Original Research Article

Analysis of lipid profile in various phenotypes of Polycystic ovary syndrome at Tertiary Care facility: An observational study

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Abstract

Objective- To assess the sociodemographic and lipid profile (Total cholesterol, Triglycerides and HDL) of various phenotypes of woman with PCOS. **Methodology-** This observational study was conducted at Tertiary care facility, Jaipur from July 2020 to December 2021 on cases of PCOS aged between 18-35 years attending Gynaecology OPD clinic. 90 women with PCOS, fulfilling the inclusion and exclusion criteria, were enrolled in the study. They were divided into four subgroups- A, B, C and D phenotype. Demographic characteristics, Anthropometric measurementwere done and lipid profile was noted and analysed using SPSS software. **Result-** Most of cases 38(42.2%) showed D phenotype, followed by A phenotype in 32(35.6%) cases, C phenotype in 12(13.3%) and B phenotype in 8(8.9%) cases. BMI was higher in phenotype A as compared to phenotype D.Mean levels of cholesterol and Triglycerides were significantly higher and mean HDL level was significantly lower in cases with phenotype B as compared to other phenotypes (p value<0.05). **Conclusion-** It can be concluded that there are raised levels of triglycerides and decreased levels of HDL in androgenic phenotype of PCOS (A, B & C).Women with PCOS should be counselled regarding life style modification including exercise and weight reduction for prevention of cardiovascular events due to dyslipidaemia.

Keywords- Dyslipidaemia, HOMA-IR, PCOS, VAI

Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine disorder in women of reproductive age, with a prevalence of 5–16% across worldwide[1]. The diagnosis of PCOS as based on the Rotterdam criteria, includes two of the following three features:" oligo-/amenorrhea, hyperandrogenism (clinical or biochemical) and polycystic ovaries on ultrasound, after exclusion of other endocrinopathies[2].

The Rotterdam diagnostic criteria have led to the generation of four distinct phenotypic subgroups: the PHO subgroup (phenotype A) with all three diagnostic features present; the HO subgroup (phenotype B) with hyperandrogenism and oligo-/amenorrhea, the PH subgroup (phenotype C) with polycystic ovaries on ultrasound and hyperandrogenism and the PO (phenotype D) subgroup with polycystic ovaries on ultrasound and oligo-/amenorrhea.2 Women with PCOS have greater menstrual irregularity, hyperandrogenism, total and abdominal obesity, and insulin resistance (IR) and have more severe risk factors for type 2 diabetes mellitus and cardiovascular disease. Among these, dyslipidaemia is a common phenomenon observed in women with PCOS[3]. Dyslipidemia has a potential impact on metabolic profile,exert a clear effect on oocyte quality and promote hyperandrogenism which is the basic pathophysiology of PCOS.So, lipid abnormality can be considered as an earliest variation in high risk PCOS women[4].

This study was conducted to assess the sociodemographic and lipid profile (Total cholesterol, Triglycerides and HDL) in various phenotypes of PCOS.

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Methodology

This observational study was conducted in the department of Obstetrics and Gynaecology at S.M.S Medical College, Jaipur on cases of PCOS aged between 18-35 years attending Gynaecology OPD clinic. Sample of 90 cases of PCOS, fulfilling the inclusion and exclusion criteria, were enrolled in the study.

Inclusion criteria

Women between 18 to 35 years who are willing to participate in the study.

Those following the Rotterdam criteria[5] (two out of the three): -

1. Ovulatory dysfunction such as oligomenorrhea or amenorrhea.
2. Clinical or biochemical evidence of hyperandrogenism (excluding other endocrine cause of hyperandrogenism as congenital adrenal hyperplasia, Cushing syndrome, adrenal adenoma)
3. Polycystic ovarian morphology on USG scan defined as presence of 12 or more cyst in size in any one ovary or both ovaries with enlarged ovaries.

Exclusion criteria

1. Hyper and hypothyroidism
2. Gonadal dysgenesis
3. Women on oral contraceptives or other hormonal treatment
4. Hypothalamic dysfunction
5. Uterine Causes for oligo/amenorrhea-Asherman syndrome
6. Pregnant female

After proper counselling regarding the purpose of the study, a written informed consent was taken from women. A detailed history including personal, past, menstrual, obstetrics and medication history was taken. Evaluation of menstrual history determined the extent of menstrual cycle disturbances. Oligo and anovulation were defined as cycle lasting for >35 days and no cycles in the past six months respectively.

Following that, a thorough general physical and systemic examination was done. Evidence of hyperandrogenism like

hirsutism, acne etc were noted. Terminal hair growth on nine regions of the body was used to calculate Ferriman-Gallwey score[5]. Secondary sexual characters were examined and systemic per abdominal and prevaginal examination was done.

