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Serum Visfatin Levels in Women with Newly Diagnosed Polycystic Ovarian Disease: A Cross-Sectional Study

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ABSTRACT

Background: Polycystic ovarian disease (PCOD) is a common endocrine disorder frequently associated with insulin resistance and metabolic dysfunction. Visfatin, a visceral adipocytokine, has been implicated in inflammation and glucose regulation.
Aim: To determine Serum Visfatin levels in newly diagnosed PCOD women and assess its relationship with body mass index (BMI).
Methods: A cross-sectional study was conducted involving 80 women (40 PCOD; 40 healthy controls). Anthropometric, hormonal, glycemic, insulin resistance index, and serum Visfatin levels were measured.
Results: Serum Visfatin was significantly increased in PCOD cases (39.25 ± 16.49 ng/mL) compared to controls (27.90 ± 8.21; $p = 0.004$). PCOD women also had significantly elevated LH, LH/FSH ratio, fasting glucose, insulin, and HOMA-IR ($p < 0.001$).
Conclusion: Serum Visfatin is significantly elevated in PCOD and correlates with metabolic dysfunction. It may serve as an early biomarker for insulin resistance and cardiometabolic risk in PCOD.

Keywords: PCOD, Visfatin, HOMA-IR, Insulin Resistance, Adipocytokines

INTRODUCTION

Obesity is characterised by increase in number and volume of adipocytes with abnormal lipid storage. The effects of obesity on female reproductive system includes impaired ovulation, irregular menstrual cycles, miscarriage and impaired reproductive potential [1]. Central obesity is commonly associated with PCOD and hence these patients are more prone for insulin resistance and metabolic syndrome & CVS complications. In obesity, fat mass expands in order to store energy. Nowadays, adipose tissue is considered to be an endocrine organ [2]. Adipose tissue plays an important role in regulating various physiological processes such as reproduction, immune response, glucose and lipid metabolism by secreting adipokines [3]. Moreover, PCOD is also associated with high rate of central obesity.
In PCOD, the increased risk of cardiovascular events and metabolic disturbances may be due to the abnormal production and release of adipokines which are the cytokines and inflammatory factors secreted by adipose tissue [4]. Though

considered as a storage organ, several researchers proved that adipose tissue is an endocrine organ whose function when altered may produce cardio metabolic disturbances in PCOS. Dysregulated adipocyte function and obesity play major role in PCOS [5]

Visfatin, also referred to as pre-B cell colony enhancing factor [PBEF], has been identified as a multifunctional adipocytokine. It was formerly said to be as Nicotinamide Phosphoribosyl transferase [Nampt], which is the rate limiting enzyme in NAD (Nicotinamide Adenine Dinucleotide) biosynthesis [6]. It was named so, since it is mainly secreted from visceral adipose tissue. It is a 52k Dalton protein, found in various tissues such as adiposities, lymphocytes, bone marrow, liver and muscle [7]. The mechanism of action of visfatin seems to be, binding to the insulin receptor, at a site different from insulin. It exerts various insulin mimetic effects such as increasing glucose uptake in adipose tissue and muscle, decreasing the release of glucose in hepatocytes and increasing the formation of triglycerides.

Visfatin regulates the sensitivity of insulin in the liver through its autocrine actions [8]. Visfatin mediates inflammation. Visfatin enhances cell proliferation and NAD synthesis. It is also found that visfatin levels are elevated in type two diabetes mellitus. Visfatin gives rise to insulin resistance and hyperinsulinemia in PCOD women. Visfatin has insulin-mimetic actions. Visfatin tends to increase and compensate for the already developed insulin resistance [9]. On the other hand, increased visfatin levels are associated with endothelial dysfunction by activating Nuclear Transcription factor NT F – KB and Metalloproteinases 2/9 in vascular endothelial cells thereby increasing the risk of cardiovascular events [10]. Hence, increased visfatin in PCOS patients with Insulin Resistance are prone for cardiovascular risk. Visfatin is not only produced by adipocytes. It is also secreted by inflammatory cells, such as activated macrophages. It also gets infiltrated in the Adipose Tissue and thereby increased in obesity [11]

AIM

To determine whether Serum Visfatin levels are elevated in women with Polycystic ovarian disease and to find its relationship with Body Mass Index.

