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Research Article

Risk of metabolic syndrome in the first degree relatives of women with polycystic ovarian syndrome

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ABSTRACT

Background: Poly cystic ovarian syndrome (PCOS) is a common disorder, affecting approximately 4 – 8% of women in the reproductive age group. First degree relatives (FDRs) of women with PCOS are at high risk developing endocrine and metabolic co-morbidities of PCOS such as obesity, insulin resistance (IR), and impaired insulin sensitivity, hyperlipidemia and metabolic syndrome (MBS). Present study was conducted with the objectives to evaluate the risk of metabolic syndrome in FDRs of women with PCOS and to compare the risk of metabolic syndrome in FDRs of women with PCOS with age, sex and relation matched FDRs of women with no PCOS. To give suitable recommendations based on the findings of the study.
Methods: Hospital based cross sectional analytical study was carried out. 50 FDRs of women with PCOS were compared against same number of age, sex and relation matched FDRs of women with no PCOS.
Results: The prevalence of MBS was significantly higher i.e. 34% among $FDRs_{PCOS}$ compared to $FDRs_{Controls}$ (8%) ($p < 0.05$). The $FDRs_{PCOS}$ were 5.92 times more at risk of MBS than FDRsControls ($p < 0.05$). Relation wise it was found that the $father_{PCOS}$ and $mother_{PCOS}$ were 1.5 times at risk, $brother_{PCOS}$ were 14.26 and $sister_{PCOS}$ were 12.43 times at risk of MBS than their counterparts.
Conclusions: Thus it is concluded that the $FDRs_{PCOS}$ are at high risk of MBS than $FDRs_{Controls}$.

Keywords: Metabolic syndrome, Polycystic ovarian syndrome, Disorder, Obesity

INTRODUCTION

Poly cystic ovarian syndrome (PCOS) is a common disorder, affecting approximately 4 – 8% of women in the reproductive age group.[1]

The classic form of this syndrome includes amenorrhoea, anovulation, infertility, hirsutism, obesity and enlarged bilateral ovaries with cysts. There is an increased risk of diabetes mellitus (DM) and cardiovascular (CVS) diseases. In addition, insulin resistance (IR) and hyperinsulinaemia commonly occur. Lipoprotein abnormalities are also common in PCOS. Other PCOS complications include sleep apnoea and infertility. The high familial incidence of PCOS suggests its genetic origin.[2]

In view of high prevalence of affected individuals within families of PCOS women, a genetic basis for this syndrome has been suggested. This has been evaluated in different population through phenotypic and family aggregation studies.[3]

First degree relatives (FDRs) of women with PCOS are at high risk developing endocrine ad metabolic co-morbidities of PCOS such as obesity, IR, and impaired insulin sensitivity, hyperlipidaemia and metabolic syndrome.[4]

Abnormalities in insulin action and secretion, glucose tolerance, and lipid levels demonstrate familial aggregation in FDRs of women with PCOS. Elevated LDL cholesterol levels are the most consistent lipid abnormality in affected women as well as their sisters and mothers. In addition the prevalence of metabolic syndrome is increased in these groups.[5]

Keeping in view this increased risk of metabolic syndrome in FDRs of women with PCOS, we decided to study the risk of metabolic syndrome in FDRs of women with PCOS.

Early detection of metabolic syndrome in FDRs of women with PCOS is important as metabolic syndrome may predict a higher risk for CVS component. Hence early detection of metabolic syndrome can prevent the risk of CVS diseases in FDRs of women with PCOS. An opportunity for preventive health care exists and may alleviate risks for metabolic and CVS abnormalities in FDRs of women with PCOS.

With this idea, the present study was planned to compare the risk of metabolic syndrome in FDRs of women with PCOS with age, sex and relation matched controls in FDRs of women with no PCOS. This probably will help to establish that FDRs of women with PCOS are at high risk of developing metabolic syndrome.

Another reason that led us to consider to plan this study is that in our literature search of more than 50 studies on PCOS, there are very few studies from Asia and very rare of Indian origin.

