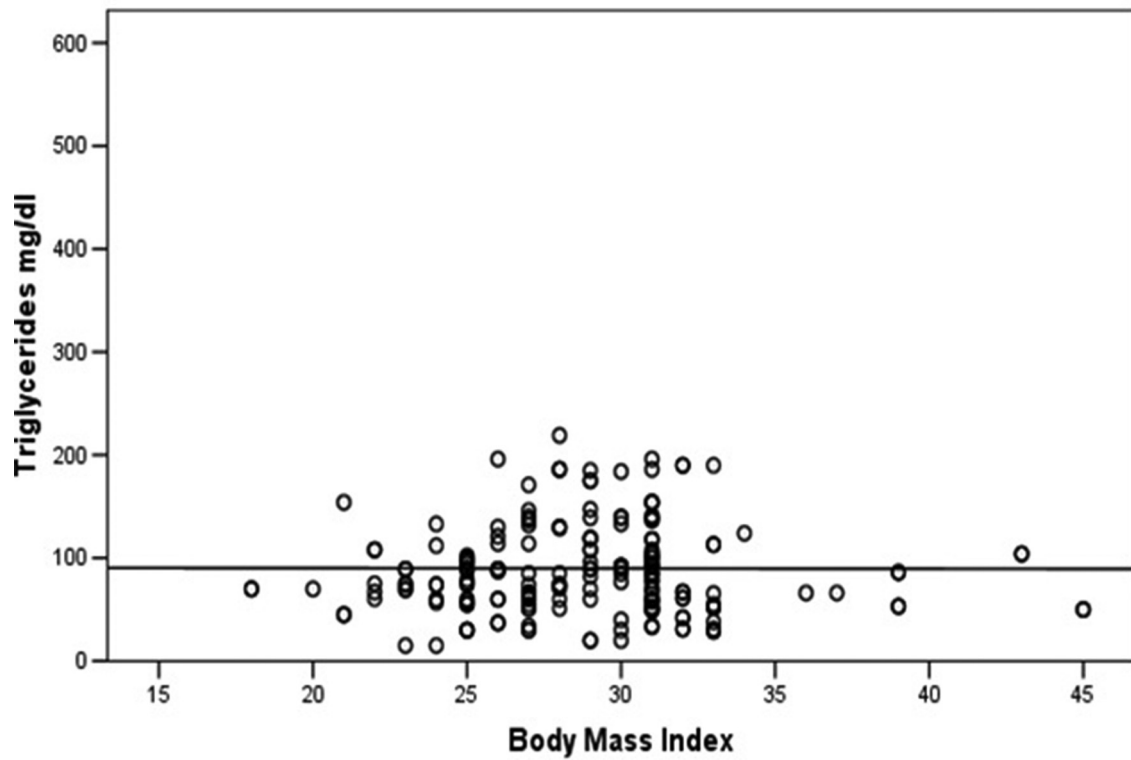


Figure 1: A scatter plot showing the correlation between body mass index (BMI) and triglycerides in the study group ($r = 0.00$).

