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**Original article:**

# Is obesity associated with polycystic ovarian disease? – an observational analytical study

**Dr. Archana Shirsath *, Dr. Anupam S. Khare **, Ms. Honey Mittal*****

* Assistant Professor, Department of Physiology, D.Y. Patil University, School of Medicine, Nerul,Navi Mumbai

** Assistant Professor, Department of Physiology, B.J. Government Medical College, Pune

***III MBBS Student, D.Y. Patil University, School of medicine, Nerul,Navi Mumbai

Corresponding Author: Dr. Anupam S. Khare

**Abstract:**

**Introduction-**Polycystic ovarian disease (PCOD) is a common but complex endocrine disorder and is a major cause of anovulation and consequent infertility. The stressful lifestyle is leading to increased prevalence of polycystic ovarian disease in young adolescent and early reproductive population and it has association with many ongoing complications such as infertility, insulin resistance, dyslipidemia, endothelial dysfunction and overt diabetes mellitus. Obesity has an aggravating role in PCOD and several studies have documented that at least 30-40% of PCOD patients were obese or over weight.[1]The present study was undertaken to analyze the body mass index (BMI) in females having polycystic ovarian disease and compare them with age and gender matched healthy subjects.

**Material and Methods:** 60 subjects within the age group of 20 to 30 years,were divided into two groups. Group I consisted of 30 diagnosed female patients of PCOD and Group II comprised of 30 age matched control group with normal menstrual cycles. Body mass index (BMI) was measured in all 60 subjects.To assess body mass index (BMI), height and weight of the subjects wasmeasured and by using the Quetelet's index [2, 3, 4] body mass index (BMI) was calculated which is the most widely used method to gauge obesity. Body mass index of subjects was compared by applying unpaired t test.

**Results:** Body mass index was significantly increased in PCOD patients as compared to healthy controls.

**Conclusion:** In the present study, majority of polycystic ovarian disease patients were having obesity which may lead to complications of disease like infertility as well as other systemic disorders like diabetes mellitus, cardiovascular dysfunction and dyslipidemia. In the polycystic ovarian disease patients,loss of weight improves the fertility and also helps to prevent further complications of obesity.

**Key words:** Polycystic ovarian disease, Obesity, Oxidative stress, Malonyldialdehyde, Vitamin C.

## Introduction:

Polycystic ovarian disease (PCOD) is a common health problem which is increasing in teenage girls and young women. It is one of the most common endocrine disorders of women in reproductive age group, with prevalence of 15% [5] which occurs in almost all races and nationalities and is a leading cause of infertility. [6] In India, the

prevalence of PCOD is from 2.2% to 26%.[7]PCOD is an anovulatory cause of infertility affecting 6-10% of premenopausal women.[8] PCOD often presents as hyperandrogenism, hirsutism and oligomenorrhea or amenorrhea. Metabolic, endocrinologic and cardiovascular disorders may also coexist in PCOD.

Obesity may play a pathogenic role in the development of the disease in susceptible individuals. Approximately 50% of PCOD women are overweight or obese and most of them have the abdominal phenotype. This may be partly responsible for insulin resistance and associated hyperinsulinemia in women with PCOD.[9]

With this background, the present study was undertaken to analyze the body mass index (BMI) in females having polycystic ovarian disease and compare them with age and gender matched healthy subjects.

**Material and Methods:**

This study was an observational analytical study. Synopsis of the study protocol was submitted to the Institutional ethics committee and approval was obtained.

The study was conducted in the Department of Physiology in collaboration with the Department of Biochemistry and the Department of Obstetrics and Gynecology of the institute. This study was conducted from August 2015 to July 2017.

The subjects were selected from Obstetrics and Gynecology outpatient department (OPD) of the institute. A questionnaire was designed to obtain detailed history from patients and basic information on demography including weight and height was noted.Female patients in the age group of 20-30 years were included in present study.

Patients were diagnosed as having PCOD by using revised diagnostic criteria (Rotterdam criteria)[10]if 2 out of 3 from the following were present: Oligomenorrhoea and/or anovulation, Clinical and/or biochemical signs of hyperandrogenism, Polycystic ovaries.

Diagnosed cases of diabetes mellitus, thyroid dysfunction, Cushing's syndrome, congenital andrenal hyperplasia, hyperproloctinemia, androgen secreting tumor, renal and liver disorders; subjects taking medicines like ovulation induction agents, antiandrogens, antidiabetic, antiobesity, hormonal drugs and current or previous use of oral contraceptives within last 6 months; smokers and alcoholics were excluded from the present study.

Based on inclusion and exclusion criteria, a total of 60 subjects were selected for the present study. Subjects were divided into two groups. Group I consisted of 30 diagnosed female patients of PCOD and Group II comprised of 30 age matched control group with normal menstrual cycles

The study protocol was explained in detail to all the subjects and informed written consent regarding participation in the study was obtained from them.

