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**Original Research Article** 

# Study of Insulin Resistance and Lipid Profile in Obese and Non Obese in Polycystic Ovarian Syndrome (PCOS)

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#### Abstract

**Background & objectives:** Polycystic ovary syndrome (PCOS) is the most common disorder of ovarian pathology in women of reproductive age group Insulin resistance and hyperinsulinemia may play an important role in pathophysiology of PCOS. To estimate fasting plasma glucose, plasma insulin levels, insulin resistance and lipid profile in obese and non obese female infertile women with PCOS. To study the association between insulin resistance, lipid profile and obesity in female infertility in PCOS. *Methods:* 30 obese infertile women diagnosed by ultrasonogram as PCOS and with a BMI>  $30 \text{Kg/m}^2$  attending Gynecology OPD at Narayana medical college and hospital were taken as cases. 30 age matched non obese infertile women with a BMI< $30 \text{Kg/m}^2$  were taken as controls. *Results:* In our study the fasting glucose, fasting plasma insulin resistance was significantly elevated among cases when compared to controls (P value <0.001). *Interpretation & conclusions:* This clearly shows the importance of screening for dyslipidemia and insulin resistance in Indian Women with PCOS. Hence periodic monitoring of lipid profile and B.P specially for obese PCOS patients may be advised.

Keywords: Insulin Resistance, Lipid Profile, Obesity, Polycystic Ovary Syndrome.

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# **INTRODUCTION**

The polycystic ovarian syndrome (PCOS) is the most common endocrine disorder affecting female fertility and menstrual disorders mostly due to anovulation [1]. Obesity is highly prevalent in the general population and in PCOS women and is an independent risk factor for CAD [2]. Obesity in adolescents is correlated with insulin resistance (IR) and dyslipidemia [3]. PCOS related ovulatory dysfunction in adolescents often correlates to adolescent obesity [4].

In India among the age group of 20-40 years indicated that the prevalence rate of obesity was 31%. Out study showed a 37.5% prevalence rate of obesity in women with PCOS [5].

Multiple components of insulin signaling path ways (insulin receptor, insulin related receptor & IGF-1 receptor). Insulin resistance is a condition in which a normal concentration of insulin produces a less than normal biological response [6].

Diabetes mellitus and impaired glucose tolerance are associated with insulin resistance and obesity. Insulin sensitivity is influenced by a number of factors including age, dietary factors, obesity, ethnicity, body fat, modifications in lifestyle and medications [7]. During peripheral insulin resistance, circulating insulin concentrations rise high enough to activate IGF–1 receptors [8].

Insulin resistance and hyperinsulinemia may play an important role in PCOS pathophysiology. Women with PCOS have a higher prevalence and a greater degree of hyperinsulinemia and insulin resistance than weight-matched control subjects [9].

Insulin resistance develops as a consequence of excess accumulation of fat in liver and skeletal muscles. Free fatty acid level increases, exceeds the capacity of mitochondrial oxidation and spillover to cytoplasm where it is reesterified [10]. The consequent increase in Diacylglycerol, a second messenger, leads to activation of Protein Kinase C. Phosphorylation of Insulin Resistance Substrate by Protein Kinase C (PKC) reduces the normal signal transduction pathway by insulin and leads to insulin resistance [11].

Hyperinsulinemia probably acts at the level of hypothalamic-pituitary axis and stimulates LH secretion which leads to anovulation with irregular menstruation [12]. It decreases the production of sex hormonebinding protein and IGF-1-binding protein in liver, which results in an increase in free androgen in the blood and an increase in free IGF-1 in the ovary [13].

Hence, this study was undertaken to assess the insulin resistance and lipid profile in obese and non obese female infertile women.

#### **MATERIALS & METHODOLOGY**

30 obese infertile women diagnosed by ultrasonogram as PCOS and with a BMI>  $30Kg/m^2$  attending Gynecology OPD at Narayana medical college and hospital were taken as cases. 30 age matched non obese infertile women with a BMI< $30Kg/m^2$  were taken as controls. The study was approved by institutional ethical committee and an informed written consent was obtained from all the subjects. 5 ml of fasting blood sample was collected by venepuncture and allowed to clot for serum separation. The serum is

used for the estimation or detection of the biochemical parameters.

