RESEARCH PAPER

Biochemical Evaluation of Adiponectin and Leptin in Patients with Polycystic Ovary Syndrome

Anshika Mishra[1*], Ritesh Kumar Vishwakarma[2], Gyanendra Kumar Yadav[3]

[1*]Phd Research Scholar, Index Medical College and Research Center Indore ,MP, Anshikaanumishra@gmail.com

[2]Assistant Professor, Department Of Biochemistry , Index Medical College & Research Center, Indore, MP

[3]Senior Resident, Autonomous State Medical College Sultanpur, UP, Gyanendra02071995@gmail.com

ABSTRACT

Background: polycystic ovary syndrome (pcos) is a multifactorial hormonal and metabolic condition affecting women during their reproductive years. It may also be accompanied by metabolic complications, particularly obesity and reduced insulin sensitivity. In some affected individuals, darkened and thickened areas of the skin, commonly referred to as acanthosis nigricans, may also be observed. The development of pcos is believed to involve a complex interaction of genetic predisposition and environmental influences. Adiponectin levels are likely to be more attributed to obesity itself rather than pcos. The role of adipose tissue hormones adiponectin has been playing a major role in the pathogenesis of pcos. Leptin signaling pathways, potentially dissociating circulating leptin levels from adipose tissue mass in this population. Second, androgens have been shown to suppress leptin secretion from adipose tissue. It is important in regulating energy homeostasis and has an impact on the reproductive systems in diverse ways.

Material and method: a total of 238 subjects, including 119 diagnosed polycystic ovary syndrome cases and 119 healthy controls were chosen for the study after imposing certain inclusion criteria and exclusion criteria for cases and controls. The samples were analyzed on cobas e 411 for fsh, lh and prolactin levels. Estimation of adiponectin and leptin level by using a double- antibody sandwich method enzyme linked immunosorbent one step process assay.

Results: present study was significant higher in lh, prolactin and leptin levels between the case and control groups. However, the fsh and adiponectin levels were significantly lower in the case group compared with those in the control group and there was no significant correlation between lh with fsh, adiponectin leptin and bmi levels. The only significant correlations were between leptin and adiponectin levels, as well as prolactin and fsh levels. The positive correlations between prolactin with fsh levels and leptin with adiponectin levels. negative correlations between adiponectin with BMI levels. **Conclusion**: adiponectin levels were significantly lower in the pcos group compared with healthy controls. Adiponectin contributes to insulin sensitivity, inflammatory regulation, and androgen metabolism. Leptin concentrations were higher in obese subjects in both the pcos and control groups. Leptin is involved in metabolic and reproductive regulation and may contribute to pcos through its association with obesity.

Keywords: LH, FSH, Prolactin Adiponectin and Leptin

How to cite this article: Mishra A, Vishwakarma RK, Yadav GK, Biochemical evaluation of adiponectin and leptin in patients with polycystic ovary syndrome. Int J Drug Deliv Technol. 2026;16(90s): 2148-2153; DOI: 10.25258/ijddt.16.90s.215

Source of support: Nil

Conflict of interest: None

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a prevalent endocrine disorder, characterized by biochemical hyperandrogenemia, persistent anovulation, and the presence of polycystic ovaries. Polycystic ovarian syndrome (PCOS) was first characterized by Stein and Leventhal in 1935. It constitutes a diverse array of signs and symptoms, exhibiting minor manifestations in some cases while causing significant disruption to reproductive

and endocrine functions. The underlying mechanisms seem to be complex and polygenic. The characterization of PCOS is widely contested, however it encompasses prominent characteristics such as irregular menstrual cycles, hyperandrogenism, and obesity [2].

Polycystic ovary syndrome (PCOS), referred to as hyperandrogenic anovulation (HA) or Stein–Leventhal syndrome, is a prevalent endocrine and metabolic condition marked by hormonal dysregulation in women of childbearing age. It is linked to several clinical symptoms, such as irregular or missing menstrual cycles, heavy menstrual flow, hirsutism, acne, pelvic pain, and challenges in achieving pregnancy. Additional related characteristics include insulin resistance, adiposity, and alterations in skin coloration, including acanthosis nigricans. Genetic and environmental influences may furthermore play a role in the onset of PCOS [3,4]. Polycystic ovary syndrome (PCOS) impacts about 5–10% of women of childbearing age and is recognized as one of the most prevalent endocrine illnesses within this demographic [4,5].