Standing heights of subjects were recorded using stadiometer with heels together and heels, calf, buttocks and preferably back touching the stadiometer. The height was measured, without footwear to the nearest one centimeter. The weight was measured to the nearest 0.1 kg, in standing position; subjects were wearing light clothes and were bare footed.

Body mass index (BMI)/Quetelet's index: [3,4] was calculated by following formula:

BMI= Weight (Kg) / Height ($m^2$).

The data was analysed by using SPSS (Statistical package for social sciences) software version 10 by using unpaired t test. Significance level was set at P<0.05 and considered as significant. p<0.001 was considered statistical highly significant.

**Results:**

**Table1: Descriptive characteristics of baselineparameters in control group and PCOD group:**

| Baseline Parameters | Control group (n=30) Mean±SD | PCOD group (n=30) Mean±SD | p value |
|---|---|---|---|
| Age(years) | 25.60±2.88 | 25.50 ±2.53 | >0.05 |
| Height(cm) | 159.80±7.9 | 157.80±5.54 | >0.05 |
| Weight(kg) | 53.90±7.94 | 62.23±5.47 | <0.001 |

Table 1 depicts the physical characteristic of the normal control as well as the patients of PCOD.Difference in mean values of agebetween control group (25.60±2.88)and PCOD group(25.50±2.53)was found to be statistically non-significant (p >0.05).

Similarly, the difference in mean values of height between control group (159.80±7.90) and PCOD group (157.80±5.54) was statistically non-significant (>0.05)

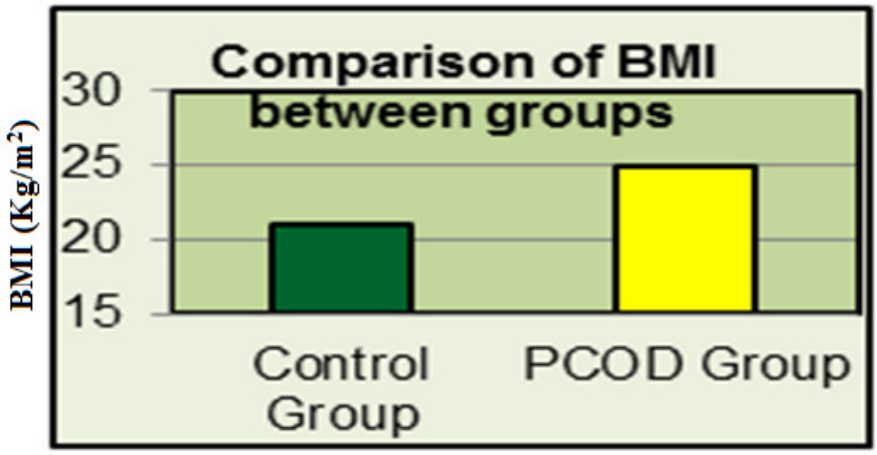
Thus PCOD group and control group had almost equal age and height, which made the baseline parameters equal and the study group comparable.

However when the weight of controlgroup(53.90±7.94) was compared with PCOD group,(62.23±5.47) difference was statistically significant (<0.001). Thus PCOD group was having more weight as compared to control group.

**Table 2- Comparison of Body Mass Index(BMI) in Control group and PCOD group.**

| **Parameter** | **Control group ( n=30) Mean±SD** | **PCOD group (n=30) Mean±SD** | **p value** |
|---|---|---|---|
| **BMI ($kg/m^2$)** | 21.05±2.17 | 25.02±2.16 | <0.001 |

**Graph 1-Comparison of BMI between control group and PCOD groups**




The present study showed that BMI of PCOD group was on higher side as compared to control. The difference in their mean values of BMI of PCOD group (25.02±2.16) was higher than the control group (21.05±2.17). The difference was found to be statistically significant ($p < 0.001$) (Table 2, Graph 1).

**Discussion**:

Our study observedthat BMI of PCOD group was significantly higher as compared to control ($p < 0.001$) (Table 2, Graph 1).Similar results were found by- Nestler JE et al also found the same result and observed that obesity permits full phenotypic expression of PCOD in women predisposed to develop this condition by generating an insulin-resistant and consequently hyperinsulinemic state which may produce hyperandrogenism by affecting multiple facets of androgen metabolism.[11]This in accordance with previous studies. [12,13,14]

Cosar E et al found that there was significantly less subcutaneous adipose tissue in the control group than the PCOD women at the triceps ($p = 0.04$) and subscapular region ($p = 0.04$). Waist-to-hip ratio of PCOD women was significantly higher than that of control subjects ($p = 0.04$). Thus he suggested that upper-half type body fat distribution is linked, with high free testosterone levels and insulin resistance in PCOD.[15]