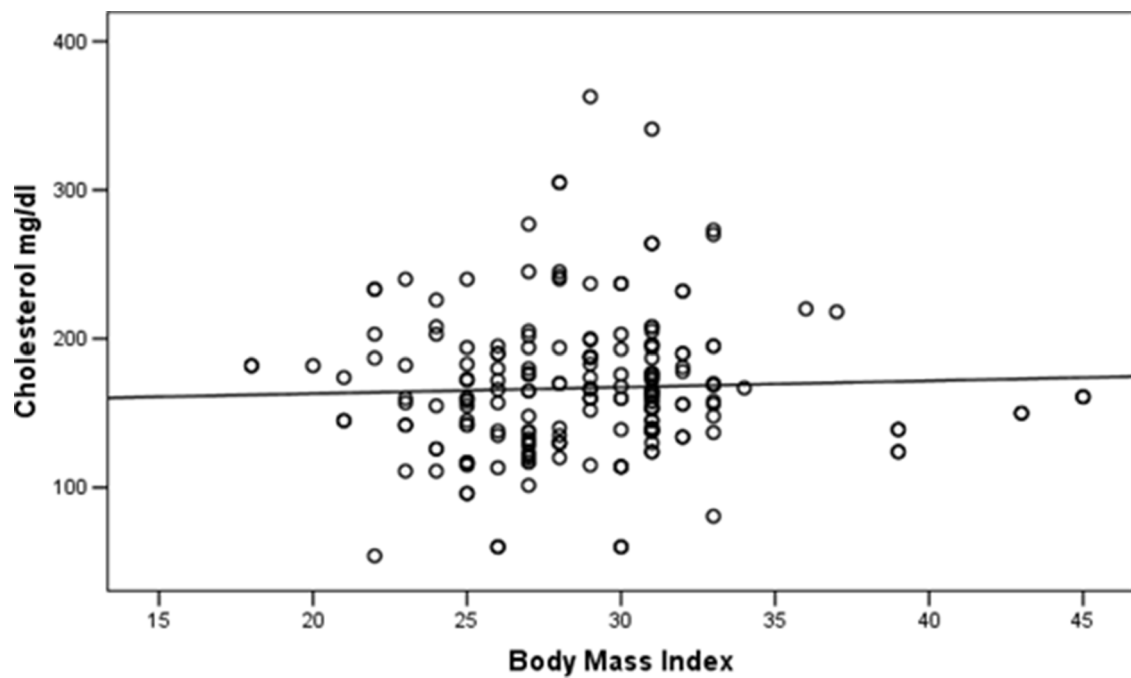


Figure 2: A scatter plot showing the correlation between body mass index (BMI) and cholesterol in the study group ($r = 0.04$, $P = 0.55$).