Hence present study was undertaken with the main objective to study the risk of metabolic syndrome in FDRs of women with PCOS.

METHODS

Study type and design

Hospital based cross sectional analytical study.

Study population

FDRs of women with PCOS & FDRs of women with no PCOS

Place of study

Malla reddy hospital which is attached to Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana, India.

Duration of study

The study was conducted from January 2014 to October 2014.

Sample size

50 FDRs of women with PCOS were compared against same number of age, sex and relation matched FDRs of women with no PCOS.

Selection criteria

A. For women with PCOS

Inclusion criteria

Known cases of PCOS as diagnosed by Gynecologist

Exclusion criteria

1. Not willing to participate in the study.

B. For FDRs of women with PCOS.

Inclusion criteria

1. First degree relatives only
2. Not on treatment for DM, Hyperlipidemia and HT

Exclusion criteria

1. Not willing to participate
2. Seriously ill

C. For women with no PCOS

Inclusion criteria

1. No history of PCOS
2. Not on treatment for DM, Hyperlipidemia and HT

Exclusion criteria

1. Not willing to participate
2. History of PCOS in their families
3. Seriously ill

D. For FDRs of women with no PCOS.

Inclusion criteria

1. First degree relatives only
2. Not on treatment for DM, Hyperlipidemia and HT

Exclusion criteria

1. Not willing to participate
2. Seriously ill

Ethical considerations

Institutional ethics committee permission was obtained. Informed consent was obtained from the women with PCOS and no PCOS as well as their FDRs if they are more than 18 years of age. If they are less than 18 years of age, then their mother's or father's consent was obtained. The study participants found at risk of metabolic syndrome, were given counseling and health education. Those with abnormal investigation reports were referred to the appropriate department for further management.

Methodology

Women with PCOS as diagnosed by Gynecologists were contacted. Necessary permission from the department was obtained. The nature of study was explained to women with PCOS. Confidentiality was ensured. They were asked to come with their FDRs on a fixed day early morning with 8-10 hours of fasting. The FDRs were included only if they satisfied the selection criteria.

The socio-demographic information, anthropometry and blood pressure were recorded in a pre-designed questionnaire. Then their blood samples were collected by the laboratory technician to be analyzed for lipid profile and fasting glucose. The blood samples were sent to the hospital laboratory and reports were collected.

The study subjects were contacted and asked to collect their reports. At this point of time, they were informed about the risk of metabolic syndrome and CVS diseases based on the reports. Health education was given with respect to adoption of healthy life styles and its importance; nutrition education and its importance. Those with abnormal reports were referred to endocrinologist.

The control group included age, sex and relation matched FDRs of women with no PCOS. A confirmation that these women do not have PCOS was obtained from the Gynecologist. Same procedure as mentioned above for experimental group was followed for this group also.

Metabolic syndrome was defined as follows:

The expert panel on detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III [ATP III]) recently published a consensus definition on insulin resistance syndrome (also termed as metabolic syndrome) based on abnormality in three or more of five component traits as follows.[6]

1. Abdominal obesity (waist circumference)
a. Men > 102 cm
b. Women > 88 cm
2. Blood pressure > 130/ > 85 mmHg
3. Triglycerides > 150 mg/dl
4. HDL Cholesterol
a. Men < 40 mg/dl
b. Women < 50 mg/dl
5. Fasting glucose > 110 mg/dl

Height was measured with the help of a height rod. The person was asked to remove shoes or chappals. The height was measured to the nearest of 0.5 cm. For recording weight, the weight machine was standardized. It was ensured that it is kept on a flat and firm surface with its arrow on zero mark every time. The person was asked to stand with minimum clothing and without shoes or chappals. The weight was recorded to the nearest of 0.5 kg. Based on height and weight, body mass index (BMI) was calculated as weight in kg divided by height in square meter.

