

Serum Obestatin Levels in PCOS and its Relation with Homa-IR

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Abstract

Background: Is to investigate the level of Obestatin in PCOS patients and compare with controls and also see the correlations between the serum Obestatin levels and HOMA-IR.

Patients and methods: We analyzed 30 patients with PCOS and 20 normal women as controls. PCOS patients were divided into two groups based on obese group and non-obese group Serum Obestatin levels, Insulin Homeostasis Model Assessment for Insulin Resistance (HOMA-IR) determined and compared among both groups.

Results: Serum Obestatin levels were significantly lower in obese PCOS group than non-obese and control.

Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine disorder in females, especially in women of reproductive age. PCOS could be diagnosed by infertility, acne, amenorrhea or oligomenorrhea, hirsutism, insulin resistance, obesity, hyperandrogenism, and polycystic ovaries by ultrasonography. Association of PCOS with infertility is well studied and is thought to be responsible for 40% of female infertility.

PCOS was first reported by Stein and Leventhal in 1935. With rising urbanisation, the disease picked up pace and it was only in 2003 that Rotterdam criteria for the diagnosis of PCOS was formulated. Primary symptoms include abnormal facial and skin growth (hirsutism) and baldness, acne, weight gain, irregular or absence of menstrual cycle and increased levels of male hormones.

PCOS is common among members of younger generation, with almost 10 million people affected globally. Its worldwide prevalence varies from 2.2% to 26% and according to latest statistics, in India one in every four young women is said to have Polycystic ovary disease (PCOD).

PCOS is often associated with number of cardiometabolic disorders such as obesity, dyslipidemia, hypertension, insulin resistance, hyperinsulinemia, glucose intolerance and type 2 diabetes mellitus (T2DM). Obesity is considered as one of the major factor predisposing to the development of PCOS, since 35-80% of the women suffering from PCOS are reported to be overweight or obese. Many of the metabolic abnormalities associated with PCOS such as insulin resistance (IR), impaired glucose tolerance, type 2 diabetes mellitus (DM2) etc. are known to get worsened by concurrent obesity. However, it is not the only factor, since many PCOS patients are lean although many of these findings can be largely explained by the increased prevalence of abdominal obesity, even in normal-weight PCOS patients.

Obestatin, a novel 23 amino acid peptide encoded by the same gene with ghrelin and derived from the precursor protein Proghrelin. Obestatin behaves as a physiological opponent to ghrelin in inhibiting food intake, body weight gain and gastric emptying. Further studies explained that

obestatin was involved in inhibiting thirst and anxiety, improving memory, regulating sleep and increasing the secretion of pancreatic juice enzymes. Overstating could stimulate adipose cell proliferation and inhibit the secretion of IGF-1.

The present study was, therefore, undertaken to investigate the correlations between serum levels of Overstating in PCOS women and to evaluate their relationship with obesity and insulin resistance.

Methods

We analyzed 30 patients with PCOS and 20 normal women as controls. PCOS patients were divided into two groups based on obese group and non-obese group Serum Obestatin levels, Insulin Homeostasis Model Assessment for InsulinResistance (HOMA-IR) determined and compared among both groups.

Women were recruited from the department of Obstetrics and Gynecology RajkiyaMahilaChikitsalaya, Ajmer between January and August 2023

Exclusion Criteria

1. Patients with Diabetes Mellitus.
2. Patients with Hepatic and Renal dysfunction.
3. Patients with Thyroid dysfunctions.
4. Pregnant women.
5. Patients with Cardiovascular disease.

Biochemical Parameters:

Following Estimations Will Be Carried out-
Serum obestatin- ELISA method.