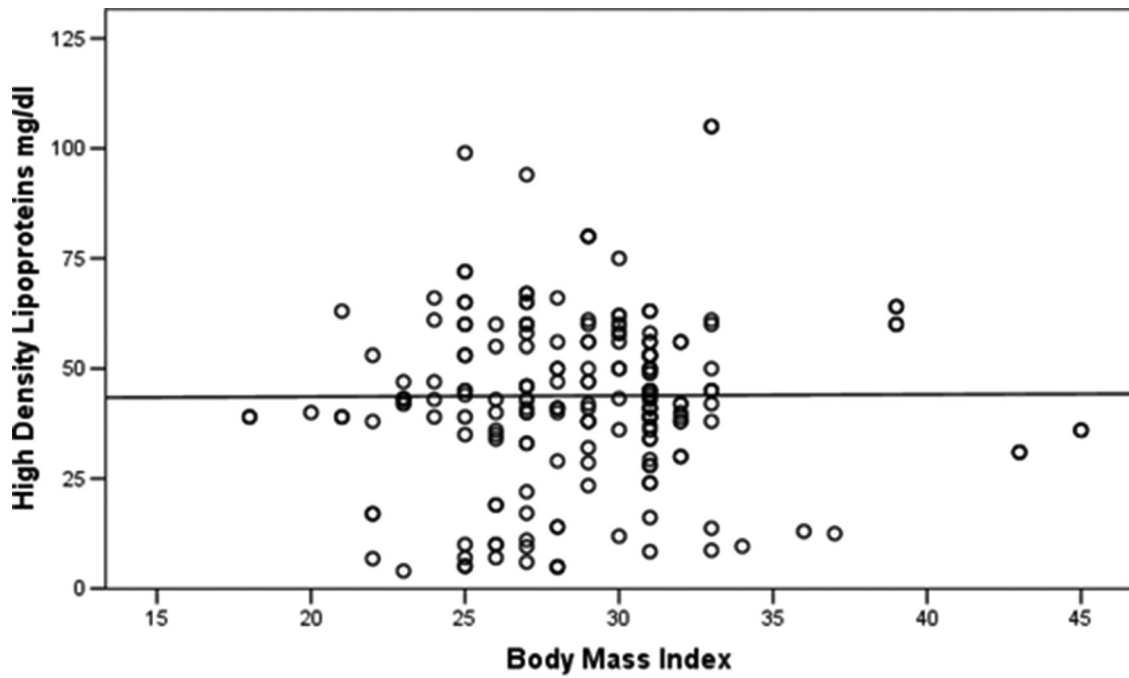


Figure 3: A scatter plot showing the correlation between body mass index (BMI) and high density lipoproteins (HDL) in the study group ($r = 0.01$, $P = 0.84$).

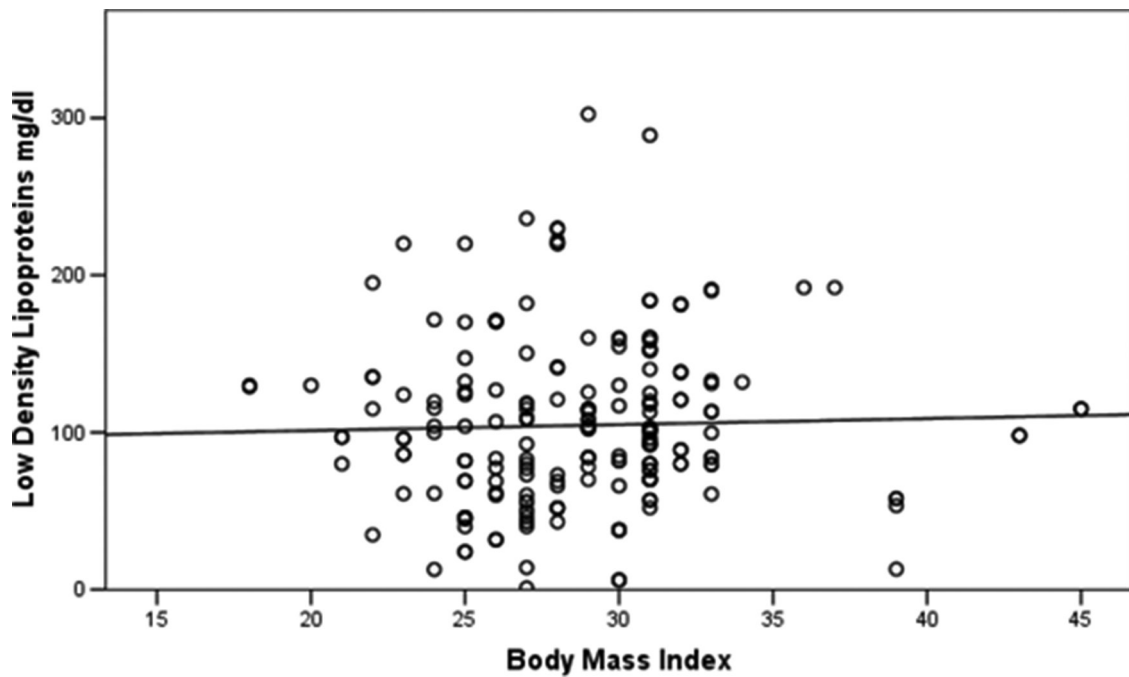


Figure 4: A scatter plot showing the correlation between body mass index (BMI) and low density lipoproteins (LDL) in the study group ($r = 0.04$, $P = 0.56$).

Discussion

The study was conducted to evaluate the changes in lipid profile parameters in Sudanese PCOS patients and healthy women as control group. PCOS is one of the important endocrine disorders causing reproductive abnormalities in women, which has heterogeneous clinical features and multifactorial etiology.^[16] Dyslipidemia is one of the important risk factors associated with PCOS. Obesity and insulin resistance occur frequently in association with this syndrome. Cardiovascular risk factors seem to cluster in women with PCOS when compared with general population.^[17]

In our study, levels of total cholesterol, TG, and LDL-C were statistically higher, and level of HDL-C was lower in PCOS patients, when compared with age-matched healthy females [Table 2]. This is similar to the results observed in PCOS patients in another study.^[18] Moreover, similar results found by some studies suggest that PCOS patients were hyperlipidemic with higher total cholesterol, LDL-C, and TGs concentrations and lower HDL-C levels than control.^[19,20] On the other hand, our results were not concurrent with the study done by Bickerton *et al.*^[21] who demonstrated that there were no significant differences in lipid or lipoprotein concentrations between patients with PCOS and weight-matched controls.