Measurement of waist circumference was done with the help of a measuring tape as per the World Health Organizations (WHO) guidelines.[7] Subjects were considered to have abdominal obesity if waist circumference was more than or equal to 102 cm in males and more than or equal to 88 cm in females.[6]

After a rest for a while, blood pressure (BP) was measured with the help of a sphygmomanometer in sitting position, putting the sphygmomanometer at the level of the heart. The procedure for recording BP was followed as per the WHO guidelines.[8] As per this, three readings were obtained at an interval of five minutes and the lowest reading was recorded. For classifying the BP measurement, classification given by WHO was adopted.[8]

Investigations like fasting glucose and lipid profile {total cholesterol (TC), triglycerides (TG), and low density lipoproteins (LDL), high density lipoproteins (HDL)} was done.

Sample collection and methods of estimation

The blood samples (5ml) were collected by venipuncture of cubital vein. For accurate comparison, fasting morning serum samples were obtained. The blood samples were collected in a plain redtop venipuncture tube without additives or anti-coagulants for preparation of serum. The blood was allowed to clot for serum samples. The specimens were centrifuged to separate the serum from the cells and analyzed for Triglycerides (TG), Total cholesterol (TC), HDL cholesterol (HDL-C), LDL cholesterol (LDL-C) by the following methods:

Estimation of fasting glucose

Fasting glucose was determined by GOD-POD method.

Estimation of serum HDL-C

Serum HDL-C was estimated by direct method using Jeev diagnostic kit. The system utilizes a combination of surfactants, phosphoric acid, organic acids, and inorganic acids, specifically binding LDL-C, VLDL-C and

chylomicrons. Only HDL cholesterol was detected by the enzymatic CHOD/ POD method.[9]

Estimation of Serum Triglycerides

Serum triglyceride was estimated by Glycerol phosphate oxidase/ Peroxidase (GPO/POD) colorimetric endpoint method using Jeev diagnostic kit. Triglyceride was hydrolyzed by lipoprotein lipase to free fatty acids and glycerol. Glycerol kinase converts glycerol to glycerol phosphate, which gets oxidized to dihydroxy acetone phosphate and hydrogen peroxide by glycerol phosphate oxidase. Hydrogen peroxide so formed reacts with 4-amino antipyrine in the presence of peroxidase to give a purple colored complex which is read at 505 nm.

Calculation of LDL-C

LDL-C is obtained using Friedwald's formula as given below:[10]

LDL-C = Total cholesterol – (HDL-C + VLDL-C).

TG >150 mg/dl, HDL <40 mg/dl in women and <50 mg/dl in men, LDL >160 mg/dl was considered as abnormal.[6]

Statistical analysis

The data was entered in Microsoft Excel Work Sheet and analyzed using proportions, chi square test, students' "t" test and Odds Ratio wherever applicable.

RESULTS

A hospital based cross sectional case control study was carried out to study the risk of metabolic syndrome among FDRs of women with PCOS. For all descriptive purposes, the first degree relatives of women with PCOS will be termed as FDR_{PCOS} [$Fathers_{PCOS}$, $Mothers_{PCOS}$, $Brothers_{PCOS}$ and $Sisters_{PCOS}$] and first degree relatives of women with no PCOS will be termed as controls [$Father_{Controls}$, $Mother_{Controls}$, $Brothers_{Controls}$ and $Sisters_{Controls}$].

Table 1: Prevalence of metabolic syndrome among cases and controls.

FDRPCOS				Controls		
Metabolic Syndrome	Male	Female	Total	Male	Female	Total
Yes	08 (32)	09 (36)	17 (34)	02 (08)	02 (08)	04 (08)
No	17 (68)	16 (64)	33 (66)	23 (92)	23 (92)	46 (92)
Total	25 (100)	25 (100)	50 (100)	25 (100)	25 (100)	50 (100)

