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STUDY OF SELECTED BIOCHEMICAL PARAMETERS IN WOMEN WITH POLYCYSTIC OVARIAN SYNDROME

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ABSTRACT

Introduction

PCOS is the multisystemic endocrinopathy expressed with wide varieties of aetiologies and variable clinical signs in reproductive age group female individuals, classically described, as a triad consisting of amenorrhea, hirsutism and obesity. PCOS is a composite disorder that commence during puberty and affects reproductive age women. It is characterized by a complex and often serious array of metabolic and endocrine implications such as diabetes, obesity, infertility etc.,

Objectives:

To analyse and correlate the biochemical parameters (Glucose, Magnesium, Uric Acid and lipid profile) in women with PCOS.

Materials and Methods:

After Ethical Committee Approval, blood samples were collected from 60 diagnosed PCOS cases and 60 healthy controls (premenopausal women); aged 18 to 40 years. Fasting plasma glucose, serum magnesium, uric acid and lipid profile were investigated in both PCOS patients and controls. The correlation between these biochemical parameters were then studied in the PCOS group. Data analysis done using student t' test.

Result:

There was a remarkable increase in fasting plasma glucose and serum uric acid levels with decrease in serum magnesium levels in PCOS patients as compared to controls. PCOS women had higher BMI with increased total cholesterol, TGL, LDL-C, VLDL-C and lower HDL-C ($P < 0.05$) as compared to the controls which was statistically significant. The levels of glucose showed significant positive correlation with uric acid ($P < 0.01$), total cholesterol($P<0.01$), triglycerides($P<0.05$), LDL-C ($P < 0.01$) and significant negative correlation with magnesium ($P<0.01$) and HDL-C. Uric Acid showed significant negative correlation with HDL-C and significant positive correlation with LDL-c ($P<0.05$).

Conclusion: The above findings of this study comply with the association between Glucose, Magnesium, Uric Acid, BMI and dyslipidaemia in PCOS and may help to identify women with PCOS at risk of cardio metabolic syndrome thereby confirming the association between PCOS and cardiovascular risk factors.

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Keywords: Cardio Metabolic Syndrome , Dyslipidaemia ,Polycystic Ovarian Syndrome, Premenopausal women

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is the multisystem endocrinopathy with ovarian expression of metabolic disturbances and a wide spectrum of clinical features seen in women of reproductive age.[1] It is characterized by increased ovarian and adrenal androgen secretion, hyperandrogenic metabolic syndrome symptoms such as hirsutism, acne and/or alopecia, menstrual irregularity and polycystic ovaries. It is not only a reproductive endocrinopathy but also a metabolic disorder.[2] It is prevalent in about 7 percent of the women worldwide and in approximately 4 to 11 percent of Indian women in the reproductive age.[3]

The pathophysiology is complex involving the hypothalamus-pituitary-ovarian axis, ovarian theca cell hyperplasia, hyperinsulinemia and a multitude of other cytokine and adipocyte-driven factors.[5]

Women with PCOS share many features in common with the metabolic syndrome in particular.[1] One of the most prominent metabolic symptoms of PCOS is insulin resistance, which includes hyperinsulinaemia and impaired glucose tolerance. Women with PCOS are known to be at increased risk for insulin resistance.[6]

Magnesium, a cofactor of many enzymes involved in glucose metabolism, is required for both proper glucose utilization and insulin signalling. In particular it has been shown that magnesium plays the role of a second messenger for insulin action.[8] Low magnesium concentrations are associated with impaired glucose tolerance and increased risk for Type2 diabetes mellitus. It is currently unknown whether women with PCOS exhibit serum magnesium deficiency and its potential association with glycemic levels.

Uric Acid is a metabolic end product of purine metabolism. It is a strong reducing agent and potent antioxidant. The possible relationship between androgens and serum uric acid concentrations is supported by animal experiments showing that androgens may increase serum uric acid levels by inducing the hepatic metabolism of purines.[9] The studies available at present regarding serum uric acid levels in PCOS patients are scarce and led to controversial results.

