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Lipid Profile in Sudanese Patients with Polycystic Ovary Syndrome

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ABSTRACT

Polycystic ovarian syndrome **(**PCOS) is associated with dyslipidaemia and may render the affected women "at risk" of developing cardiovascular disease. Objective of this study was to evaluate lipid parameters in Sudanese Patients with Polycystic Ovary Syndrome. The study was case control hospital based carried, out on 32 diagnosed PCOS subjects aged (18-35 years) and 32 age-matched healthy women. Blood samples were collected in a fasting state and lipid parameters were estimated by Biosystem Kits using Spectrophotometer. The mean ± standard deviation of total cholesterol**,** triglycerides, LDL, VLDL, HDL respectively in cases were (194±36.3, 95.8±29.0, 128.4±32.1, 19.1±5.8, 47.1±14.1). while the mean and standard deviation of total cholesterol**,** triglycerides, LDL, VLDL, HDL respectively in control were (166.22±16.7, 86.1±12.6, 89.9±21.2, 17.22±2.53, 59.5±12.3). There were significant elevation in most lipid profile. The study concluded that, most lipid profile were significantly increased except triglycerides and VLDL.

Keywords: Polycystic ovarian syndrome, lipid profile, Sudanese.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a syndrome of ovarian dysfunction that is characterized by anovulation, hyperandrogenism and/or the presence of polycystic ovary morphology[1]. PCOS is the commonest endocrinopathy affecting reproductive aged women with a reported prevalence of 6-18%, depending on the diagnostic criteria and population studied[2]. Metabolic syndrome, including Dyslipidemia, Obesity and Insulin resistance occur frequently in association with this syndrome are likely to be the major risk factors for cardiovascular diseases [3-5]. Dyslipidemia in PCOS is characterized by elevated plasma levels of low- density lipoproteins (LDL), very-low-density lipoproteins (VLDL) and triglycerides with concomitantly reduced concentration of high-density lipoproteins (HDL)[6]. These changes are consistent with the lipid profile that is typically found in association with insulin resistance. Increased secretion of very low-density lipoprotein (VLDL) particles by the liver results in elevated plasma TG concentrations. Subsequently, TGs are exchanged for cholesteryl ester (CE) by the activity of CE transfer protein. This process results in TG enriched HDL particles that are catabolized more rapidly, and CE-enriched VLDL particles that are converted into small dense low-density lipoprotein (LDL) particles[7]. Increased level of VLDL, LDL cholesterol and hypertriglyceridemia, and decreased level of HDL cholesterol predispose patients to vascular disease in the polycystic ovary syndrome[8]. Both hyperandrogenemia and insulin resistance contribute to this atherogenic lipid profile[9]. The objective of this study was to evaluate the changes in serum lipid parameters in Sudanese patients with PCOS**.**

MATERIAL AND METHOD

The study was case control Hospital based carried out in 32 diagnosed PCOS patients attended Omdurman Maternity Hospital and Dr. Elsir Abu Elhassan Fertility Centre between September 2014 and May 2015 with amenorrhea (absence of vaginal bleeding for at least 6 months) or oligomenorrhea (Interval between menstrual periods more than 35 days) and serum FSH concentration within normal limits (1-10 iu/l), *i.e.* normogonadotropic anovulation (classification according to the World Health Organization) (10). The age of PCOS subjects and control range is (18-35 years). The diagnosis of PCOS is based on the Rotterdam criteria: presence of any two of (i) chronic anovulation; (ii) clinical/ biochemical parameters for hyperandrogenism; and (iii) polycystic ovaries on ultrasonography (11). In Addition to estimation of antimularrian hormone (AMH) which is more than 6 ng/ml and vaginal ultrasound examination (presence of 12 follicles or more in one or both ovaries and/or increased ovarian volume (>10 ml). Exclusion criteria were non fasting state at investigation, Diabetes Mellitus, congenital adrenal hyperplasia and Cushing's syndrome. 32

healthy females matched for age but without PCOS were included in the control group, exclusion criteria for controls were breast feeding, Diabetes Mellitus, irregular menstrual cycle at investigation, and non fasting state. Baseline data including detailed medical history, age, and body-mass index was calculated as weight (kg)/ [height (m)]^2, and Duration of the disease in case of PCOS patients.

Blood Sample

Five ml venous blood sample was obtained after overnight fasting of 12 hours by venepuncture from both cases and controls and the serum was isolated after centrifugation at 3000 rpm for 10 min at 20 C and stored at _20 C until Analysis. Total cholesterol (TC), serum triglycerides (TG), HDL cholesterol, were estimated using commercial kits (Biosystems, Spain). Serum very low density lipoprotein (VLDL) cholesterol and LDL cholesterol were calculated from the values of TC, TG and HDL cholesterol by applying Friedewald's formula. LDL (mg/dl) = total cholesterol (mg/dl) – HDL (mg/dl) – (triglycerides (mg/dl)/5) (12). TC/HDL and LDL/HDL ratios were determined.