* Figures in the parentheses indicate percentages

Maximum study subjects were in the age group of 15-24 years (48 % FDR_{PCOS} and 46% controls). Minimum were in the age group of 45 – 54 years. Very few study subjects were illiterate i.e. a total of 9%. (14% of FDR_{PCOS} and 4% controls). 38% of study subjects were educated more than Intermediate. Only 2% of study subjects were unemployed. 22% were home maker and 33% were students. No one was found belonging to class V. Only 4% belonged to class IV. Distribution of study subjects in social classes I to III is almost similar ranging from 30 – 35%. This is because; they were matched for relation with index case. Maximum FDRs are brothers (35%) followed by sisters (28%) and mothers (26%). Fathers are only 11%. Prevalence of metabolic syndrome (MBS) is shown in table 1. The prevalence of MBS among FDR_{PCOS} is 34% compared to only 8% among controls. Among male FDR_{PCOS} it is 32% compared to 8% controls and among female FDR_{PCOS}, it is 36% compared to 8% controls. Table 2 shows the risk of MBS in FDR_{PCOS} which is the main objective of the present study. As also mentioned in table 1, the prevalence of MBS among FDR_{PCOS} is 34% compared to only 8% among controls. This difference is found to be statistically significant. (Chi square = 10.2, p = 0.001). It is observed that, the FDR_{PCOS} are 5.92 times more likely to develop MBS than their counterparts. And this risk is also found statistically significant. (Z = 2.962, p = 0.0031).

Table 2: Risk of metabolic syndrome in first degree relatives of women with PCOS.

	Metabolic Syndrome Present	Metabolic Syndrome Absent	Total
FDRPCOS	17 (34)	33 (66)	50 (50)
Controls	04 (08)	46 (92)	50 (50)
Total	21 (21)	79 (79)	100 (100)

* Figures in the parentheses indicate percentages
X2 = 10.2; df = 1; p = 0.001; Odds Ratio = 5.9242; 95% C.I. = 1.8251 – 19.2299; Z = 2.962; p = 0.0031.

Table 3 shows clinical and biochemical characteristics in $brothers_{PCOS}$ and $brother_{Controls}$. Mean age, anthropometric and blood pressure characteristics are almost similar in both the groups (p >0.05). Among biochemical mean

values, FBG, HDL, LDL, and TC are significantly more among $brothers_{PCOS}$ than $brother_{Controls.}$

Table 4 shows clinical and biochemical characteristics in $mothers_{PCOS}$ and $mother_{Controls.}$ Among all the characteristics studied, only BMI is found to be significantly higher among $mothers_{PCOS}$ than $mother_{Controls}$ ($p < 0.05$). For all other characteristics, the difference of mean is not found to be significant ($p > 0.05$).

Table 3: Clinical and biochemical characteristics in $Brother_{PCOS}$ and $Brother_{Controls.}$

	$Brother_{PCOS}$ (18)	$Brother_{Controls}$ (17)	T	p
	Mean±SD	Mean±SD		
Age	21.44±4.24	21.41±4.37	0.0344	0.9728
BMI	22.42±4.10	21.02±2.99	1.1484	0.2591
WC	85±12.83	78.88±9.93	1.5714	0.1256
SBP	116.44±11.55	11.76±6.35	1.4729	0.1503
DBP	80±7.66	75.29±7.17	1.8753	0.0696
FBG	97.38±14.47	86.47±9.10	2.6516	0.012
Triglycerides	129.61±37.3	129.76±54.99	0.0095	0.9925
HDL	37.11±4.6	29±7.936	3.7273	0.0007
LDL	119.27±41.11	87.47±43.88	2.2137	0.0339
VLDL	24.72±80.48	25.58±11.09	0.2586	0.7975
TC	228±44.38	151.23±40.89	5.3131	0.0001

BMI=body mass index, WC=waist circumference, DBP=diastolic blood pressure, SBP=systolic blood pressure, FBG=fasting blood glucose, HDL=high density lipoproteins, LDL=low density lipoproteins, VLDS=very low density lipoproteins, TC=total cholesterol

Table 4: Clinical and biochemical characteristics in $Mother_{PCOS}$ and $Mother_{Controls.}$