Obesity and excess weight are major chronic diseases in western world countries. Obesity increases hyperandrogenism, hirsutism, infertility and pregnancy complications both independently and by exacerbating PCOS. Likewise, in PCOS obesity worsens insulin resistance and exacerbates reproductive and metabolic features.[10]

PCOS is associated with long-term health risks including Type2 diabetes mellitus and coronary artery disease.[4,5] Insulin resistance, hyperandrogenism and dyslipidemia are likely to be the major risk factors for CVD in women with PCOS. The reason for the increase in the risk is not yet clear; hyperandrogenism has not yet been recognized as a risk factor for cardiovascular disease and studies on pre- and post-menopausal women do not show a clear association between hyperandrogenism and the risk of future cardiovascular events. Insulin resistance and dyslipidemia seem to have an important role on the risk of cardiovascular pathology in women with PCOS.

PCOS is a chronic disease with manifestations across the lifespan and represents a major

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health and economic burden. Diagnosis of PCOS is extremely important because it in turn identifies risk for potential metabolic and cardiovascular diseases. Several biochemical and clinical features of PCOS resemble those of metabolic syndrome. Therefore, it is recommended that women with PCOS be routinely screened so that treatment can be initiated earlier. The increased incidence of cardiovascular disease in women with PCOS has prompted researches to look for indicators of early metabolic changes in these patients. In view of this, the present study was undertaken to analyse and correlate the biochemical parameters that may help to identify women with PCOS at risk of Cardiometabolic syndrome.

OBJECTIVES

1. To estimate the levels of plasma Glucose, serum magnesium, serum Uric acid and total cholesterol, triglycerides, HDL-c, LDL-c and VLDL cholesterol in Women with PCOS and age matched healthy controls.
2. To compare the levels of plasma Glucose, serum magnesium, serum uric acid and total cholesterol, triglyceride, HDL-c, LDL-c, VLDL cholesterol between PCOS cases and controls.
3. To correlate the levels of biochemical parameters plasma glucose, serum magnesium, serum uric acid and total cholesterol, triglyceride, HDL-c, LDL-c, VLDL cholesterol in PCOS cases.

METERIALS AND METHODS

Source of data

The study was conducted at NRI Medical College & General Hospital, from August 2022 - October 2022. Study consists of 60 premenopausal Female patients of PCOS diagnosed by Rotterdam criteria as cases and 60 age matched healthy female patients and volunteers with regular menstrual cycles and with no clinical or biochemical features of hyperandrogenism as controls thereby excluding diagnosis of PCOS. Institutional ethical committee approved the study and informed consent obtained from all the study subjects.

Study design: observational case-control study

Inclusion criteria:

Cases: Female patients diagnosed with PCOS based on Rotterdam Criteria, not on any treatment, in the age group of 18-40 years.

Diagnosis based on Rotterdam criteria (2003)

a. Oligomenorrhoea / Amenorrhoea

b. Clinical / Biochemical signs of hyperandrogenism Hirsutism
Acne
Alopecia
Elevated androgen levels

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c. Presence of Polycystic ovaries on USG

Controls: Age matched healthy female volunteers with regular menstrual cycles and no signs of clinical or biochemical hyperandrogenism.

Exclusion criteria

1. Volunteers with DM, HTN
2. Volunteers with thyroid disorders, Renal diseases, cardiovascular diseases, cushing syndrome
3. Pregnant or lactating women
4. Women on Oral contraceptive pills
5. Volunteers on drugs like hypoglycemic agents / lipid lowering drugs
6. Volunteers on Harmonal medicines within 6 weeks

Method of collection of data

Informed consent was taken from patients and controls. A pre-structured and pretested proforma was used to collect the data. Baseline data including age, BMI, detailed medical history, family history, clinical examinations were included as part of the methodology.

Biochemical Parameters Estimation

Estimation of Glucose in blood (GOD/POD method) [11]

Estimation of serum Magnesium by Xylidyl blue method[12]

Estimation of serum Uric Acid by Modified Trinder Method, End point [13]

Estimation of Total Cholesterol in serum (Dynamic extended stability- Cholesterol oxidase/ peroxidise method, End point) [14]

Estimation of serum Triglycerides (GPO-Trinder method end point)

[15] Estimation of serum HDL-c (Phosphotungstic Acid Method, End point)[16]Estimation of Serum Very Low Density Lipoprotein-Cholesterol [17]

Estimation of Serum Low Density Lipoprotein-Cholesterol [18]

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Data Analysis was performed using SPSS 16 Software. The values were expressed as mean

± Standard Deviation. Deviation and the findings were analysed by student "t" test.Pearson's correlation coefficients were calculated to assess the correlation between the biochemical parameters in the study group.
A 'P' value of < 0.05 was considered statistically significant.

