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RESEARCH ARTICLE

# PREVALENCE OF OBESITY AMONG INFERTILE WOMEN WITH HA- PODS OR METABOLIC REPRODUCTIVE SYNDROME

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**ABSTRACT**

PCOS is mostly ahyperandrogenic disorder and is possibly the most common endocrinopathy of premenopausal women. The primary defect in PCOS appears to be an exaggerated androgen synthesis and secretion by the ovaries and the adrenal glands. In a substantial proportion of PCOS patients, the primary defect in androgen secretion is triggered by factors such as the hyperinsulinism resulting from insulin resistance and/or the secretion of metabolically active substances by visceral adipose tissue. The prevalence of obesity in PCOS patients is increased when compared to the general female population and conversely, the prevalence of PCOS is increased in overweight and obese women when compared to their lean counterparts. Almost 50% of women with PCOS are obese.[1]

Obesity exerts a major impact on the PCOS phenotype, particularly on the metabolic association and complications of the syndrome. Women with PCOS are at significantly increased risk of developing type 2 DM. Studies in isolated adipocytes and cultured skin fibroblasts from PCOS women have demonstrated intrinsic post binding defect in insulin mediated glucose metabolism. In fibroblasts, the mitogenic pathway of insulin action is intact, consistent with a selective defect in insulin signaling. While PCOS skeletal muscle is resistant to insulin in vivo, cultured muscle cells have normal insulin sensitivity, consistent with a major role of extrinsic factors in producing insulin resistance in this tissue. Excessive serine phosphorylation of insulin receptor or downstream signaling protein may be involved in the pathogenesis of insulin resistance in PCOS. The explanations for tissue specific and signaling pathway- specific differences in insulin action in PCOS may involve differential roles of insulin receptor substrate (IRS)-1 and IRS – 2 in insulin signal transduction.

Obesity without PCOS is associated with suppressed levels of SHBG, leading to higher free androgen levels which prolong follicular phase without affecting ovulation, leading to longer menstrual cycles.[4].



## INTRODUCTION

PCOS, one of the most common causes of infertility due to anovulation, affects 4-7% of women. According to the National Institutes of Health, basic diagnostic criteria should be the presence of hyperandrogenism and chronic oligo-anovulation. A consensus conference held in Rotterdam agreed on the appropriateness of including ultrasound morphology of the ovaries as a further potential criteria to define PCOS. It also established that atleast two of the following criteria are sufficient for the diagnosis:

1. Oligo and / or anovulation
2. Clinical and / of biochemical signs of hyperandrogenism
3. Polycystic ovaries on ultrasound.[2]

However the name PCOS neither reflects the hyperandrogenism, nor the metabolic derangements. The name hyperandrogenic persistent ovulatory dysfunction syndrome or HA-PODS was proposed to overcome the diagnostic pitfalls of previous nomenclature.[3]. Now the term PCOS has been changed to metabolic reproductive syndrome by the PCOS society.

## METHOD

Retrospectively 50 women with PCOS attending infertility clinic in RMMCH were analysed and the prevalence of obesity among them estimated.

BM1 was calculated by dividing weight in kg by height in $m^2$. Underweight was defined as BMI <18.5, normal as BMI-18.52 to 24.99 and overweight as BMI 25-29.99 and obese as BMI > 30.

## RESULT

| BMI | NO | Waist circumference | Percentage |
|---|---|---|---|
| <18.5 | 2 | 75.5±2.8 | 4% |
| 18.5-24.9 | 16 | 80.6±5.2 | 32% |
| 25-29.99 | 12 | 83.6±6.6 | 24% |
| ≥30 | 20 | 95.7±6.9 | 40% |

## DISCUSSION

Obesity leads to decrease in SHBG and this may lead to an increased fraction of free androgen. SHBG levels are regulated by a complex of factors, including oestrogen, iodothyronines and growth hormone as stimulating factors and androgen and insulin as inhibiting factors. The net balance of this regulation, with the dominant role of insulin, which inhibits SHBG synthesis in the liver, may be responsible for the decrease of SHBG concentrations observed in obesity.

The abdominal phenotype of obesity can be defined as a condition of relative functional hyperandrogenic state. Due to the specific action of androgen on the morphology and metabolic activity of the visceral adipocytes, it has also been argued that this endocrine milieu may in turn play a crucial role in preferentially determining an enlargement of visceral adipose tissue, thereby causing the abdominal obesity phenotype in women. This may be relevant for PCOS, this condition being associated a high prevalence of abdominal fatness, even in those presenting with normal BMI.[2]

## CONCLUSION

In our study conducted in RMMCH, we found that the prevalence of obesity was 64% among PCOS patients and underweight and normal weight women with PCOS were 36%. In summary, in women with PCOS, abdominal obesity per se may play a key role in determining both altered androgen metabolism and insulin resistance. This may be an important consideration when phenotyping PCOS and in deciding therapeutic strategies to reduce both hyperinsulinemia and hyperandrogenism.

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