UDC 577.17:618.11-006.2

SERUM LEPTIN WITH IT`S POSITIVE CORRELATION OF ESTRADIOL LEVEL IN WOMEN WITH POLYCYSTIC OVARY SYNDROME

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Introduction

Polycystic Ovary Syndrome (PCOS) affects approximately 5–10 % of women of reproductive age and is characterized by menstrual disorders, chronic anovulation and hyperandrogenism [1]. About 50–60 % of PCOS women were obese, hyperinsulinemia and insulin resistant. A relationship between body weight and reproductive function has been observed. Women with low body fat stores such as dancers, Olympic runners and those with nervous anorexia frequently show infertility. At the other end of the spectrum, obese women present an elevated incidence of oligomenorrhea or amenorrhea and infertility. The discovery of leptin opened new perspectives about the relation between body fat and menstrual disorders. Leptin is secreted by adipocytes in proportion to the amount of body fat and exerts a potent inhibitory action on food intake. In humans, serum leptin concentrations correlate positively with the percentage of body fat. Leptin also stimulates all levels of the reproductive axis, i.e., hypothalamus, pituitary and ovary. In view of these effects, the possibility was raised that high levels of leptin may contribute to the physiopathology of PCOS, although others report that the significance of the increase is lost when leptin concentrations are adjusted for body mass index (BMI) [2]. Since obesity exerts complex effects, it is necessary to control these effects by separately studying groups of obese and non-obese PCOS patients and comparing them to control groups of obese and non-obese ovulatory women [3].

Aim

The aim of the article was to determine the relationship between serum leptin and estradiol and investigate the potential association with BMI (body mass index) in PCOS patients and weight-matched ovulatory women.

Materials and Methods of research

This article carries out a prospective clinical study among PCOS women. The data were collected from the Endocrinology and Obstetrics & Gynaecology department of Medi-cover Hospital, Hyderabad, India. Therefore, in this clinical study 30 women of reproductive age (18–43 years) were allocated to four groups: 8 obese women with PCOS (BMI ≥30 kg/m^2), 7 obese controls, 8 non-obese women with PCOS (BMI <30 kg/m^2), and 7 non-obese controls. The data was carried out as means, medians and standard deviation and analysed statistically by ANOVA for comparison of the means and Simple Spearman linear correlation and regression were used to determine the correlation between variables of BMI and estradiol.

Results and Discussion

This article consumes about 30 patients (15 PCOS patients and 15 control women) and been carrying out the clinical and metabolic characteristics of all subgroups (obese and non-obese women with PCOS and obese and non-obese controls). Using Anthropometric assessment weight was assessed on the basis of BMI by dividing weight (in kilograms) by the height squared and by ANOVA, the mean

values of BMI in PCOS patients (obese and non-obese) 35,12 ± 3a and 23,7 ± 1,2b and control women (obese and non-obese) 31,8 ± 1,3a and 23,07 ± 1,4b. Therefore, this analysis shows that BMI is almost similar in PCOS and control women and also for leptin levels we used the same test and provided the mean values. The mean serum leptin levels were similar in patients with PCOS and controls (42,26 vs 41,24 ng/ml, respectively, P = 0,93) and when compared between obese and non-obese women in both groups, they were significantly higher in the obese women (70,45 ng/ml, obese vs 9,09ng/ml, non-obese, P = 0,01). Using Spearman and regression tests, correlation analysis was performed between leptin levels and all other parameters including BMI and estradiol. There was no significant correlation between serum leptin levels and BMI both in the PCOS group and in the control group (r = 0,74, P = 0,75 and r = 0,82, P = 0,75, respectively) and between obese groups (PCOS and controls r = 0,16, P = 0,01) and non-obese groups (PCOS and control r = 0,10, P= 0,39) the correlation is demonstrable only in the obese groups. In the control group, leptin levels correlated positively with estradiol (r = 0,06, P = 0,59). However, all of these parameters correlated significantly with BMI, suggesting that these correlations were due only to BMI but estradiol did not show any significant correlation with BMI.

Our results confirm a positive correlation of leptin with body weight measurement in control groups. This article shows the effects of leptin on PCOS pathogenesis and observed higher concentrations of leptin in some women in spite of their BMI suggesting that leptin signaling anomalies in the reproductive axis could be involved in the physiopathology of PCOS. In our findings that leptin levels were not higher in patients with PCOS compared to the control group and it was correlated with the amount of fat tissue not only in patients with PCOS but also in healthy women confirm these reports. However, our most interesting result was the significantly positive correlation between leptin and E2 (estradiol), suggesting that E2 can be an important regulator of leptin production in women.

Conclusion

The fact that leptin levels are normal in a large proportion of women with PCOS does not exclude the possibility that leptin is involved in the pathophysiology of this syndrome. Although leptin values do not differ significantly between PCOS patients and controls, it is not known if body composition can affect leptin concentrations. On the other hand, leptin is secreted in a pulsatile way, and it is not known if the normal pattern of pulsatile secretion of leptin is altered in PCOS. Further studies are required to clarify the effects of leptin on the ovary and its possible pathophysiological role in PCOS. The idea that circulating leptin concentrations in PCOS patients could differ from those of subjects with regular cycles was not confirmed in the present investigation. However, it may be possible to approach the question of the role of leptin in PCOS by ways other than the simple measurement of circulating leptin levels. It appears that leptin does not play a role in LH, FSH, androgen or lipid secretion in PCOS patients or in control subjects. However, a possible link between leptin and estradiol may exist. They were positively correlated in PCOS patients regardless of their weight.

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