RESULTS

Table 1: Age distribution in study group

Age in years	Cases		Controls		Total
	No.	%	No.	%	
18-20	6	10	6	7	12
21-25	18	30	18	28	36
26-30	27	42	27	45	54
31-35	5	12	5	11	10
36-40	4	6	4	9	8
Total	60	100.0	60	100.0	120
Mean age ±SD	26.19 ± 3.88		27.40 ±5.020		
P value	P < 0.05				

The age group of subjects was between 18-40 years. The distribution of the study samples according to the age is given in table 9 and graphically represented in fig.7. The cases and controls are divided into 5 groups (≤ 20years, 21-25yrs, 26-30yrs, 30- 35yrs, 36-40yrs) Maximum numbers of cases are in the age group of 26-30yrs (48%). The mean age and standard deviation in cases was 26.16±3.77 years and in controls it was 27.38±5.015 years. The mean age between the cases and controls is not statistically significant (P>0.05). The cases and controls are age matched.

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Table 2: Comparison of mean age between two group:

Groups	Mean ± SD	t-value	P value
Cases	26.19 ± 3.88	1.49	P < 0.05
controls	27.40 ±5.020		NS

The mean age between the cases and controls is statistically significant

(P< 0.05)

Anthropometric data

The anthropometric data of the study groups and control is shown in Table no 17.

Body Mass Index [BMI]

The [mean ±SD] BMI of the study groups was 27.50± 2.54 kg/m2 and of the controls 25.9± 2.21 kg/m2. The mean difference in BMI between the two groups was statistically significant.

Waist circumference

The (mean±SD) waist circumference of the study group was 85.47± 5.46 cm and of the controls 78.2 ± 4.34 cm. The mean difference in waist circumference between the two groups was statistically significant.

Waist hip ratio: The (Mean±SD)waist hip ratio of the study group was 0.786±0.0551 and of the controls78.2 ± 4.34. There was no statistically significant mean difference in waist hip ratio between cases and controls.

Comparison of Anthropometric parameters

Comparison of Anthropometric parameters BMI, Waist circumference, Waist hip ratio has been done between the two groups.

Table 3: Comparison of Anthropometric parameters in cases and controls

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Anthropometric parameters	**Cases N=50 Mean ± SD**	**Controls N = 50 Mean± SD**	**t - value**	**P – value**
BMI (kg/m^2)	28.50± 3.54	26.9± 2.41	4.06	P < 0.01 **
Waist circumference in Cm	86.49± 6.46	79.2 ± 5.45	6.04	P <0.001 ***
Waist hip ratio	0.876±0.0268	0.7884± 0.053	0.273	P < 0.05 NS

Statistically significant Higher mean BMI and Waist circumference is recorded in cases than in controls but Waist hip ratio shows higher mean in cases than controls but the difference in mean waist hip ratio between the two groups is statistically significant (P<0.05).

Blood pressure

The systolic blood pressure (SBP) and diastolic blood pressure (DBP) was measured using sphygmomanometer in the patients left arm.

The values of systolic blood pressure and diastolic blood pressure and their standard deviation (SD) are presented .

The mean SBP and the mean DBP for cases is 119.50 ± 7.79 mmHg, 78.92 ± 6.473 mmHg respectively. For the control group SBP was 112.96 ± 4.922 mmHg and DBP was 73.59 ± 3.89mmHg.

Table No.4: Showing mean ± SD and the significant difference in the mean

Systolic Blood pressure (mmHg) and Diastolic Blood pressure (mmHg) between

the study groups

Parameters	Cases N = 50 Mean ± SD	Controls N = 50 Mean ± SD	t-Value	P value
SBP	119.50 ± 7.79	112.96 ± 4.922	5.06	P < 0.001 ***
DBP	78.92 ± 6.473	73.59 ± 3.89	5.12	P < 0.001 ***

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Fasting Blood Glucose

The (Mean±SD) FBS of the study group was 98.92±8.298mg/dl and 91.20±9.20 mg/dl of the controls. The mean difference in Fasting Blood Glucose between cases and controls was statistically significant. The mean value of FBS and its standard deviation for cases and controls.

