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A COMPARATIVE STUDY ON PROFILE OF INSULIN RESISTANCE IN VARIOUS PHENOTYPE OF PCOS AT SMS MEDICAL COLLEGE, JAIPUR RAJASTHAN

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Conflicts of Interest: Nil

ABSTRACT:

Background- Hyperinsulinemia and insulin resistance (IR) are thought to be key pathological factors for PCOS.

Methods- This prospective case-control study was undertaken to assess the insulin resistance (HOMA-IR). 216 women newly diagnosed with PCOS were classified into one of the four potential PCOS phenotypes based on history, examination and investigations.

Results- Mean HOMA-IR value was maximum in phenotype B (5.31 ± 2.13) followed by phenotype A (4.41 ± 2.21), phenotype C (3.15 ± 1.26) and minimum in phenotype D (3.08 ± 1.52) and controls (1.38 ± 0.98). Statistically significantly higher value of HOMA-IR was seen in all phenotype of PCOS with respect to controls (p-value <0.001). Phenotype A and B also showed statistically significant difference with respect to phenotype D (p<0.001) in relation to HOMA-IR.

Conclusion- Phenotype (A and B) were more insulin resistant, had higher fasting insulin and HOMA-IR values compared to women of phenotypes C and D.

Keywords: Insulin resistance, Polycystic ovary syndrome, Phenotypes.

Introduction

The polycystic ovary syndrome (PCOS) is an endocrine metabolic disorder affecting 7% to 10% of women of reproductive age. It is one of the main causes of infertility resulting from chronic anovulation. This syndrome was first described in 1935 by Stein-Leventhal who observed among some patients menstrual disorders and polycystic ovaries (the "billiard ball" sign on ultrasound examination). [1]

Hyperinsulinemia and insulin resistance (IR) are thought to be key pathological factors for PCOS. The association between IR and PCOS has important clinical ramifications, particularly as IR is thought to be the uniting pathogenic factor in the associations between hypertension, glucose intolerance, obesity, lipid abnormalities and coronary artery disease, which together constitute metabolic syndrome (MetS) or syndrome 'X.'[2]

The newer phenotypes generated by the Rotterdam criteria are inadequately studied and reported. This study was undertaken to characterize the various phenotypes of PCOS, assess their distribution and to report the prevalence and risk factors for metabolic syndrome. The results will highlight the importance of early and regular screening in these women so that appropriate steps can be taken at the right stage to avoid full blown complications later in life.

STUDY TYPE

A case control prospective type of study.

STUDY PERIOD

From April 2017 to Oct 2018.

STUDY LOCATION

Hospital based study conducted in the department of Obstetrics & Gynaecology, S.M.S. Medical College, Jaipur.

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STUDY POPULATION

Study population (Infertile women of age group 18 to 38 years) was divided into two groups: -
Case- Infertile PCOS women
Control- Infertile Non PCOS women

SAMPLE SIZE

Sample size was calculated at 90% confidence level assuming prevalence of type B PCOS in 11.37% of patient as per reference study. A the precision of 5% (absolute allowable error) minimum 156 patients of PCOS and 50 controls were required at sample size for comparison of clinical, biochemical and other variables.

INCLUSION CRITERIA

Case - Infertile Women of reproductive age (18-38 years) group who were willing to participate in the study, diagnose as having PCOS according to ESHRE/ASRM ROTTERDAM CRITERIA 2003.
Control – Women of the same age group visiting OPD with complaint of infertility unrelated to PCOS, thyroid or prolactin dysfunction.

EXCLUSION CRITERIA

Infertile Women of reproductive age (18 to 38 years) who were having: -

- Hypothyroidism,
- Hyperprolactinaemia,
- Congenital adrenal hyperplasia
- Androgenic tumors,
- Cushing disease,
- Women on medication for ≤ 6 months prior to the study eg- hormonal therapy or medication for dyslipidemia.

STATISTICAL ANALYSIS

Data collected was entered in MS Excel sheet. Qualitative data was expressed as proportion and percentage and Quantitative data was expressed as mean and standard deviation. Qualitative data was analysed by using χ^2 test and quantitative data was analysed by using ANOVA test and unpaired 't' test (significant was set at $p<0.05$ and highly significant at $p<0.001$).

OBSERVATION

Table 1: ***Profile of Insulin Resistance of Various Phenotypes and Controls***

Parameters	PCOS Cases				Controls	p-value
	Phenotype A	Phenotype B	Phenotype C	Phenotype D		
F. Insulin (µU/ml)	24.18±9.12	27.08±8.26	13.89±6.52	13.76±6.34	6.35±1.55	0.001
2 hr Insulin (µU/ml)	68.84±18.13	87.44±14.98	67.15±16.24	49.83±15.88	10.08±3.29	0.001

In our study, mean fasting hyperinsulinemia was maximum in phenotype B (27.08±8.26) followed by phenotype A (24.18 ± 9.12), phenotype C (13.89 ± 6.52) and least in phenotype D (13.76 ± 6.34) ($p<0.001$). Apart from controls, phenotype A and B, both group also showed statistically significant higher value with respect to phenotype D ($p<0.001$).

