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Research Article

APELIN LEVELS LINKED TO POLYCYSTIC OVARY SYNDROME

Dr. Bhavya G[1], Dr. Lakshmi Sindhu

[1]Associate professor, Sri lakshminarayana Institute of medical sciences, Puducherry, India.
[2]Assistant Professor, Sri lakshminarayana Institute of medical sciences, Pondicherry, India.

ABSTRACT
Compared to risk marks in high level obese women with regular menses, the present study was designed to see if they differed. Study design: Thirty lean patients with polycystic ovary syndrome and thirty healthy subjects participated in this study. A comparison was made between groups in terms of serum apelin levels. Results: There was no significant difference between lean polycystic ovary syndrome patients and controls when it came to serum apelin levels. Conclusion: Unlike polycystic ovary syndrome itself, apelin levels do not change in response to pcos. Several more studies will be needed to determine whether this syndrome is associated with a consistent or variable pattern.

Key words: - PCOS, Obesity, BMI

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INTRODUCTION

Polycystic ovary syndrome affects 4-8 percent of women of reproductive age (polycystic ovary syndrome) [1]. Metabolic and hormonal abnormalities are characteristic of polycystic ovary syndrome. Diabetes type 2 and dyslipidemia, as well as other cardiovascular diseases, are associated with this syndrome. Anovulation, hyperandrogenism, and insulin resistance are some of the main characteristics of the syndrome [1-4]. polycystic ovary syndrome has not been able to definitively explain the underlying mechanism that causes metabolic disorders. Abnormal hormone level is still associated with metabolic abnormalities because of central obesity. The metabolic disorders and insulin resistance seen in women with lean polycystic ovary syndrome have been reported as well [5]. polycystic ovary syndrome patients with obesity as well as nonobese polycystic ovary syndrome patients with insulin resistance and inflammation have dysfunctional adipokinesin secretion [6].

Corresponding Author: **Bhavya G**

Human mature adipocytes secrete apelin, one of the newly described adipokines.

Insulin resistance is associated with apelin, which regulates normal glucose and lipid metabolism [7,8]. Plasma apelin levels are positively correlated with body mass index (BMI). Adipocytes secrete more apelin and plasma apelin levels are higher in obese adults with hyperinsulinemia, according to animal studies [9-11]. As well as hypertension, heart disease, obesity, glucose intolerance, and type 2 diabetes, patients with the apelinergic system were found to have an activated system. Plasma apelin levels were found to be lower in polycystic ovary syndrome patients in most research studies. 30 women with lean polycystic ovary syndrome were compared with 30 controls based on their serum apelin levels [12,13].

Materials and method

A control group of 30 healthy subjects who were age and BMI matched with the polycystic ovary syndrome patients was also included in the study [14]. Rotterdam

group criteria were used to diagnose abnormal hormonal level diagnosed. The diagnosis of ovulation required that patients had at least two of the following features (oligo- or amenorrhea, clinical and/or biochemical signs of hyperandrogenism, and polycystic ovaries on ultrasound), once other causes such as hyperprolactinemia and nonclassical adrenal hyperplasia had been excluded. We calculated the BMI by dividing the weight by the square of the height (kg/m^2). A lean patient is defined as having a BMI between 17.5 and 23.9 kilograms per square meter. The study excluded patients with increase the fat level disease condition of more than 25 kg/m^2.

Physical examinations were conducted on all participants. A modified Ferriman-Gallwey scoring system was used to assess the presence of hirsutism (>8 points indicated clinical hyperandrogenism) [15]. Antral follicle count, ovarian volume, and waist circumference (cm) were also determined through transvaginal ultrasound in addition to BMI and blood pressure. A longitudinal measurement and a transverse measurement were taken of the ovaries. The anteroposterior diameter of the transducer was measured after turning it 90 degrees. We calculated the volume of uterus and ovary by this formula [16].