	$Mother_{PCOS}$(12)	$Mother_{Controls}$(14)	T	p
	Mean±SD	Mean±SD		
Age	39.16±5.78	39.71±6.79	0.2203	0.8275
BMI	25.53±5.41	22.17±4.26	2.0905	0.0473
WC	82.25±17.92	86.64±10.9	0.7670	0.4505
SBP	110.83±9	115±6.5	1.3683	0.1835
DBP	74.16±9	73.57±8.41	0.1727	0.8644
FBG	103.83±26.46	108.7±68.77	0.2007	0.8426
Triglycerides	115.5±64.17	164.14±89.87	1.5624	0.1313
HDL	38±5.84	36.92±4.66	0.5245	0.6047
LDL	120.41±44.21	134.85±38.48	0.8909	0.3819
VLDL	26.58±16.98	32.85±17.78	0.9150	0.3692
TC	182.08±46.04	204.28±37.16	1.3609	0.1862

BMI=body mass index, WC=waist circumference, DBP=diastolic blood pressure, SBP=systolic blood pressure, FBG=fasting blood glucose, HDL=high density lipoproteins, LDL=low density lipoproteins, VLDS=very low density lipoproteins, TC=total cholesterol

Table 5 shows clinical and biochemical characteristics in $fathers_{PCOS}$ and $father_{Controls.}$ The difference of mean values between $fathers_{PCOS}$ and $father_{Controls}$in all studied parameters is statistically not significant ($p > 0.05$) except mean HDL ($P < 0.05$).

Table 6 shows clinical and biochemical characteristics in $sister_{PCOS}$ and $sister_{Controls.}$Only mean value of FBG is significantly higher in $sister_{PCOS}$ than $sister_{Controls}$ ($p < 0.05$). Difference in mean of all other characteristics is statistically not significant ($p > 0.05$).

DISCUSSION

The present study was conducted with the objective to study the risk of metabolic syndrome (MBS) in FDRs of

women with PCOS. They were compared with age, sex and relation matched FDRs of women with no PCOS.

For all descriptive purposes, the first degree relatives of women with PCOS will be termed as FDR_{PCOS} ($Fathers_{PCOS}$, $Mothers_{PCOS}$, $Brothers_{PCOS}$ and $Sisters_{PCOS}$) and first degree relatives of women with no PCOS will be termed as controls ($Father_{Controls}$, $Mother_{Controls}$, $Brothers_{Controls}$ and $Sisters_{Controls}$).

50, FDR_{PCOS} and age, sex and relation matched 50 controls were studied.

We found that the prevalence of MBS was very high among FDR_{PCOS} i.e. 34% whereas among controls, it was only 8%. This difference was statistically significant. (X^2 = 10.2, p = 0.001). Similarly, the risk of MBS was 5.9 times more for FDR_{PCOS} than controls. This risk is also found statistically significant (Z = 2.962, p = 0.0031).

Table 5: Clinical and biochemical characteristics in $father_{PCOS}$ and $father_{Controls}$.

	$father_{PCOS}$ (6)	$father_{Controls}$ (5)	t	p
	Mean±SD	Mean±SD		
Age	46.33±6.59	45.6±7.09	0.1769	0.8635
BMI	23.20±2.85	23.35±4.49	0.0675	0.9477
WC	81.66±21.07	87±15.93	0.4656	0.6526
SBP	123.33±5.16	120±7.07	0.9040	0.3896
DBP	83.33±10.32	80±0	0.7149	0.4928
FBG	104.16±9.43	93.4±13.14	1.5822	0.1481
Triglycerides	118.16±34.85	157±43.25	1.6711	0.1290
HDL	40±5.93	33±2.64	2.4299	0.0380
LDL	126±41.08	122±43.15	0.1572	0.8785
VLDL	25±10.27	31±8.86	1.0248	0.3322
TC	184.66±44.86	186±39.22	0.0536	0.9548

BMI=body mass index, WC=waist circumference, DBP=diastolic blood pressure, SBP=systolic blood pressure, FBG=fasting blood glucose, HDL=high density lipoproteins, LDL=low density lipoproteins, VLDS=very low density lipoproteins, TC=total cholesterol

Table 6: Clinical and biochemical characteristics in $sister_{PCOS}$ and $sister_{Controls}$.