OBJECTIVES

1. To determine Serum Visfatin levels in women with newly diagnosed Polycystic ovarian disease.
2. To estimate the Body Mass Index.

MATERIALS & METHODS

This cross-sectional study was conducted in the Tertiary care centre in Chennai, with the objective of assessing metabolic parameters and serum Visfatin levels in women with Polycystic Ovarian Disease (PCOD). The study population consisted of 80 women, including 40 newly diagnosed PCOD cases and 40 age-matched healthy controls. All participants belonged to the reproductive age group of 18–40 years. The diagnosis of PCOD was established based on the Rotterdam criteria, ensuring uniformity in case selection.

Prior to enrolment, written informed consent was obtained from all participants. A detailed clinical history was recorded, followed by a thorough general and systemic examination. These steps were undertaken to identify and exclude conditions that could confound the study variables. Women who were pregnant or presented with disorders known to mimic PCOD - such as hypothyroidism, hyperprolactinemia, androgen-secreting tumors, or idiopathic hirsutism - were excluded. Participants with any neoplastic, hepatic, renal, or cardiac illness were also not considered for inclusion.

Anthropometric measurements including height and weight were recorded using standard equipment and protocols. Body Mass Index (BMI) was calculated using the Quetelet Index formula (BMI = weight in kilograms / height in meter^2). All biochemical samples were collected after an

overnight fast, between 8:00 and 10:00 AM, to minimize diurnal and dietary variations in metabolic parameters. Blood was drawn using aseptic precautions, and serum was separated and stored at –20°C until further analysis.

Fasting blood glucose and fasting serum insulin concentrations were measured using standardized laboratory procedures. Insulin resistance was assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), calculated using the following formula:

$$\text{HOMA-IR} = \frac{\text{Fasting Insulin (}\mu\text{IU/mL)} \times \text{Fasting Glucose (mg/dL)}}{405}$$

For the estimation of serum Visfatin, samples were collected specifically during the early follicular phase of the menstrual cycle to ensure hormonal consistency. Visfatin levels were quantified using a commercially available Human Visfatin ELISA kit (ElabScience) based on the sandwich enzyme-linked immunosorbent assay technique. All pre-analytical and analytical procedures—including sample handling, storage, and assay preparation—were performed strictly according to the manufacturer's instructions to maintain accuracy and reproducibility of results.

The structured methodology, uniform sample processing, and carefully defined inclusion and exclusion criteria ensured that the study results reflect reliable and valid comparisons between PCOD cases and healthy controls.

Statistical Analysis

Data were analysed using SPSS version 21, with descriptive statistics presented as mean ± standard deviation. Pearson correlation analysis was performed to explore relationships between variables, considering p-values less than 0.05 as statistically significant

RESULT

Table 1. Comparison of Anthropometric and Biochemical Parameters Between Controls and PCOD Cases

Parameter	Controls (n = 40)	PCOD Cases (n = 40)	p-value
Age (years)	28.98 ± 6.90	29.35 ± 7.33	0.813
Height (cm)	156.53 ± 4.45	156.65 ± 5.12	0.918
Weight (kg)	64.65 ± 13.34	71.78 ± 18.73	0.147
BMI (kg/m²)	26.32 ± 5.00	29.11 ± 6.91	0.122
Fasting Blood Sugar (mg/dL)	72.45 ± 10.53	86.68 ± 12.68	**0.000***
Serum Insulin (µU/mL)	8.78 ± 2.04	25.93 ± 11.74	**0.000***
HOMA-IR	1.58 ± 0.46	5.49 ± 2.49	**0.000***
Serum Visfatin (ng/mL)	**27.90 ± 8.21**	**39.25 ± 16.49**	**0.004***