Insulin resistance calculated by using Homeostasis model

Fasting plasma glucose [mmol/L] X Fasting insulin [mU/L]/22.5

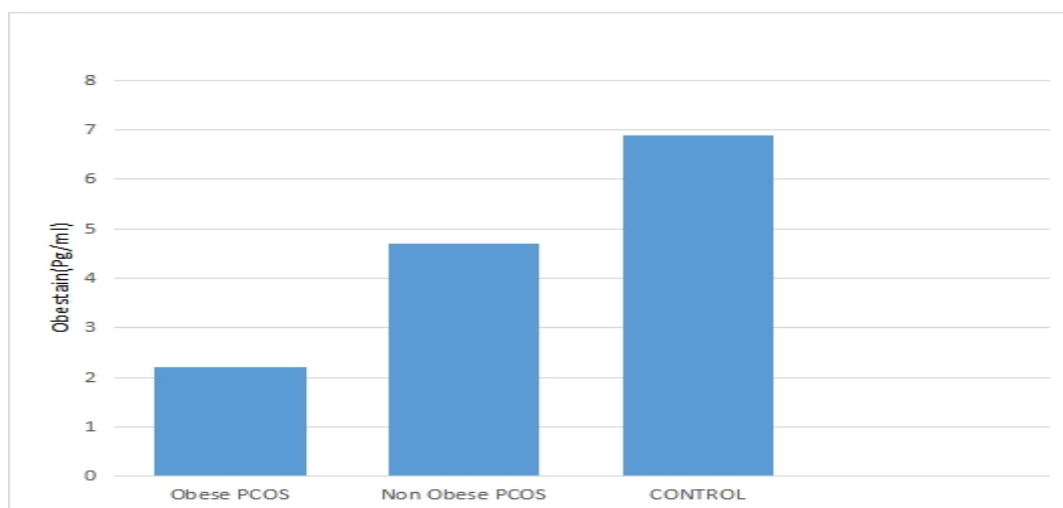
Results

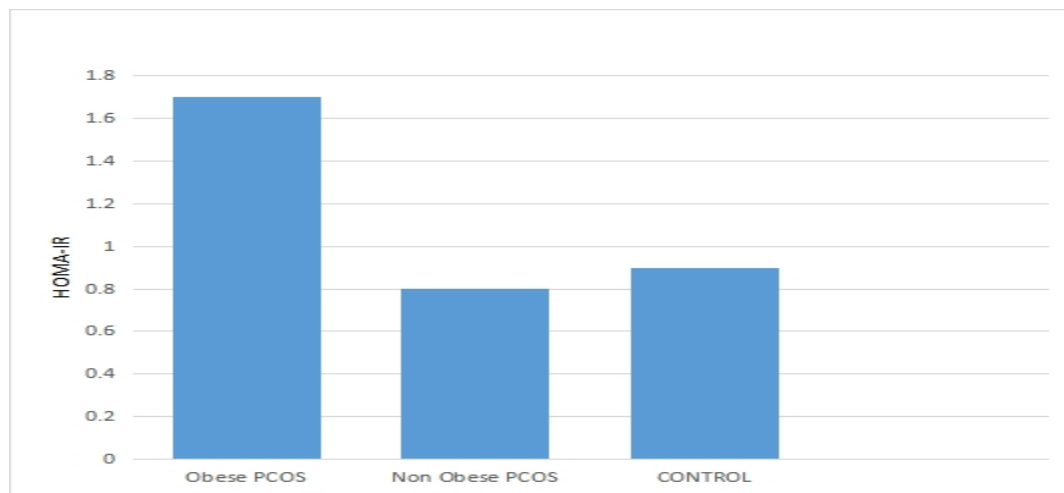
Serum obestatin levels were significantly decreased in obese PCOS patients when compared to both control and non-obese ones.

Obestatin was negatively correlated with HOMA-IR in PCOS patients.

LEVELS OF OBESTATIN AND HOMA-IR IN OBESE PCOS AND NON OBESE PCOS AND COMPARISON WITH CONTROL

	Obese PCOS	Non Obese PCOS	Control
Obestatin (Pg/ml)	2.2	4.7	6.9
HOMA-IR	1.7	0.9	0.8





Discussion

Polycystic ovaries (PCO) are the morphological ovarian phenotype in women with the polycystic ovary syndrome (PCOS). Several studies performed to attempt to determine the prevalence of PCO as detected by ultrasound alone in the general population, and have found prevalence rates in the order of 17–33%. The current study, we correlates the serum levels of Obestatin with obesity and insulin resistance in polycystic ovary, through the estimation of the serum levels of Obestatin and carotid artery intima-media thickness, brachial artery flow mediated dilatation and other metabolic and hormonal parameters in obese and non-obese women with PCOS and healthy controls.

In the present study, the obese women with PCOS have a significantly higher level of triglycerides, HOMA-IR, total testosterone, CRP, blood pressure and WHR values and lower LDH levels when compared to control and non-obese ones.