	$sister_{PCOS}$ (14)	$sister_{Controls}$(14)	T	P
	Mean±SD	Mean±SD		
Age	22.64±6.40	21.85±4.31	0.3831	0.7048
BMI	19.35±3.7	18.17±2.92	0.9367	0.3575
WC	75.42±13.91	78±11.3	0.5387	0.5947
SBP	110.71±11.41	105±5.18	1.7050	0.1001
DBP	76.42±12.15	68.57±9.49	1.9052	0.0679
FBG	94.21±12.87	85.57±6.42	2.2477	0.0333
Triglycerides	118.14±38.64	139.21±62.17	1.0770	0.291
HDL	36.14±5.85	32.21±4.44	2.0022	0.0558
LDL	112.92±27.96	97.42±25.65	1.5285	0.1385
VLDL	23.5±7.6	27.5±12.42	1.0243	0.3153
TC	172.64±29.58	157.14±24.37	1.5132	0.1423

Shabir I et al also reported from Kashmir that the prevalence of MBS was 22% among FDR_{PCOS}. Akbarzadeh et al also observed a high prevalence of MBS as 29.35% in $fathers_{PCOS}$ compared to only 8.8% of fathers of women in the control group.[2,11]

These findings suggest that the high prevalence of MBS in FDR_{PCOS} is due to familial aggregation.

We observed that, the prevalence of MBS was 50% in $fathers_{PCOS}$ and 40% in $father_{Controls}$.

Shabir I et al revealed very high prevalence i.e. 56%. Coviello et al observed that the prevalence of MBS was 42% in $fathers_{PCOS}$ and 32% in $father_{Controls}$.[11,12] Akbarzadeh et al observed that the prevalence of MBS was 29.35% in $fathers_{PCOS}$ as against 8.8% in $father_{Controls}$. Gabrielli L et al reported prevalence of diabetes (DM) and impaired glucose tolerance (IGT) as 27% and 31% among $father_{PCOS}$.[2,13]

Prevalence of MBS is 41.6% in $mother_{PCOS}$ compared to 14.28% in $mother_{Controls}$ in the present study.

Shabir I et al reported almost double i.e. 80% prevalence of MBS in $mother_{PCOS.}$ Gabrielli L et al observed that the prevalence of DM & IGT was 16% and 30% respectively in $mother_{PCOS}$, and that of impaired fasting glucose (IFG) was 3% in $mother_{PCOS.}$[11,13]

In the present study, among $brother_{PCOS}$ the prevalence of MBS was 27.7% compared to nil among $brother_{Controls.}$

But Shabir I et al did not find any $brother_{PCOS}$ with MBS. Whereas Coviello et al observed this rate as 22% in $brother_{PCOS}$ compared to only 9% among $brother_{Controls.}$ Gabrielli L et al found no DM in $brother_{PCOS}$ but IFG was present in 4% of $brother_{PCOS.}$[11-13]

We observed that the prevalence of MBS in $sister_{PCOS}$ was 28.57% and no case of MBS in $sister_{Controls}$.

Shabir I et al also reported that 25% of $sister_{PCOS}$ were having MBS. Bulent O et al found no DM in $sister_{PCOS}$ but reported that prevalence of IGT was 5%.[11,13]

In the present study, we compared the mean values of clinical and biochemical parameters among FDR_{PCOS} and $FDR_{Controls.}$

For brothers, we found that FBG, HDL, LDL & TC mean values were significantly higher in $brother_{PCOS}$ than $brother_{Controls.}$ But the difference in mean values of all other parameters was not significant statistically ($p > 0.05$).[5] Susan Sam et al found that SBP was similar among $brother_{PCOS}$ and $brother_{Controls}$ but the difference in DBP was significant. Akbarzadeh et al found no statistically significant difference for blood pressure which is similar to our findings.[14] Susan Sam et al reported no significant difference of mean BMI and WC and we also observed the same.[5] But Shabir I et al reported a prevalence of 73.6% of BMI > 23 kg/m^2 in $brother_{PCOS.}$[11] Likewise, Coviello AD et al also found higher BMI in $brother_{PCOS}$.[12] For $brother_{PCOS}$ we found that mean value of TC, LDL, HDL& FBG is significantly higher. Susan Sam et al also reported that the mean value of TC, LDL higher, similar to our study but TG was also higher in $brother_{PCOS}$ which is not significantly higher in the present study.[5] For HDL, they found no significant difference. Shabir I et al observed that TG $\geq$ 150 mg/dl was present in 42% of $brother_{PCOS.}$[11] Similar to our study findings, Murat Y et al also reported that difference in mean of age, BMI, WHR, SBP, DBP was not significant and that of TC, LDL was significant for brothers.[15]