Table No.5: Showing mean ± SD and the significant difference in the mean

fasting plasma glucose values (mg/dl) between the study groups

Parameter	Cases Mean ± SD	Controls Mean ± SD	t-test	P value
FBS	98.92±8.298	91.20±9.20	5.20	<0.001 ***

Serum magnesium

The (Mean±SD) serum magnesium of the study group was 1.692 ± 0.205and 2.196 ± 0.31of the controls. The mean difference in serum magnesium between cases and controls was statistically significant. The mean value of serum magnesium and its standard deviation is given in table 6

Serum Uric acid

The (Mean±SD) serum Uric acid of the study group was 5.12 ±1.52mg/dl and of the control group was 3.20 ±0.892mg/dl. The mean difference in serum uric acid between cases and controls was statistically significant. The mean value and standard deviation of serum uric acid is shown in table 6.

Table No.6: Showing mean ± SD and the significant difference in the mean serum magnesium values (mg/dl) and serum Uric acid values (mg/dl) between the study groups.

Parameter	Study group Mean ± SD	Control group Mean ± SD	t-value	P value

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Serum magnesium mg/dl	1.692 ± 0.205	2.196 ± 0.31	4.59	<0.001 ***
Serum uric acid mg/dl	5.12 ±1.52	3.20 ±0.892	5.59	<0.001 ***

Lipid profile

The mean value and standard deviation of serum total cholesterol, serum triglyceride, serum HDL-c, serum LDL-c and serum VLDL-c .

Table No.7: Showing mean ± SD and the significant difference in the mean serum total cholesterol (mg/dl), serum triglyceride (mg/dl), serum HDL-c 9mg/dl), serum LDL-c (mg/dl) and serum VLDL-c values (mg/dl) between the study groups

Parameter	Study group Mean ±SD	Control group Mean ±SD	t-value	P value
Total cholesterol (mg/dl)	195.54 ±29.08	155.69 ± 18.20	4.91	< 0.001 ***
Triglyceride (mg/dl)	148.2 ± 38.22	111.19 ± 22.88	3.92	< 0.01 **
HDL-c (mg/dl)	46.06 ± 9.82	58.48 ± 6.66	2.99	< 0.05 *
LDL-c (mg/dl)	132.72 ± 30.19	99.977 ± 19.55	5.39	<0.001 ***

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VLDL-c (mg/dl)	39.71 ± 9.999	18.99 ± 9.567	4.47	<0.01**

The(mean±SD) serum total cholesterol of the study group is 195.54 ±29.08mg/dl and of the controls is 155.69 ± 18.20mg/dl. Mean total cholesterol value is significantly higher in cases than the controls. The (Mean±SD)serum triglyceride of the study group was 148.2 ± 38.22mg/dl and of the controls is 111.19 ± 22.88 mg/dl. There is significant difference in mean TG values between cases than controls, mean values being higher in cases than controls.

The mean HDL-c values are significantly lower in cases 46.06 ± 9.82 as compared to controls with 58.48 ± 6.66 where as the mean LDL-c values are significantly higher in cases was 132.72 ± 30.19 than controls 99.977 ± 19.55. The (Mean±SD) serum VLDL-c of the study group was 39.71 ± 9.999 mg/dl and of the controls is 18.99 ± 9.567mg/dl. The mean VLDL-c values are significantly higher in cases than controls.

Total cholesterol (mg/dl)

The (Mean±SD) serum total cholesterol of the study group was 187.44±25.08 mg/dl and of the control was165.52±19.21 mg/dl. The mean and standard deviation value of total cholesterol .