A per abdominal sonography for whole abdomen and a transvaginal sonography was done. It was done to diagnose Polycystic ovarian morphology. Polycystic ovary on transvaginal ultrasound is defined as 12 or more follicles measuring 2-9 mm in diameter and ovarian volume >10 mm^2.

Demographic characteristics like Age, Residence, Occupation and Socioeconomic status were noted. Anthropometric measurement like weight, height and body mass index were noted for all enrolled women. Height and weight were recorded by standard methods. Height was measured to the nearest 0-1 cm using a wall mounted stadiometer. Patients were weighed in light clothing without shoes. BMI (body mass index) was defined as body weight in kilograms divided by height in meter square. Lipid profile was estimated by using enzymatic colorimetric technique. Lipid profile was considered abnormal if cholesterol >200 mg/dl; triglycerides ≥150 mg/dl; and HDL < 40 mg/dl.

Women enrolled were divided into four phenotypes. These four phenotypes were-

A- All three diagnostic features present (P+H+O)

B- Hyperandrogenism and oligo-/amenorrhea (H+O)

C- Polycystic ovaries and hyperandrogenism (P+H)

D- Polycystic ovaries on ultrasound and oligo-/amenorrhea (P+O)

Statistical analysis

The data was entered in a predesigned proforma in excel spread sheet (version Microsoft office 19). Nominal/categorical variables were summarized as proportions and were analyzedby using Chi square test. Continuous variables were summarized as Mean and Standard Deviation. ANOVA test was used for analysis of continuous variables. The software used in the analysis was Statistical Package for Social Sciences (SPSS 25.0) and p-valueless than 0.05 was considered as level of significance.

Result

In the present study, maximum i.e. 63(70%) women were of 20–29 years age group. Mean age of cases was 26.19±4.17 years. Majority 69(76.7%) of cases were Hindu and the rest 21(23.3%) were Muslim. Around one third i.e. 58(64.4%) cases were from urban area and the rest 32(35.6%) cases were from rural area. Maximum i.e. 81(90%) cases were literate and the rest 9(10%) cases were illiterate.

(Figure-1) When these women were divided in various phenotypes groups, it was found that 38(42.2%) cases showed D phenotype, A phenotype was seen in 32(35.6%) cases, C phenotype in 12(13.3%) and B phenotype in 8(8.9%) cases.

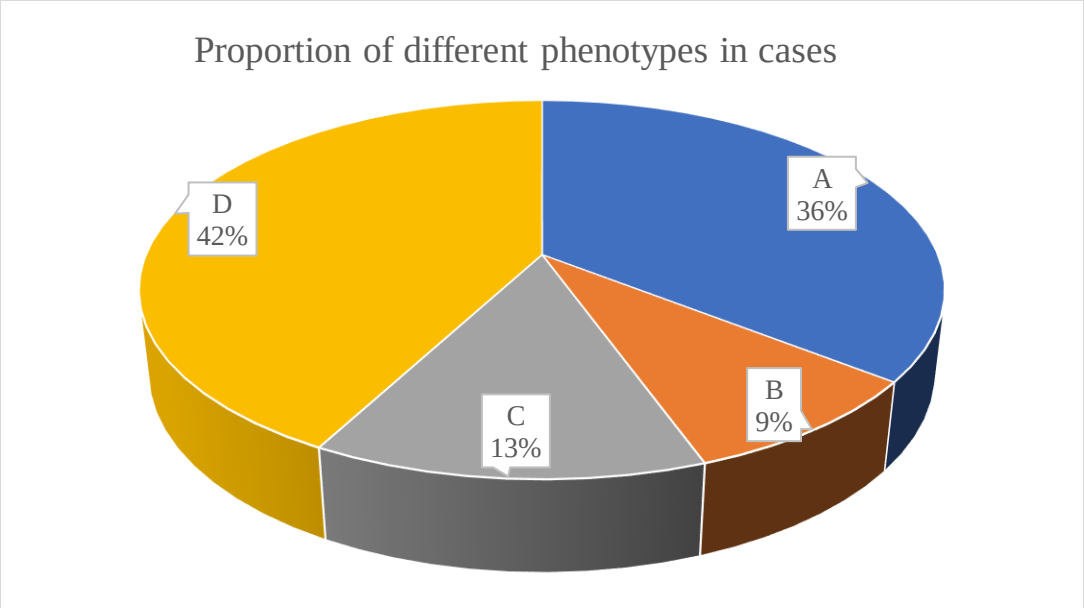

Figure 1- Distribution of cases according to Phenotype of PCOD

(Table 1) It was observed that BMI was higher in phenotype A as compared to phenotype D. The difference in BMI levels of different phenotypes was statistically significant (p value<0.05)