Mean difference in clinical and biochemical characteristics for mothers in the present study revealed that except higher significant BMI value in $mother_{PCOS,}$ all other characteristics were similar for $mother_{PCOS}$ and $mother_{Controls.}$ Akbarzadeh found that the average blood pressure was high among $mother_{PCOS.}$[14] SBP >130 mmHg and DBP >85 mmHg was present in 47% each of $mother_{PCOS}$ in a study by Shabir I et al.[11] They also found that the prevalence of BMI >23 kg/m^2, fasting glucose >100 mg/dl, and TG $\geq$150 mg/dl was 95.4%, 12% and 76% respectively. Murat Y et al revealed that age, BMI, HDL mean value were not significantly different but mean WHR, SBP, DBP, TC, LDL, TG were significantly higher in $mother_{PCOS.}$[15] He also found that prevalence of glucose intolerance was 40% among $mother_{PCOS.}$

For fathers, all characteristics were almost similar ($p > 0.05$) except HDL, which was significantly higher among $father_{PCOS.}$

Whereas Murat Y et al reported that except age and BMI, all other characteristics were significantly higher in $father_{PCOS.}$[15] Akbarzadeh M et al revealed that average blood pressure was significantly more in $father_{PCOS.}$[14] Shabir I et al reported that the prevalence of SBP >130 mmHg, DBP >85 mmHg, BMI >23 kg/m^2, IFG >100 mg/dl, TG $\geq$ 150 mg/dl, was 45%, 45%, 82.6%, 20%, and 65% respectively among $father_{PCOS.}$[11] Akbarzadeh et al also reported that 14.7% of $father_{PCOS}$ were hypertensive and 29.4% were diabetics and 17.6% had high total cholesterol levels.[2] Coviello AD et al[12] found higher BMI in $father_{PCOS.}$ Murat Y et al observed that 52% of $father_{PCOS}$ were having glucose intolerance.[15]

In the present study, among sisters, only mean FBG was significantly higher among $sister_{PCOS}$ than $sister_{Pontrols.}$ All other characteristics mean was not significantly different. Murat Y et al also found that age, BMI, WHR, SBP, DBP, TC, LDL, HDL, TG were similar for $sister_{PCOS}$ and $sister_{Pontrols}$ and this finding is similar to our study.[15] But Akbarzadeh et al found that average blood pressure was higher in $sister_{PCOS.}$[11] Shabir I et al reported the prevalence of SBP > 130 mmHg, DBP > 85 mmHg, BMI > 23 kg/m^2, IFG > 100 mg/dl, TG $\geq$ 150 mg/dl, as 15%, 15%, 77.2%, 12%, and 45% respectively for $sister_{PCOS.}$[14]

Thus from the present study, it is clear that the $FDRs_{PCOS}$ are at high risk of having MBS than their counterparts. Some findings in the present study are not found to be significant, especially when the comparison was made between individual relationships. This may be due to a very small sample of fathers etc. But jointly, when compared with controls, $FDRs_{PCOS}$ are significantly at high risk of MBS.

CONCLUSION

It was found that the prevalence of metabolic syndrome among FDRsPCOS is significantly very high i.e. 34% vs 8% in FDRsControls. The risk of MBS is significantly high for $brothers_{PCOS}$ and $sisters_{PCOS}$ but not for $fathers_{PCOS}$ and $mothers_{PCOS}$. This study warrants further research into prevalence of MBS and other androgenic abnormalities among FDRsPCOS. This will help establish that screening should be carried out for FDRs of women who are diagnosed as having PCOS in any hospital.

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