We performed oral glucose tolerance tests, triglycerides, a profile of early follicular phase hormones, a profile of high-density lipoproteins, low density lipoproteins, very low-density lipoproteins, and a measure of insulin, blood glucose, and insulin levels in polycystic ovary syndrome patients after 12 hours of fasting on the third day of menstruation, as well as hemoglobin a1c (HbA1c) and apelin levels. Except for the oral glucose tolerance test, all tests were performed in the control group. A hormone profile was performed that included thyroid stimulating hormones (TSH), luteinizing hormones (LH), prolactins (PRLs), estradiols (E2), free testosterone, total testosterone, androstenediones (AS), 17-hydroxyprogesterones (17-OHP), dehydroepiandrosterone sulphate (DHEAS), and sex hormone binding globulins (SHBG). An automated chemiluminescence enhanced enzyme immunoassay system was used for measuring them using standard enzymatic methods. Using glucose oxidase, the plasma glucose level was determined [17]. Electrochemiluminescence immunoassay was used to measure plasma insulin levels. In an oral glucose tolerance test, serum glucose levels were measured after 75 grams of oral glucose were consumed. The insulin resistance was calculated using the homeostatic model assessment of insulin resistance formula (HOMA-IR = fasting insulin (mIU/mL) plus fasting glucose (mg/dL);]. A Beckman Coulter LX-20 Pro chemistry analyzer was used to analyze plasma lipid profiles. A centrifuge was used to separate serum from blood samples, and this serum In order to perform the apelin assays, the sample was stored at 80°C. Cterminus enzyme immunoassay kits were used to measure apelin levels in serum from humans, mice, and rats. There was a 10% intra-assay and 15% inter-assay coefficient of variation for apelin [19]. Analyses Statistically. SPSS version 15.0 was used to analyze the study's statistics. In order to compare the results, independent sample t-tests and Levene's tests were used. Using independent samples, the data were analyzed by t-tests and Pearson's correlations. Standard deviation was calculated based on the average of measures of central tendency and changes. All results were statistically significant at P <0.05.

Table 1: The following table shows group characteristics based on their demographics.

	polycystic ovary syndrome (n = 30)	**Control participants were 30**	***P***
Body Mass Index	19.77 ± 2.08	19.04 ± 2.23	0.189
Amount of experience	21.45 ± 4,10	24.67 ± 7,09	0.428
Women's menarche age	13.45 ± 1,39	13.57 ± 1.40	0.715

Table 2: Group comparisons based on biochemical and ultrasonographic measurements.

	Polycystic Ovary Syndrome (n = 30)	**Control (n = 30)**	***P***
Alperin	5.24 ± 12,.07	6.70 ± 8.36	0.589
A sugary substance	89.15 ± 6.85	92.27 ± 12.87	0.252
A1C	4.89 ± 0.52	5.10 ± 0.63	0.165
Glucose	9.88 ± 10.93	7.85 ± 4.07	0.341
IHMA-IR	2.29 ± 3.01	1.84 ± 1.22	0.444
The Great Game	74.74± 31.04	60.54 ± 16.32	0.031
High-Level Language	47.32 ± 12.92	48.78 ± 8.08	0.599
Lipids	101.32 ± 29.92	94.67 ± 23.34	0.337
Colonel	163.65 ± 36.27	155.68 ± 25.24	0.328
STH	1.69 ± 0.84	1.42 ± 0.72	0.242
AFS	5.59 ± 1.56	7.45 ± 5.14	0.056
HL	6.41 ± 4.03	5.65 ± 5.61	0.531
APR	20.55 ± 10.21	17.73 ± 6.89	0.215

It's a 2	37.74 ± 18.31	34.61 ± 18.63	0.515
Statistical test	3.27 ± 14.87	0.64 ± 0.22	0.334
Test result: F	3.35 ± 1.48	4.88 ± 2.18	0.758
According to	4.86 ± 2.17	5.41 ± 1.75	0.313
OH-17	1.34 ± 0.82	1.18 ± 0.67	0.428
ADH	259.81 ± 115.45	309.34 ± 129.23	0.124
ASG	73.77 ± 47.34	82.96 ± 4568	0.448
Vol. Ovaries of the right	7747.18 ± 4006.47	5009.65 ± 3234.95	0.006
Vol. Ovarian left	7632.12 ± 3932.69	4871.09 ± 2366.71	0.003