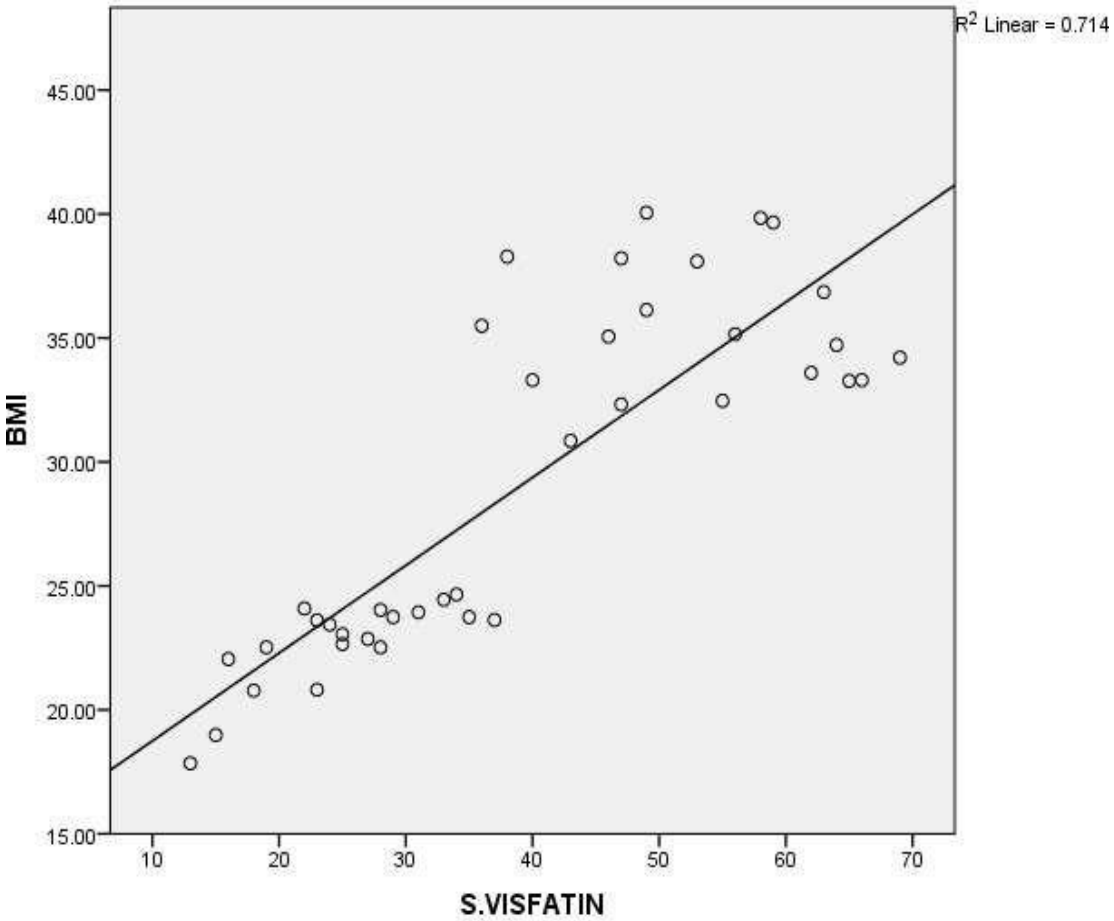
Table 1 summarizes and compares the anthropometric and biochemical characteristics between women with newly diagnosed polycystic ovarian disease (PCOD) and healthy controls. Both groups were comparable in age, height, weight, and BMI, with no statistically significant differences in these baseline variables ($p > 0.05$).

However, significant metabolic disparities were observed. PCOD participants demonstrated markedly higher fasting blood glucose, serum insulin levels, and HOMA-IR, all showing strong statistical significance ($p < 0.001$). Importantly, serum visfatin levels were significantly elevated in PCOD women (39.25 ± 16.49 ng/mL) compared to controls (27.90 ± 8.21 ng/mL) ($p = 0.004$).

