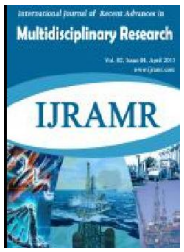


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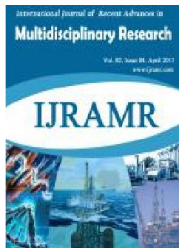


*Research Article*

# INSULIN RESISTANCE IN PCOS: A COMPARITIVE STUDY BASED ON BMI

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**ABSTRACT**

PCOS is of significant incidence in the females of reproductive age group. The easier modes of diagnostic techniques have created a unusual scenario of PCOS in the present day status.
**Aim:** To study the hormonal imbalance and Insulin resistance in patients of PCOS.
**Materials and Methods**: This is an observational study conducted in the clinical laboratory of Dept of Biochemistry, Maharajah's Institute of Medical Sciences , Nellimarla and Sri Venkateswara Medical College, Tirupati. 50 subjects were studied over a period of 9 months after dividing them into two groups based on their body mass index.
**Results:** Insulin Resistance was found to be higher in those with BMI > 25, when compared to the subjects with BMI < 25. The results were statistically significant. High Insulin Resistance influences the hormonal status of the individuals.

## INTRODUCTION

Polycystic ovary syndrome (PCOS), also called hyperandrogenic anovulation (HA), (Kollmann *et al.,* 2014) is one of the most common endocrine cause of female subfertility (Goldenberg and Glueck, 2008; Boomsma *et al.,* 2008; Azziz *et al.,* 2004; Teede *et al.,* 2010). As per the World Health Organization 3.4% of women were affected by 2010 (Vos *et al.,* 2012). The most common manifestations are irregular menstruation, amenorrhea, and infertility due to anovulation. Acne and hirsutism may be due to excess androgenic hormones. Obesity , diabetes type 2, and hypercholesterolemia may manifest as a result of Insulin resistance. The aim of the study is to focus upon hormone imbalance and the incidence of insulin resistance in patients diagnosed with PCOS in the reproductive age group.

## MATERIALS AND METHODS

The present study was carried out in the Department of Biochemistry, Maharajah's Institute of MedicalSciences, and Sri Venkateswara medical college, Tirupati over a period of nine months in the year 2014 from January to September. 50 women who were established cases of PCOS, diagnosed through ultrasound were chosen for the study. Institutional ethical committee approval was taken and the patient consent was also taken. The studied women were classified in to two groups based on Body Mass Index (BMI). Group 1 comprised of 25 women with BMI < 25 and Group 2 also comprised of 25 women with BMI > 25. The disorders other than PCOS which influence BMI are excluded from the study. In all these women fasting plasma glucose (FPG) was estimated by GOD-POD method, serum fasting Insulin (FI), serum testosterone, serum follicle stimulating hormone (FSH), serum luteinizing hormone (LH) were assayed by CLIA and HOMA- IR was calculated based on fasting insulin and fasting plasma glucose by using the formula, fasting plasma glucose (mg/dl) X fasting insulin (µU/ml)/405.

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## RESULTS

Insulin is a molecule with diverse metabolic and mitogenic effects. The insulin receptor shows a tetrameric structure with 2α and 2β dimers linked by disulphide bonds. Insulin binds to extra cellular α sub unit of insulin receptor (IR). This causes intrinsic tyrosine kinase activity which in turn results in autophosphorylation of β subunits. Insulin receptor substrate family (IRS 1-4) gets phosphorylated by the activated insulin receptor. Phosphorylated IRS proteins act as docking sites for several intra cellular proteins which mediate different actions of insulin. Translocation of GLUT 4 receptors to the cell surface which are useful in the mobilization of glucose into muscle and adipose tissue brought about by insulin mediated PI3 kinase activation is one such action (Cheatham, 1995; Saltiel, 1996; Virkamaki *et al.,* 1999).

**Table 1. Insulin resistance in Group 1and 2**

| | Group1 BMI < 25 (n=35) | Group2 BMI >25 (n=35) |
|---|---|---|
| Age (years) | 25.70±5.06 | 27.11±3.23 |
| Fasting plasma glucose (mg/dl) | 102.72±3.22 | 119.86±1.95* |
| Fasting serum insulin (µU/ml) | 15.84±0.98 | 24.22±0.86* |
| HOMA-IR | 4.01±0.30 | 5.96±0.47* |

*p<0.001.