Post-prandial mean hyperinsulinemia was highest in phenotype B (87.44 ± 14.98) followed by phenotype A (68.84 ± 18.13), phenotype C (67.15 ± 16.24) and lowest in phenotype D (49.83 ± 15.88). Phenotype A, B and C also showed statistically significant higher value with respect to phenotype D; ($p<0.001$ for each, statistically significant difference was found) in relation to 2 hour insulin levels.

Table 2: ***Profile of Insulin Resistance of Various Phenotypes and Controls***

Parameters	PCOS Cases				Controls	p-value
	Phenotype A	Phenotype B	Phenotype C	Phenotype D		
HOMA IR	4.41±2.21	5.31±2.13	3.15±1.26	3.08±1.52	1.38±0.98	0.001

Mean HOMA-IR value was maximum in phenotype B (5.31 ± 2.13) followed by phenotype A (4.41 ± 2.21), phenotype C (3.15 ± 1.26) and minimum in phenotype D (3.08 ± 1.52) and controls (1.38 ± 0.98). Statistically significantly higher value of HOMA-IR was seen in all phenotype of PCOS with respect to controls (p-value <0.001). Phenotype A and B also showed statistically significant difference with respect to phenotype D (p<0.001) in relation to HOMA-IR.

Discussion

It was a prospective case control study conducted in Department of Obstetrics and Gynaecology, SMS Medical College and attached group of hospitals, Jaipur from April 2017 to October 2018.

Out of 206 infertile women, 161 had PCOS (cases) and 50 were non –PCOS (controls) in this study. PCOS was further classified in four phenotypes according to Rotterdam criteria.

Mean HOMA-IR value was maximum in phenotype B (5.31 ± 2.13) followed by phenotype A (4.41 ± 2.21), phenotype C (3.15 ± 1.26) and minimum in phenotype D (3.08 ± 1.52) and controls (1.38 ± 0.98). Statistically significantly higher value of HOMA-IR was seen in all phenotype of PCOS with respect to controls (p-value <0.001). Phenotype A and B also showed statistically significant difference with respect to phenotype D (p<0.001) in relation to HOMA-IR.

In the study conducted by Baldani DP et al (2013)[3], serum levels of glucose were not found to be different between groups (A, B, C, D) but higher levels of insulin, GIR and HOMA-IR were found between phenotype A and control group (p < 0.001).

Whereas, insulin resistance was higher in phenotype B in the study by Welt CK et al (2006)[4] as well as in the study conducted by Panidis D et al (2012)[5]. Similar results were found in our study.

Yilmaz M et al (2011)[6] found prevalence of metabolic syndrome and degree of insulin resistance in phenotype D was closer to control subjects than the other three phenotypes. They concluded that anthroprometrical, hormonal, and metabolic differences suggest that phenotype D is closer to control group. Similar results were found in our study.

Study conducted by Zhang HY et al (2009)[7] observed no differences in the fasting glucose among the four phenotype group. But fasting insulin levels and HOMA–IR were highest in phenotype B compared with phenotype D and controls (p <0.001), corresponding results were found in our study.

In the study of Chae SJ et al (2008)[8] results showed higher fasting insulin levels (p-value<0.001) and postprandial 2 h insulin (p-value<0.001) were noted in phenotype A and phenotype B, compared with phenotype D. Similar results were found in our study.

Ates S et al (2013)[9] study reported that there were no significant differences in the fasting glucose levels among various phenotypes of PCOS (p<0.039). But mean fasting insulin and mean HOMA–IR values were higher in phenotype A, B, C and lowest in phenotype D and controls (p<0.027; p <0.171 not statistically significant).

Study conducted by Pikee S et al (2016)[10] levels of fasting and postprandial insulin were significantly higher in all phenotypes of PCOS with respect to controls (p<0.000 and p<0.009, respectively). Fasting glucose to fasting insulin ratio was significantly lower in all phenotypes of PCOS and compared to controls (p<0.02).

Our study also suggested the same finding and correlated with the previous studies indicating that the cases with classical form of PCOS (phenotype A and B) were more insulin resistant as compared to the cases of non classic phenotype C and D. The prevalence of insulin resistance and glucose intolerance was not significantly different between phenotype D and controls.

Conclusion

Study subjects with classic phenotype (A and B) were more insulin resistant, had higher fasting insulin and HOMA-IR values compared to women of phenotypes C and D.

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