Table 2. Correlation of various parameters with Serum Visfatin in PCOD

VARIABLES	R	P
AGE (years)	0.172	0.288
HEIGHT (cm)	0.164	0.311
WEIGHT (kg)	0.806	0.000**
BMI (kg/m2)	0.845	0.000**
F.B.S(mg/dl)	0.156	0.335
S. Insulin(mu/l)	0.856	0.000**
HOMA-IR	0.898	0.000**

The above table shows positive correlation between serum visfatin levels with Body Mass Index, Serum Insulin and HOMA-IR which is statistically significant.



GRAPH 1: SCATTER PLOT (SERUM VISFATIN AND BMI)

Graph 1 illustrates the scatter plot showing the relationship between serum visfatin levels and body mass index among women with PCOD. The distribution of data points demonstrates a clear positive linear trend, indicating that visfatin levels increase proportionally with rising BMI. This visual representation supports the statistical findings exhibited in Table 2 and reinforces visfatin's association with adiposity in PCOD patients.

DISCUSSION

This study demonstrated significantly elevated serum Visfatin levels in newly diagnosed PCOD women. Elevated Visfatin correlated positively with BMI and HOMA-IR, reinforcing its role as a metabolic mediator in PCOD. These results align with studies suggesting that visfatin reflects adiposity-driven insulin resistance [12,13].

In the current study, the purpose was to estimate and investigate the role of visfatin as

a novel potential biomarker of insulin resistance in the PCOS subjects. This was with the view of Polak K et al., (2016) that serum visfatin levels could be used as an early identification of the Insulin resistance and its related metabolic alterations, which would in turn enable us to make early interventions [14].

In our study, the BMI, the insulin resistance and the serum visfatin levels were estimated and compared in patients with PCOS and normal controls. Serum visfatin levels are correlated with BMI and Insulin Resistance in cases and controls.

The findings of the study indicate that serum visfatin levels are significantly elevated in women with PCOS compared to healthy controls and show a positive correlation with both insulin resistance and Body Mass Index. Visfatin (52k Dalton) is a recently diagnosed adipocytokine, secreted preferentially in the visceral adipose tissue. It plays an essential role in number of biological processes such as inflammation, immunity, insulin resistance, endothelial cell dysfunction and angiogenesis. It also has insulin mimicking action and can serve as a proinflammatory mediator and immunomodulator.

Studies by Tania Romacho et al., (2013) and Dinka Pavicic Baldani et al., (2014) have clearly shown that serum visfatin levels may predict the occurrence of metabolic syndrome, type 2 diabetes and cardiovascular events, and hence it can be used as a predictor of severity of insulin resistance and hence as a biomarker for treatment of polycystic ovarian disease.

This study has shown that PCOS patients were more insulin resistance than control women leading to the suggestion that visfatin could be a specific marker of insulin sensitivity, possibly contributing to the pathogenesis of PCOS.

Results by Hassan M Salama et al., (2015) have showed that serum visfatin levels are positively correlated with Insulin Resistance and Body Mass Index which are consistent with our results [15].

CONCLUSION

The findings of the present study demonstrate that Serum Visfatin levels are significantly elevated in women with Polycystic Ovarian Disease (PCOD) when compared with healthy controls. This rise in Visfatin appears to parallel metabolic alterations commonly observed in PCOD. A strong and positive correlation was noted between Visfatin levels and both Body Mass Index (BMI) and HOMA-IR, emphasizing the close association between Visfatin and insulin resistance. These observations suggest that Visfatin may play a contributory role in the pathophysiological mechanisms underlying metabolic dysfunction in PCOD. Given its strong correlation with established metabolic markers, Visfatin shows promising potential as an early biomarker for metabolic screening, risk stratification, and timely intervention in women with PCOD. Further large-scale studies are warranted to validate its clinical utility and explore its applicability in routine diagnostic practice.

Declaration by Authors

Ethical Approval: Approved

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