Insulin receptor signaling is defective in PCOS, causing insulin resistance in adipocytes, fibroblasts and skeletal muscle. Though there is no reduction in the number of insulin receptors, there is a significant decrease in the glucose transport due to reduction in the GLUT 4 transporters (Rosenbaum *et al.,* 1993). A decrease in insulin stimulated tyrosine phosphorylation of normal receptors along with an increase in serine phosphorylation due to post receptor abnormalities of insulin signaling at the level of IRS 1 phosphoryl or PI3 kinase activation is the cause of insulin resistance (Dunaif, 1997). Plasma cell differentiation factor-1 (PC-1) and Tumor necrosis factor- α (TNF α) induce insulin resistance by decreasing tyrosine kinase activity and increasing serine phosphorylation respectively (Maddux *et al.,* 1995; Rosenbaum *et al.,* 1993). Increased free fatty acids (FFA) can induce insulin resistance in skeletal muscle by activating protein kinase C theta that in turn causes serine phosphorylation of IRS-1 causing alterations in the insulin signaling cascade (Griffin *et al.,* 1999).

**Table 2. Hormonal status in Group 1and 2.**

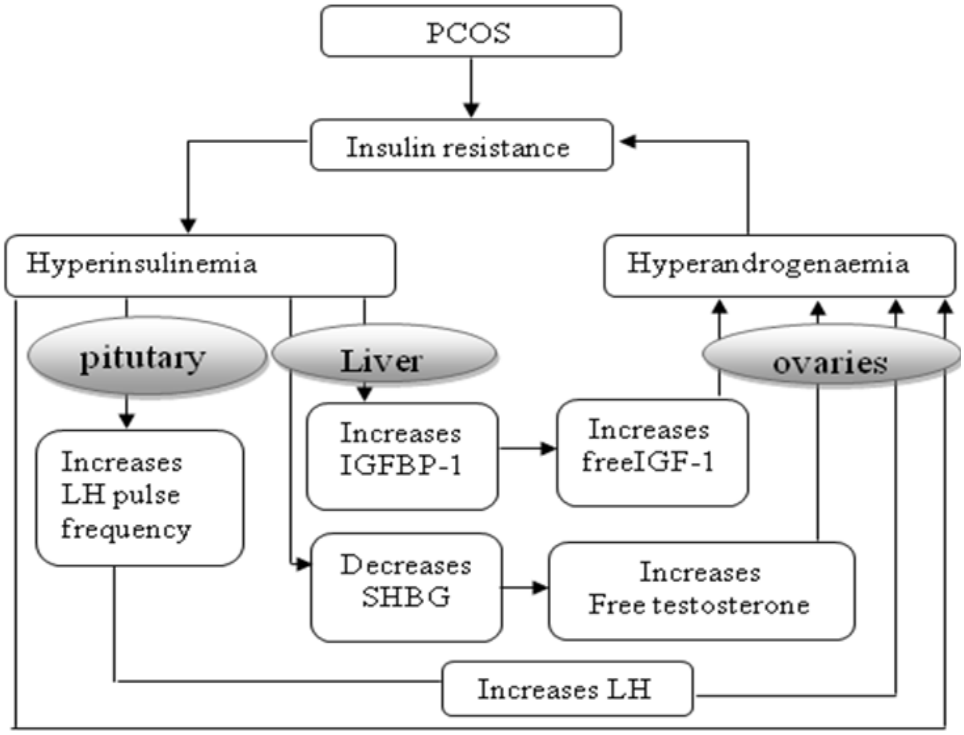
| | Group1 | Group 2 |
|---|---|---|
| Serum Testosterone (ng/dl) | 90.2±8.42 | 119.6±5.62* |
| Serum LH (IU/ml) | 4.12±1.83 | 6.4±2.47* |
| Serum FSH(mIU/ml) | 6.2±1.25 | 5.3±0.14* |

*p<0.001

Insulin either increases androgen synthesis directly or through LH mediated androgen production. It can also reduce steroid hormone binding globulin (SHBG) synthesis from liver which regulates the testosterone availability. Insulin can influence the synthesis of insulin like growth factor binding protein 1(IGFBP 1) which affects androgenesis through free IGF 1.Because of these varied effects of insulin , there is an increase of testosterone which is responsible for the manifestations of PCOS (Srabani Mukherjee, 2010).

**Conclusion**

Women with PCOS are prone for frank Diabetes mellitus type 2 and consequent dyslipidemia in association with Insulin resistance and high blood glucose levels.




The affect is considerably higher in those with greater body mass index. The incidence of atherosclerotic outcomes like Myocardial infarction and stroke can be prevented if Insulin resistance is identified at an early stage of PCOS.

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