Table 8: Distribution of total cholesterol (mg/dl) among cases and controls

Total cholesterol (mg/dl)	Cases		Controls		Total
	No	%	No	%	
<200 mg/dl	43	72%	45	76%	88
200-239 mg/dl	14	23%	15	24%	22
>240 mg/dl	3	6%	0	0%	10
Total	60	100%	60	100%	120

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From the table it can be inferred that the cases and controls are highest in the <200mg/dl category (Desireable level of TC). While 72% of cases and 76% of controls are in this group. 23% of cases and 15% of controls are in the borderline group which ranges from 200-239mg/dl. In the >240mg/dl category 6% cases and no controls are seen.

Serum triglyceride(mg/dl)

The (Mean±SD) serum triglyceride of the study group was 138.3±40.32 mg/dl and of the control was 104.69±32.88 mg/dl. The mean and standard deviation of serum triglyceride of study groups .

Table 9: Distribution of serum triglyceride (mg/dl) among cases and controls

Serum triglyceride (mg/dl)	Cases		Controls		Total
	No	%	No	%	
< 150 mg/dl	23	38%	32	72%	55
150 – 199 mg/dl	29	45%	22	22%	51
200 – 499 mg/dl	8	16%	6	6%	14
Total	60	100%	60	100%	120

From the table it can be inferred that 38% of cases and 72% of controls are in the normal range (< 150 mg/dl). 45% of cases and 22% of controls are in the borderline range (150-199mg/dl).16% of cases and 6% of controls are in the higher range (200- 499mg/dl).

HDL-c mg/dl (high density lipoprotein cholesterol)

The (mean ±SD) HDL-c of the study group was 39.64±8.87 mg/dl and of the controls was 23±5.86 mg/dl. The mean values and standard deviation of serum HDL-c mg/dl is shown in table 10. The distribution of serum HDL-c mg/dl among cases and controls .

Table 10: Distribution of serum HDL-c (mg/dl) among cases and controls

Serum	Cases	Controls	Total

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HDL-c (mg/dl)	No	%	No	%	
≤ 40 mg/dl	39	59%	23	33%	62
>40 mg/dl	21	41%	37	67%	58
Total	60	100%	60	100%	120%

From the table it can be inferred that 59% of cases and 33% of controls are in the

≤40mg/dl . 41% of cases and 67% of controls are in > 40mg/dl range.

LDL-c mg/dl (Low density Lipoprotein cholesterol)

The (Mean ±SD) serum LDL-c of the study group was 128.10±24.01 mg/dl and of the controls was 88.79±19.45 mg/dl. The mean and standard deviation of serum LDL-c mg.

Table 11: Distribution of serum LDL-c mg/dl among cases and controls

Seum LDL-c (mg/dl)	Cases		Controls		Total
	No	%	No	%	
≤ 129 mg/dl	38	63%	54	88%	92
>129mg/dl	22	37%	6	12%	28
Total	60	100%	60	100%	120

From the table it can be inferred that 63% of cases and 88% of controls are in normal range (≤129mg/dl). 37% of cases and 12% of controls are in the higher range (>129 mg/dl).

VLDL-c mg/dl (very low density lipoprotein cholesterol)

The (Mean ±SD) VLDL-c of the study group was 26.064±8.064 mg/dl and of the controls was 20.93±6.576 mg/dl. The mean value and standard deviation of serum VLDL-c is shown in table 15 and graphically represented in Fig 13. The distribution of serum VLDl-c mg/dl among cases and controls is shown in table 15.

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Table 12: Distribution of serum VLDL-c mg/dl among cases and controls

Serum VLDL-c (mg/dl)	Cases		Controls		Total
	No	%	No	%	
< 40 mg/dl	34	72%	52	96%	86
>40mg/dl	26	28%	8	4%	34
Total	60	100%	60	100%	120

From the table it can be inferred that 72% of cases and 96% of controls are in the normal range (<40mg/dl). 28% of cases and 4% of controls are in the higher range (>40mg/dl).

Correlation between different parameters

Table 13: Correlation between BMI and other parameters

Parameter	BMI (kg/m^2)	
	Pearson coefficient (r)	P value
Fasting blood sugar (mg/dl)	0.767	<0.01**
Uric acid (mg/dl)	0.462	<0.01**
Magnesium (mg/dl)	-0.351	<0.01**
Total cholesterol (mg/dl)	0.961	<0.01**
Triglyceride (mg/dl)	0.658	<0.01**
HDL-c (mg/dl)	-0.103	<0.05*
LDL-c (mg/dl)	0.660	<0.01**
VLDL-c (mg/dl)	0.894	<0.01**

From the table it can be inferred that BMI (kg/m^2) has significant positive correlation with

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Fasting blood glucose, uric acid, total cholesterol, triglyceride, LDL-c and VLDL-c where as significant negative correlation with magnesium and HDL-c.