Lipid abnormalities were closely related to insulin resistance independent of obesity.^[22] The increase in TGs may be because of the accumulation of TGs, which may occur owing to the increased lipogenesis, decreased clearance, or reduced oxidation of fatty acids. Increased secretion of very-low-density lipoprotein (VLDL) particles by the liver resulted in elevated plasma TGs concentration. This may occur because of insulin resistance, which is seen in PCOS patients. Insulin resistance also contributes more catabolism of HDL-C particles and formation of LDL-C.^[23] Hyperandrogenism also contributed for altered lipid profile. Hyperandrogenism has been associated with increased hepatic lipase activity and has a role in the catabolism of HDL-C particles. Increased serum concentrations of TGs have also been recognized as a risk factor for CVD.^[24]

Our study showed that there was no correlation between the BMI and the serum TGs [Figure 1]. Moreover, in our study, the serum level of HDL-C was significantly decreased in the subjects in the PCOS group [Table 2] ($P < 0.05$). Similar results were found in study done by Wild *et al.*^[25] They found that women with PCOS showed lower HDL-C levels, higher LDL/HDL ratios, and higher TG levels than regularly menstruating women.

More recently, Slowinska-Srzednicka *et al.*^[26] have drawn the attention to the role of insulin in the lipid abnormalities observed in hyperandrogenic women with PCOS. These investigators compared 27 women with PCOS with 22 eumenorrheic control subjects stratified by weight (obese and nonobese). Women with PCOS showed significantly lower levels of HDL₂ and higher levels of apoB and TGs.

Our study, demonstrated that women with PCOS showed higher significant increase in the serum levels of LDL-C when

compared with the control subjects; similar results were found by Pirwany^[27] On the other hand, this study showed an insignificant correlation between the BMI and the serum levels of LDL [Figure 2]. This agreed with the results of a study done by Pirwany^[27] who concluded that women with PCOS show increased hepatic lipase activity and LDL relative to BMI-matched controls with normal menstrual rhythm and normal ovaries. Furthermore, these metabolic disturbances appeared to be related more closely to abnormal insulin metabolism and circulating androgen levels.

Our study showed that women with PCOS showed significantly decreased serum levels of HDL-C [Table 1] when compared with the control subjects; this result was similar to the study done by Wild *et al.*^[25] in 1985 who found that women with PCOS showed lower serum levels of HDL-C in comparison with healthy control subjects.

Recently, Slowinska-Srzednicka *et al.*^[26] have drawn the attention to the role of insulin in the lipid abnormalities observed in hyperandrogenic women with PCOS, when they compared 27 women with PCOS with 22 eumenorrheic control subjects stratified by weight (obese and nonobese); they found that women with PCOS showed significantly lower levels of HDL and an insignificant correlation between the BMI and the serum levels of HDL [Figure 3].

Finally, in our study, there was a significant increased serum level of total cholesterol when compared with the control subjects and an insignificant correlation between the BMI and the serum levels of total cholesterol [Figure 2]

Conclusion

From this study, it is concluded that women with PCOS have altered lipid profile, with higher levels of (TGs, total cholesterol, and LDL) and low level of serum HDL; this difference may play a role in the pathophysiology found in women with PCOS. Further studies are needed to clarify the role of lipids in these women. Moreover, elevated serum lipids reflected the metabolic state in those patients; all patients should be screened for dyslipidemia for effective cardiovascular risk prevention.

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