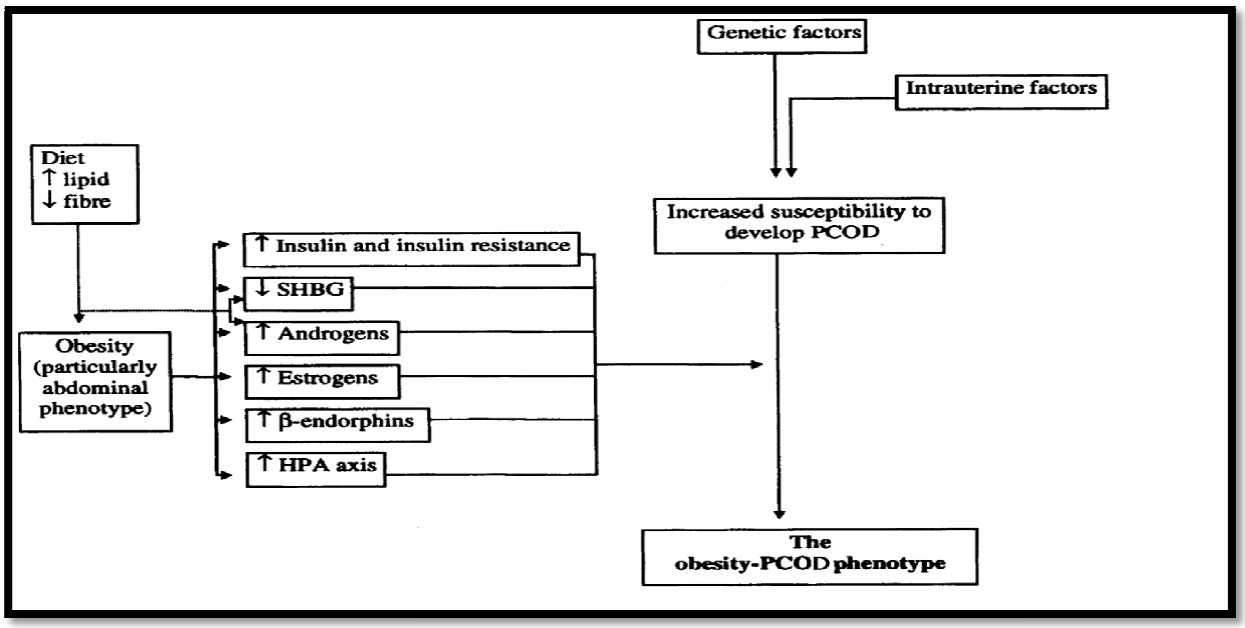
Pasquali R et al found abdominal obesity may also further worsen the hyper androgenic state in PCOD women. [16]

Holte et al found a highly significant correlation between free fatty acid (FFA) concentrations and insulin resistance, which supports the concept that an increase of FFA flux from the highly lipolytic abdominal fat to the liver and muscles may represent the most important link between abdominal obesity and the insulin resistance state.[17] Hence PCOD women may have a more unfavorable lipid profile, namely higher triglyceride and very-low-density lipoprotein (VLDL) and lower HDL cholesterol concentrations.[16]

The presence of obesity in PCOD women appears to increase the availability of active androgens and estrogens and worsen hirsutism, menstrual cyclicity and fertility rate. Therefore, obesity-related hyperinsulinemia may play a key role in favouring hyperandrogenism in these women.[9]

The pathophysiology of obesity associated with polycystic ovarian disease remains complex as obesity itself may simultaneously be the cause and the effect of the disease. [18]

Pathogenetic mechanisms by which obesity may promote or maintain PCOD is as follows.[9]




Hence, along with the usual pharmacological drugs like use of combined oral contraceptive pills, antiandrogen drugs, ovulation induction drugs, the use of antioxidants supplementation, will also be helpful for better improvement in response to this usual treatment modality.

Derangement in levels of thyroid hormone produces symptoms which may be masked by PCOD symptoms. Hence thyroid function test should be performed for early diagnosis of subclinical hypothyroid condition in PCOD patients.

Treatment of obesity through lifestyle interventions can improve insulin resistance, reproductive and metabolic symptoms. Therefore along with pharmacological treatment, weight control management is very essential in polycystic ovarian disease.

Hence the best therapeutic strategy in obese PCOD women seems to be weight loss which has proved to be effective both in reducing hyperandrogenism and improving fertility. Therefore attention should be predominantly focused on weight control or weight loss in such obese PCOD subjects.

Weight loss improves menstrual abnormality, ovulation and fertility rate. It causes reduction of Testosterone (T), androstenedione(A)and dehydroepiandrosterone-sulphate (DHEAS) levels and increase of SHBG concentrations responsible for the amelioration of the signs and symptoms reported after weight loss in obese PCOD women. Weight control decreases LH pulse amplitude which can be followed by reduced androgen production as well as reduction of the insulin level which is obviously associated with an improvement of the insulin resistant state. In addition, reduction of leptin associated with the weight loss may lead to a deactivation of the neuroendocrine control of ovarian steroid secretion. [9]

In summary, in women with PCOD, obesity per se may play a key role in determining both altered androgen metabolism and insulin resistance in a vicious circle manner. This may be of importance in phenotyping PCOD and in the therapeutic strategy aimed at reducing both hyperinsulinism and hyperandrogenism.

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