Table 14: Correlation between Fasting Blood Glucose and other parameters

Parameter	**Fasting blood sugar (mg/dl)**	
	Pearson coefficient (r)	**P value**
Uric acid mg/dl	0.535	<0.01**
Magnesium mg/dl	-0.838	<0.01**
Total cholesterol mg/dl	0.590	<0.01**
Triglyceride	0.377	<0.05*
HDL-c mg/dl	-0.151	< 0.05
LDL-c mg/dl	0.969	<0.01**
VLDL-c mg/dl	0.231	<0.05*

Fasting blood sugar level has significant positive correlation with serum uric acid, serum total cholesterol, serum triglyceride, serum LDL-c and serum VLDL-c where as significant negative correlation with serum magnesium and with serum HDL-c.

DISCUSSION

In the present study titled "Study of selected biochemical parameters in women with PCOS", I have got my parameters fasting blood glucose, serum magnesium, serum uric acid, serum total cholesterol, serum triglyceride, serum HDL-c, Serum LDL-c and Serum VLDL-c, which can be interpreted as follows the fasting blood glucose levels are considered, as patients with IFG and/or IGT are now referred to as having "pre-diabetic" indicating the relatively high risk for development of diabetes in these patients. IFG and IGT are not clinical entities in their own right but rather risk factors for

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future diabetes as well as cardiovascular diseases. Serum magnesium levels are taken, as low magnesium concentrations are associated with impaired glucose tolerance and increased risk for Type2 diabetes mellitus. Uric acid is considered as it exerts proinflammatory, prooxidant and proliferative actions at the endothelial cell level that may increase cardiovascular risk and is frequently associated with abdominal adiposity, obesity, insulin resistance, chronic low-grade inflammation. Although increased uric acid may influence some of these associations, the studies available at present regarding serum uric acid levels in PCOS patients are scarce and led to controversial results. Obesity increases hyperandrogenism, hirsutism, infertility and pregnancy complications both independently and by exacerbating PCOS. Likewise, in PCOS obesity worsens insulin resistance and exacerbates reproductive and metabolic features.[10] The main pathogenic mechanism relating all these parameters is insulin resistance with compensatory hyperinsulinaemia leading to altered glucose levels, Dyslipidaemia.

Considerable evidence has accumulated for the coexistence of the metabolic syndrome and PCOS. A key alteration in the former appears to be insulin resistance which is associated with an increased morbidity and mortality due to coronary arterydisease with its enormous public health implications. It has been suggested that inherited defects leading to peripheral insulin resistance and concurrent hyperinsulinaemia are among the causative factors for the development of PCOS.

Our study included a total number of 50 patients with confirmed diagnosis of PCOS and 50 healthy controls. The age of the patients in our study ranged from 18-40 years. The mean age between cases and controls in our study is not found to be statistically significant($P>0.05$). This is in agreement to other studies who found no difference in age between PCOS and control group.[13]

Obesity

Obesity is defined as BMI of > 30 and overweight as BMI of 25-29.9. PCOS patients display central or abdominal or android obesity, which is characterized by an increased waist-hip ratio. This visceral distribution of adipose tissue can be inferred clinically by a waist-hip ratio of more than 0.85.[19] In our study the mean BMI in normal healthy women (controls) is 25.9±2.21(kg/m^2) and in PCOS women(cases) is 27.50±2.54(kg/m^2). The mean BMI was higher in PCOS cases than controls and the mean difference was statistically significant ($P<0.01$). This is in accordance with other study who showed that excess visceral fat seems to be predictive not only of the metabolic syndrome but also of CVD.[18] A person's waist circumference is the simplest way to assess central obesity. Waist circumference has been shown to be one of the most accurate anthropometrical indicators of abdominal fat.[20] Waist circumference≥80 cm in women is considered as a positive indicator of abdominal obesity as per the consensus India guidelines for Indian population.[21] A Study done by Ian Jassen et al also showed that waist circumference is a better indicator of abdominal obesity.[22]

In our study mean waist circumference for normal healthy women (controls) is 78.2±4.34 cm and in PCOS women(cases) is 85.47±5.46 cm. There was highly significant statistical difference in the mean waist circumference values ($P<0.001$).