#### **Biochemical Parameters**

estimated Plasma insulin was by chemiluminiscence method using Beckman coulter Access II with ultrasensitive Insulin Reagent Pack (50 tests/ pack) with Cat.No.33410: 100 determinations, 2 packs was used, Fasting glucose, TGL, Total Cholesterol were estimated by enzymatic method in autoanalyser humastar 600. HDL was estimated by kit method using humastar 300, GMBH, GERMANY. LDL cholesterol was estimated by Friedwald equation. HOMA IR method was used to calculate insulin resistance. (HOMA IR= Plasma insulin mIU/LXPlasma glucose mg/dl / 405).

# RESULTS

Continuous variables were compared using SPSS. Mean; standard deviation and P value were calculated using the soft ware. P value < 0.05 was considered significant. In our study the fasting glucose, fasting plasma insulin values and insulin resistance was significantly elevated among cases when compared to controls (P value <0.001). The Mean  $\pm$  S.D values of LDL cholesterol were elevated in cases when compared to controls. (P value <0.001) The decrease in HDL cholesterol was statistically significant among cases when compared to controls. However, the increase in total cholesterol was not significant statistically (P value <0.17).

S. NO	VARIABLES	Cases	Cases	Controls	Controls	p VALUE
		MEAN	SD	MEAN	SD	
1	AGE	32.73	4.4	28.6	4.17	< 0.001
2	HDL	42.06	3.28	46.3	4.23	< 0.001
3	LDL	122.6	7.12	96.48	18.3	< 0.001
4	TOTAL CHOLESTEROL	168.56	8.05	159.3	18.97	< 0.17
5	TRI GLYCERIDES	156.93	8.36	119.96	14.13	< 0.001
6	FASTING GLUCOSE	131.96	8.9	93.93	12.2	< 0.001
7	FASTING INSULIN	15.5	5.88	5.1	1.68	< 0.001
8	HOMA-IR	5.11	2.13	1.2	0.45	< 0.001
9	BMI	34.6	2.89	23.8	2.1	< 0.001

#### Table-1: Mean ± SD values of the main characteristics of obese and non obese infertile women with PCOS

NOTE: Cases – PCOS with BMI >30 kg/m<sup>2</sup>. Controls – PCOS with BMI <  $30 \text{ kg/m}^2$ 

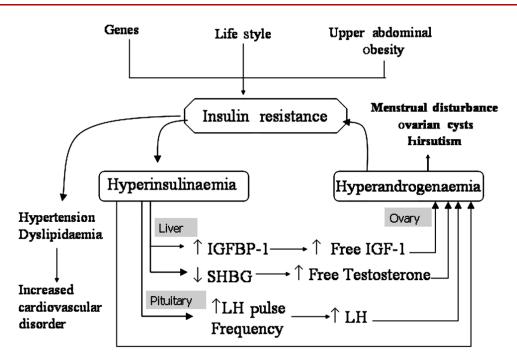


Fig-1: Pathway showing pathophysiology of PCOS

# DISCUSSION

The present study was conducted on 30 infertile women with BMI>30 kg/m<sup>2</sup> as cases and 30 age matched infertile women with BMI of  $< 30 \text{ kg/m}^2$ as controls. The dyslipidemia found in metabolic syndrome, which features elevated triglycerides and low HDL cholesterol, has been reported in association with obesity in PCOS. This is independent of obesity markers such as BMI. The prevalence of insulin resistance in our PCOS women was 76.9% and this emphasizes the importance of screening for insulin resistance and dyslipidemia in Indian women with polycystic ovary syndrome. Confirmed that insulin resistance was associated with dyslipidemia in PCOS. Hence periodic monitoring of blood pressure, lipids, and an annual OGTT especially for obese PCOS patients may be advised.

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