Clinical and endocrine examination

Anovulatory patients underwent a standardized initial examination that was performed after an overnight fast on a random day between 0900 and 1100 h. Clinical examination included menstrual history and anthropometric measurements (height and weight). Vaginal ultrasound examination was performed to assess ovarian volume and follicle count for both ovaries. Blood samples were obtained by venipuncture and processed within 2 h. Serum was isolated after centrifugation at 3000 rpm for 10 min at 20 C and stored at -20 C until assayed. Endocrine evaluation included serum levels of gonadotropic hormones (LH, FSH) and prolactin were measured by immunoassay.

Ethical Issues

The University Ethical Committee approved the study protocol. Informed consent was obtained from all the participants. The obtained result was analyzed using SPSS-version20

RESULTS AND DISCUSSION

There was no statistically significant difference in Age (p = 0.12) and BMI (p = 0.41) between cases and controls. Compared with healthy women, women with PCOS had higher levels of Total cholesterol (mean: 194 *vs*. 166 mg/dl; *P* 0.001), LDL-cholesterol (mean: 128 *vs*. 89 mg/dl; *P* 0.00), higher mean TC/HDL (mean: 4.4 *vs*. 2.9 mg/dl; *P* 0.00) and LDL-C/HDL-C (mean: 3.0 *vs*. 1.6 mg/dl; *P* 0.00). HDL-cholesterol displayed lower levels in PCOS patients compared with healthy women (mean: 47 *vs*. 59 mg/dl; *P* 0.001). No statistically significant difference was observed between groups in terms of triglycerides levels (mean: 95 *vs*. 86 mg/dl; *P* 0.259) and VLDL (mean 19.1 *vs*. 17.2 mg/dl; *P* 0.102). The

demographic features and biochemical results of patients with PCOS and healthy controls are displayed in Table 1.

Table 1: Comparison of basic characteristics and lipid profile between women with PCOS and healthy controls

Parameters	PCOS	Controls	p-value
Age(years)	29.34± 5.72	31.41±4.8	0.125
BMI(kg/m2)	26.83±5.23	25.50±6.3	0.4101
Total cholesterol(mg/dL)	194.12±36.34	166.22±16.7	0.001
TG (mg/dL)	95.81±29.02	86.10±12.6	0.259
LDL (mg/dL)	128.41±32.19	89.90±21.2	0.00
VLDL (mg/dL)	19.16±5.80	17.22±2.5	0.102
HDL (mg/dL)	47.13±14.18	59.56±12.3	0.001
TC/HDL (mg/dL)	4.48±1.53	2.914±0.68	0.00
LDL/HDL(mg/dL)	3.03±1.35	1.60±0.67	0.00
FSH	6.35±2.25	6.19±2.9	0.808
LH	11.15±5.91	5.35±2.67	0.00
Prolactin	16.19±9.36	11.63±4.4	0.024

Values are expressed as mean ± standard deviation, BMI = body-mass index; TG = triglycerides; LDL = low density lipoprotein; VLDL = very low-density lipoprotein; HDL = high-density lipoprotein

The polycystic ovary syndrome (PCOS) is one of the most common endocrine diseases affecting women in reproductive age. Characterized by chronic anovulatory cycles, oligo- or amenorrhea, infertility, insulin resistance, obesity and associated with type 2 diabetes mellitus (T2DM), hypertension, dyslipidaemia and increased risk factors for the development of CVD. The main findings of the study presented here are the high levels of the total cholesterol, and LDL cholesterol in combination with decreased HDL cholesterol in women with PCOS when compared with healthy women. The data on cholesterol, HDL cholesterol, and LDL cholesterol are consistent with prior studies of dyslipidemia in women with PCOS (13). However, no significant difference in TC, TG, LDL, HDL, VLDL, TC/HDL and LDL/ HDL in patients with PCOS and control subjects were observed in another study[14]. The reason for dyslipidaemia in PCOS may be attributed to hyperinsulinaemia and hyperandrogenemia. This causes adipocytes to undergo increased catecholamine-induced lipolysis and release of free fatty acids into the circulation. Increased free fatty acids in the liver stimulate secretion in VLDL which ultimately leads to hypertriglyceridaemia[15]. Through the reverse cholesterol transport pathway, hypertriglyceridaemia leads to low HDL cholesterol and increased LDL cholesterol levels. Further priming of adipocytes by androgens in early life predisposes to the dyslipidaemia associated with PCOS. Conversely, it is also possible that hyperandrogenaemia is a consequence of more metabolically active adipocytes[16]. Hyperandrogenism may also affect lipid metabolism by the induction of hepatic lipase activity which has a role in the

catabolism of HDL particles (17). Insulin resistance has been associated with decreased levels of HDL and increased levels of LDL and TG. This has been associated with increased risk of CAD[13].

CONCLUSION

The results of this study may indicate increased risk for cardiovascular disease in women with PCOS. However, this hypothesis still remains to be proven in prospective long-term follow-up studies of women with PCOS.

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