Reference

1. Adel A Elboghady, Abeer I, Abd EL-Fattah. Copeptin and obestatin levels in polycystic Ovary women and their relation to obesity, Insulin metabolism and cardiovascular diseases. AIMJ.2020; 27820.1197.
2. Akram M, Roohi N. Endocrine correlates of polycystic ovary syndrome in Pakistani women. J coll physician's surgpak.2015; 25: 22-26.
3. Azziz R, Sanchez LA, Knochener ES, Moran C, Lazenby J, Stephens KC. Androgen excess in women: experience with over 1000 consecutive patients. J ClinEndocrinolMetab. 2004; 89:453–462.
4. Begum GS, Shariff A, Ayman G, Mohammad G, Housam R, Khaled N. Assessment of risk factors for development of polycystic ovarian syndrome. Int J Contemporary Med Res. 2017; 4: 77–83.
5. Boomsma CM, Eijkemans MJ. A Meta analysis of pregnancy outcomes in women with PCOS. Hum Repord update. 2006; 12: 673-683.
6. Carmina E, Bucchieri S, Esposito A. Abdominal fat quantity and distribution in women with polycystic ovary syndrome and extent of its relation to insulin resistance. J ClinEndocrinolMetab.2007; 92: 2500-2505.
7. Costello MF, Misso ML, Wong j, Hart R, Rombauts L, Melder A. The treatment of infertility in polycystic ovary syndrome. Aust N Z J ObstetGynecol.2012; 52: 400-403.
8. Cupisti S, Kajaia N, Dittrich R, Duezenli H, Beckmann MW, Mueller A. Body mass index and ovarian function are associated with endocrine and metabolic abnormalities in women with hyperandrogenic syndrome. Eur J Endocrinol. 2008; 158: 711–719.

9. Ehrmann DA, Kasza K, Azziz R, Legro RS, Ghazzi MN. Effects of race and family history of type 2 diabetes on metabolic status of women with polycystic ovary syndrome. *J ClinEndocrinolMetab.*2005; 90: 66-71.
10. Gambineri A, Patton L, De Iasio R, Cantelli B, Cognini GE, Filicori M. Efficacy of octreotide- LAR in dieting women with abdominal obesity and polycystic ovary syndrome. *J ClinEndocrinolMetab.*2005; 90: 3854-3862.
11. Hahn S, Tan S, Sack S, Kimmig R, Quadbeck B, Mann K. Prevalence of the metabolic syndrome in German women with polycystic ovary syndrome. *Exp ClinEndocrinol Diabetes.*2007; 115: 130–135.
12. Holst JJ, Gromada J. Role of incretin hormones in the regulation of insulin secretion in diabetic and non diabetic humans. *American Journal of Physiology.Endocrinology and Metabolism.*2004; 287: 199-206.
13. Tang SQ, Jiang QY, Zhang YL, et al. Obestatin: its physiochemical characteristics and physiological functions. *Peptides* 2008; 29:639–645.
14. Khan MJ, Ullah A, Basit S. Genetic Basis of Polycystic Ovary Syndrome (PCOS): Current Perspectives. *ApplClin Genet.* 2019; 12: 249-260.
15. Kumar AN, Naidu JN, Satyanarayan U. Metabolic and endocrine characteristics of Indian women with PCOS. *Int J Fertilsteril.* 2016; 10: 22.
16. Legro RS, Arslanian SA, Ehrmann DA, HoegerKM . Diagnosis and treatment of polycystic ovary syndrome. An Endocrine society clinical practice guideline. *J Clin endocrinal metab.*2013; 98: 4562-4592.
17. Messinis IE, Messini CI, Anifandis G, Dafopoulos K. Polycystic ovaries and obesity. *Best Pract Res ClinObstetGynaecol.* 2015; 29(4): 479–488.
18. Pagotto U, Gambineri A, Vicennati V, Heiman ML, Tschöp M, Pasquali R. Plasma ghrelin, obesity, and the polycystic ovary syndrome: correlation with insulin resistance and androgen levels. *J ClinEndocrinolMetab.* 2002; 87: 5625–5629.
19. Pasquali R, Stener-Victorin E, Yildiz BO, Duleba AJ, Hoeger K, Mason H, Homburg R, Hickey T, Franks S, Tapanainen JS, Balen A, Abbott DH, Diamanti-Kandarakis E, Legro RS. PCOS Forum: Research in polycystic Ovary Syndrome Today and Tomorrow.*ClinEndocrinol (Oxf).* 2011; 74(4):424-33.
20. Randeva HS, Tan BK, Weickert MO. Cardiometabolic aspects of the polycystic ovary syndrome. *Endocr Rev.* 2012; 33: 812–841.
21. Sam S. Obesity and polycystic ovary syndrome. *ObesManag.* 2007; 3: 69–73.
22. Svendsen PF, Nilas L, Madsbad S, Holst JJ. Incretin hormone secretion in women with polycystic ovary syndrome: roles of obesity, insulin sensitivity, and treatment with metformin. *Metabolism: clinical and experimental.* 2009; 58(5): 586-593.