The mean waist hip ratio for normal healthy women (controls) is 0.7384±0.033 and in PCOS women (cases) is 0.786±0.0551. The mean waist hip ratio was higher in cases than controls

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and the mean difference was not statistically significant.

Hypertension

Young PCOS have blood pressure within normal range, but they have increased prevalence of labile daytime blood pressure, which predisposes them to sustained hypertension in later life. These adolescents do not demonstrate fall of blood pressure at night, like the normal individuals, and this predisposes them to develop hypertension in later life. At menopause, PCOS patients have 2.5 times more risk of developing hypertension, likely due to obesity, when compared to age matched controls.[19]In our study mean systolic blood pressure in normal healthy women (controls) is 110.96±5.992 mmHg and in PCOS women(cases) it is 118.48±8.795 mmHg. The mean diastolic blood pressure in controls is 74.52±4.19 mmHg and in cases it is 78.92±5.473 mmHg. There is highly significant statistical difference in the mean blood pressure values ($P<0.001$)

Lipid parameters

Our study showed highly significant statistical difference in the mean values of TC, TG, LDL-c and VLDL-c in PCOS cases than controls, the mean values being higher in cases than controls where as mean HDL-c values were lower in PCOS cases than controls and the difference was statistically significant.

To the contrary, they also found that the most classic lipid alteration determining cardiovascular risk, increase of LDL-C, is not common in all populations with PCOS. Beyond total LDL-C concentrations, the quality of LDL may exert a direct influence on the CV risk. The National Cholesterol Education Program Adult Treatment Panel III accepts that small, dense LDL has an approximately 3-fold increased risk for coronary artery disease and is stated as an emerging CV risk factor.[16] Our study found that HDL-C is lower in PCOS group than in control group whereas higher mean VLDL was seen in PCOS compared to controls. This is consistent with the other study who showed that women with PCOS had higher triglycerides and VLDL-C with lower HDL2-C and apolipoprotein A1:A2 ratios.

A study by Anuradha Kalra et al., found no correlation between BMI with various lipid parameters.[2] But in our investigation, we found a significant positive correlation between BMI and total cholesterol, triglycerides, LDL-C, VLDL- C and significant negative correlation between BMI and HDL-C. Our study is in agreement with the another study where the total serum cholesterol, triglycerides, LDL cholesterol (LDL-C) and Very Low-density lipoprotein cholesterol(VLDL-C) were higher in the women with PCOS and higher BMI. Significantly lower levels of serum HDL-C were also noted. Positive correlations were observed between: uric acid and HDL-C, glucose and total cholesterol, triglycerides, LDL-C, andVLDL-C.

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whereas our study showed non significant negative correlation between uric acid and HDL-c. Abnormally high serum triglyceride values were noted in more than one-third of the PCOS, thereby signifying the importance of serum lipid analyses in the diagnosis andfollow-up of PCOS.[23]

Another study have compared 27 women with PCOS and 22 control subjects who were divided into obese and non-obese categories. Women with PCOS had higher apoB and triglycerides levels and lower HDL2 levels. The result of multiple regression analysis has shown fasting insulin as a significant explanatory variable for total triglyceride and apoA-1 levels. Hence, it was suggested that hyperinsulinemia is independent of obesity and may play a vital role in the lipid disturbances observed in PCOS women..

Our study showed that PCOS women had higher BMI, significantly increased total cholesterol, triglycerides, LDL-C and VLDL-C. On the other hand, serum levels of HDL-C were significantly lower in this group compared to controls. The findings of this study confirm the association between BMI and dyslipidaemia in PCOS.