Table 1- BMI in various phenotypes of PCOS

BMI	Phenotypes			
	A	B	C	D
18.5-24.9	2(6.3)	-	3(25)	21(55.3)
25-29.9	22(68.8)	6(75)	7(58.3)	12(31.6)
≥30	8(25)	2(25)	2(16.7)	5(13.2)
Total	32(100)	8(100)	12(100)	38(100)

Chi-square = 24.339 with 6 degrees of freedom, P<0.001

(Table 2) Mean levels of cholesterol and Triglycerides were higher in cases with phenotype B as compared to other phenotypes. This difference in mean cholesterol and triglycerides of phenotype B and other phenotypes was found to be significant (p value<0.05). Mean HDL was lowest in cases of phenotype B while maximum in cases of phenotype A and this difference in mean HDL of cases with different phenotypes was found to be insignificant (p value>0.05)

Table 2- Mean Cholesterol, Triglycerides and HDL among cases of different phenotypes

Variables	Phenotype (Mean Values)				Test of significance
	A (n=32)	B (n=8)	C (n=12)	D (n=38)	
Cholesterol (mg/dl)	177.58±47.74	210.08±67.15	164.22±23.04	163.53±27.63	F=3.365, Df=3, p value=0.022
Triglycerides (mg/dl)	125.61±31.59	130.74±46.95	103.68±27.94	100.23±25.74	F=5.343, Df=3, p value=0.002
HDL (mg/dl)	48.61±10.51	41.19±14.05	45.18±12.92	44.86±9.41	F=1.329, Df=3, p value=0.270

Discussion

PCOS is one of the common endocrine abnormality affecting women. It is divided in various phenotypes according to the presence of diagnostic features. BMI and lipid profile values show variations in the different phenotypes.

When the percentage division into various phenotype groups is compared with previous studies, it is seen that similar observations were reported by Zhang Hy et al (2013)[6] study where prevalence of phenotype D (52.2%) was most common followed by A (26.8%), C (13.4%) and B (7.6%) was the most uncommon. In contrast to the findings of present study, a study conducted by OlgierdGluszak et al (2012)[7] showed the maximum prevalence of phenotype A (60.2%), followed by phenotype C (18.3%), phenotype B (16.1%) and least was for phenotype D (5%). Kar S (2013)[8] also reported that percentage phenotype A was maximum among the study subjects (65.6%) followed by phenotype D (22.2%), phenotype C (11.2%) and phenotype B (0.9%).

Maximum number 22(68.8%) of phenotype A cases had BMI between 25-29.9, while among phenotype D cases most 21(55.3%) cases had BMI between 18.5-24.9. Phenotype A shows all the three manifestations of PCOS. It can be suggested that increased weight has a direct correlation in the development of these manifestations.Chae SJ et al (2008)[9] study concluded that phenotype A (22.8) and B (21.9) showed higher BMI than phenotype D (20.9). These results were in concordance to our study.

Mean levels of cholesterol and Triglycerides were higher in cases with phenotype B as compared to other phenotypes,these were lowest in phenotype D. It was also observed that women with hyperandrogenic and classical PCOS phenotype (phenotype A, B and C) had higher cholesterol and triglyceride levels but lower HDL levels as compared to non -hyperandrogenic phenotype (phenotype D). Thus, it can be said that women with hyperandrogenic PCOS (phenotype A, B, C) have an increased risk of development of metabolic syndrome and cardiovascular diseases as compared with phenotype D.

A study conducted by OlgierdGluszak et al on women with PCOS (2012)[7] reported that there were no statistical differences between the phenotype B, C, D if total Cholesterol, Triglyceride and HDL levels were considered. However, the level of total Cholesterol was significantly higher in group A as compared to other three groups in their study.

Atles S et al (2013)[10] observed that the levels of LDL were higher in phenotype A and D as compared to phenotype B ($p<0.05$). Mean HDL levels were lower in phenotype A and highest in phenotype D ($p<0.23$)

Pikee S et al (2016)[11] also studied various phenotypes of PCOS and found that maximum PCOS cases with phenotype B had cholesterol levels more than 200mg/dl. Triglyceride levels also followed the same pattern as the levels were maximum in phenotype B of PCOS. These results are in accordance to that seen in the present study.

Conclusion

Based on the results of the study, it can be concluded that there are raised levels of triglycerides and decreased levels of HDL in androgenic phenotype of PCOS (A, B & C). This can be attributed to disturbances in the anti-inflammatory and antioxidant protection thereby causing atherogenic dyslipidaemia. Women with PCOS should be counselled regarding life style modification including exercise and weight reduction for prevention of cardiovascular events due to dyslipidaemia.

Conflict of Interest: Nil Source of support: Nil

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