Results

As shown in Table 1, 30 lean abnormal level of hormone patients and 30 health controls have similar characteristics. As a result of these findings, there was no significant difference between the groups regarding the body mass index, age, blood pressure, fasting HbA1C, fasting insulin, HOMA-IR, High Density Lipoprotein, Low Density Lipoprotein, and total cholesterol, Thyroid Stimulating Hormone, Follicle Stimulating Hormone, The levels of LH, E2, PRL, free testosterone (FT), AS, 17-OH-progesterone (17-OHP), DHEAS, and SHBG were measured. In contrast, the triglyceride level was significantly higher in POLYCYSTIC OVARY SYNDROME patients than in control patients (60.053 vs. 74.73; P<0.03). Patients with POLYCYSTIC OVARY SYNDROME had significantly higher triglyceride levels (74.7331.03 versus 60.5316.31) than those with diabetes (P<0.03). There was no statistical difference between polycystic ovary syndrome patients and control subjects with regard to serum apelin levels (5.2 ± 12.1 ng/mL versus 6.7 ± 8.3 ng/mL; *P*<0.595). Table 2 shows that patients with POLYCYSTIC OVARY SYNDROME have significantly larger ovaries and follicles than controls (P<0.05). Individuals with polycystic ovary syndrome underwent oral glucose tolerance tests (OGTT) and all results were normal. Table 2 Analysis of correlation between apelin levels and other parameters (HbA1c, fasting insulin, glucose tolerance tests all hormones related and SHBG levels). Across all the groups, there was no correlation between apelin levels and any other parameter.

Discussion

This study compares Polycystic ovary syndrome and serum apelin levels patients and healthy individuals in order to diagnose the disorder. A significant difference between lean polycystic ovary syndrome patients and controls was not found in serum apelin levels. According to Chang et al., polycystic ovary syndrome patients have lower apelin levels than control subjects [7]. A study evaluating obese and non-obese cases was conducted in that study. According to Cekmez et al.'s study, apelin levels are higher in adolescents with polycystic ovary syndrome compared to controls, and apelin levels are correlated with BMI and HOMA-IR [17]. Their study evaluated obese adolescents with polycystic ovary syndrome. The mean BMI of the polycystic ovary syndrome cases in our study was lower than those in the two previous studies because patients with lean polycystic ovary syndrome were enrolled. Our results may differ from these two studies due to differences in age and body mass index. The polycystic ovary syndrome group and the control group in apelin levels. The difference in age and BMI between our study and these two is suggested as the reason for our results being different. According to these data sets, the results differ from those A study by Olszanecka- Glinianowicz et al. presents the results. In the studied groups, different ethnicities, ages, and fat distributions were weak explanations for the observed changes [18]. According to Olszanecka-Glinianowicz et al., insulin resistance is associated with increased apelin levels in women with polycystic ovary syndrome who are normal weight. Lean polycystic ovary syndrome subjects showed an increase in plasma apelin levels, whereas obese polycystic ovary syndrome women did not. polycystic ovary syndrome levels were decreased in obese subjects, but increased in lean subjects [18]. As a result of disturbed androgen synthesis, apelin may play a more complicated role in the pathogenesis of polycystic ovary syndrome. A positive correlation was found between apelin plasma levels and androstenedione serum levels in their study [18]. Although apelin levels increased in this prospective study, AS levels decreased. Even though apelin and AS are correlated, they do not differ statistically. In contrast to controls, high level fats on the obese condition patients and apelin levels were only correlated with androgen status. Several studies have shown that women with lean polycystic ovary syndrome are at high risk for cardiovascular disease, which is aggravated by hyperandrogenism [19]. Lean polycystic ovary syndrome patients' androgen levels were not significantly different from those in the control group, according to the results of the current study. Accordingly, apelin may be involved in the pathogenesis of polycystic ovary syndrome-associated insulin resistance, but apelin levels were not significantly different between lean polycystic ovary syndrome patients and control subjects. similarly, to insulin, the secretion of apelin is strongly inhibited by fasting while it increases after eating. Adipocytes' apelin gene expression appears to be controlled directly by insulin. Insulin and apelin levels are very high in obese patients [20]. Women with obesity might be at risk for metabolic abnormalities caused by apelin. Due to polycystic ovary syndrome being associated with obesity, apelin levels are likely to increase. Healthy women's apelin levels would be similar to those of

polycystic ovary syndrome patients without insulin resistance and an anthropometric measurement of BMI of 25. Appelin levels do not seem to be affected by polycystic ovary syndrome itself. This issue needs to be clarified by large series and new studies.

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