Serum uric acid

The results of our study showed the mean serum uric acid levels in PCOS cases as 4.82±0.54mg/dl and in controls as3.99±0.589 mg/dl. There was highly significant statistical difference in the mean serum uric acid values, being higher in PCOS cases than controls ($P<0.001$). There was also highly significant positive correlation between serum uric acid and BMI, FBS, TG, LDL-c and VLDL-c and non significant negative correlation with HDL-c.According to L.Anttila et al., and Manuel Luque-Ramirez etal., no statistically significant differences were found in the mean concentrations of uric acid between PCOS and control women. But, in our study, we found elevated serum uric acid levels in PCOS patients as compared to the controls which were statistically significant. According to another study Serum uric acid concentrations were positively correlated with BMI in the PCOS group. No correlation was found between the serum levels of uric acid and triglycerides[24]

Fasting blood glucose

Impaired glucose tolerance (IGT) AND impaired fasting glucose (IFG) are now referred to as "pre-diabetic" indicating the relatively high risk for development of diabetes in these persons.

In view of this the present study was undertaken to analyse fasting blood glucose levels in PCOS cases and normal healthy women and find correlation with other biochemical parameters serum magnesium, serum uric acid and lipid profile in PCOS cases as indicators of early metabolic changes.

The results of our study showed mean fasting blood glucose in normal healthy women (controls) as 90.28±8.52 mg/dl and in PCOS women (cases) it was 97.92±7.196 mg/dl. There was very highly significant statistical difference in the mean fasting blood glucose values ($P<0.001$). Our study also showed significant positive correlation between fasting blood glucose and BMI, TG and VLDL-c ($P<0.05$) and highly significant positive correlation with serum uric acid, TC and LDL-c ($P<0.01$) where as highly significant negative correlation with serum magnesium ($P<0.01$) and non significant negative correlation with HDL-c ($P>0.05$).

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Serum magnesium

Magnesium plays a key role in regulating insulin action, insulin-mediated glucose uptake, and vascular tone. Intracellular magnesium depletion may result in a defective tyrosine kinase activity at the insulin receptor level, in a postreceptorial impairment in insulin action, and clinically in a worsening of insulin resistance.

Our study showed the mean serum magnesium value of controls as 2.108±0.28 mg/dl and in cases was 1.894±0.205 mg/dl. There was highly significant statistical difference in mean serum magnesium value ($P<0.001$).

Our study showed decreased magnesium levels in PCOS cases compared to controls and highly significant negative correlation between fasting blood glucose and serum magnesium. Therefore, Intracellular magnesium deficiency may affect the development of insulin resistance and alter the glucose entry into the cell.

In recent years, increasing attention has been paid to the non-reproductive aspects of PCOS.PCOS may represent the largest under-appreciated segment of the female population at risk of cardiometabolic syndrome. The long-term impact of the metabolic disturbances associated with the disorder on women's health has focused considerable interest on follow-up studies and intervention studies.

CONCLUSION

The present study was done to analyse the biochemical parameters glucose, magnesium, uric acid and lipid profile in PCOS cases and controls and to find correlation between these parameters in PCOS cases as indicators of early metabolic changes that can be used to diagnose PCOS women at risk of cardiometabolic syndrome. The results of this study also provided the evidence showing significantly higher serum uric acid concentrations in PCOS patients as compared to controls and showed a positive correlation between uric acid and blood glucose levels thereby establishing the association between glucose, magnesium and uric acid. PCOS women had higher BMI, significantly increased total cholesterol, triglycerides, LDL-C and VLDL-C. On the other hand, serum levels of HDL-C were significantly lower in this group compared to controls. The findings of this study confirm the association between BMI and dyslipidaemia in PCOS. All the above derangements confirm that polycystic ovary syndrome contributes to the development of an atherogenic lipid profile and places the patient at a higher risk of metabolic syndrome. In addition, we observed a significant positive correlation between glucose and total cholesterol, triglycerides, LDL-C, VLDLC along with a non significant negative correlation between uric acid and HDL-C. The above results suggest the association between glucose, uric acid and dyslipidaemia. In conclusion, the use of these simple and cost-effective biochemical parameters might prove to be biomarkers in early etection of these metabolic changes and may help to identify women with PCOS at risk of cardio metabolic syndrome, confirming the association between PCOS and cardiovascular risk factors.

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Conflict of Interest

None

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