Diagnostic criteria: In 2006, the Androgen Excess and PCOS Society (AE-PCOS) established diagnostic criteria for polycystic ovary syndrome (PCOS). Based on these standards, the diagnosis necessitates the existence of hyperandrogenism (either clinical or biochemical), ovarian dysfunction (including oligo-ovulation, anovulation, and/or polycystic ovarian morphology), and the elimination of alternative conditions linked to androgen surplus.

Adipokines, which are hormones released by adipose tissue, significantly contribute to the metabolic irregularities seen in PCOS. Alterations in adipokine concentrations might influence insulin sensitivity and inflammation, hence complicating the clinical scenario. Comprehending these biochemical indicators facilitates a more holistic strategy for controlling PCOS, addressing both reproductive and metabolic dimensions of the condition [7,8]. Consequently, this study was designed to assess the hormonal profile, namely LH, FSH, Prolactin, as well as adiponectin and leptin levels in patients.

MATERIALS AND METHODS

Study Design and Subject Selection

The current case-control study was carried out at the index medical college hospital & research center, Department of Biochemistry in indore, India. A total of 238 subjects, including 119 diagnosed polycystic ovary syndrome cases and 119 healthy controls were chosen for the study after imposing certain inclusion criteria and exclusion criteria for cases and controls. Subjects included for polycystic ovary syndrome cases group were subjects aged 20-40 years old diagnosed with PCOS according to the National Institutes of Health Criteria (NIH), while healthy individuals aged 20-40 years were included as subjects for the control group. Subjects with a history of Female with any other reproductive disorders Thyroid, hepatic, renal disease and Cardiovascular illness were excluded from the hypothyroidism cases. Meanwhile, subjects with a history of Conditions where phlebotomy is contraindicated, Female are suffering from any chronic illness And Subjects are suffering from any acute illness were excluded from the control group.

Sample Collection and Serum Separation

Five mL of venous blood were drawn from each subject under aseptic conditions. The blood sample was allowed to clot at room temperature before being centrifuged for 5 minutes at 3000 rpm to separate the serum, which was then used to assess the levels of FSH, LH, Prolactin, adiponectin and leptin.

Estimation of FSH, LH, Prolactin, adiponectin and leptin.

The samples were analyzed on Cobas e 411 in Department of Biochemistry for FSH, LH and Prolactin levels. Estimation of adiponectin level by using a double-antibody sandwich method enzyme linked immunosorbent one step process assay (ELISA) and Leptin level by using a double- antibody sandwich method enzyme linked immunosorbent one step process assay (ELISA).

Statistical analysis

Statistical analysis was carried out on Microsoft excel. Differences in the continuous variables was analyzed using student t-test if the distribution was normal. Continuous parameters were expressed as mean ±SD. Student´s t test was applied to the data. Pearson correlation was used to correlate LH, FSH, prolactin, adiponectin and leptin. P value < 0.05 was considered statistically significant.

RESULT

Table 1 shows the biochemical and anthropometric parameter values between the two study groups. There was significant higher in LH, Prolactin and leptin levels between the case and control groups. However, the FSH and adiponectin levels were significantly lower in the case group compared with those in the control group {Figure 1a, b}.

There was no significant correlation between LH with FSH, adiponectin and leptin levels. The only significant correlations were between Leptin with adiponectin levels, as well as Prolactin with FSH levels (Table 2). (Figure 2a, b) shows the positive correlations between Prolactin with FSH levels as well as Leptin and adiponectin levels. Figure 2c shows the negative correlations between adiponectin and BMI.

Table 1: Characteristics Of Subjects

Parameter	Cases (N=119)	Controls (N=119)	p-value
Prolactin	12.94± 5.64	11.43± 5.12	0.0001
FSH	4.74± 1.23	7.64± 1.52	0.0001
LH	9.83±4.58	5.32±1.05	0.0001
Adiponectin	8.20±3.57	9.64±3.03	0.0009
Leptin	15.82±4.95	11.63±6.32	0.0001
BMI	25.27±4.39	22.23±4.43	0.001

Table 2: Correlation Between Variables:

Parameters	LH	FSH	Prolactin	Adiponectin	Leptin	BMI
LH	1.000	-.013	-.135	-.030	.153	-.085
FSH	-	1.000	.0202**	-.029	-.052	.002
Prolactin	-	-	1.000**	.127	-.007	.032
Adiponectin	-	-	-	1.000*	.205*	-.243**
Leptin	-	-	-	-	1.000	-.051
BMI	-	-	-	-	-	1.000

*p<0.05 significant, ***

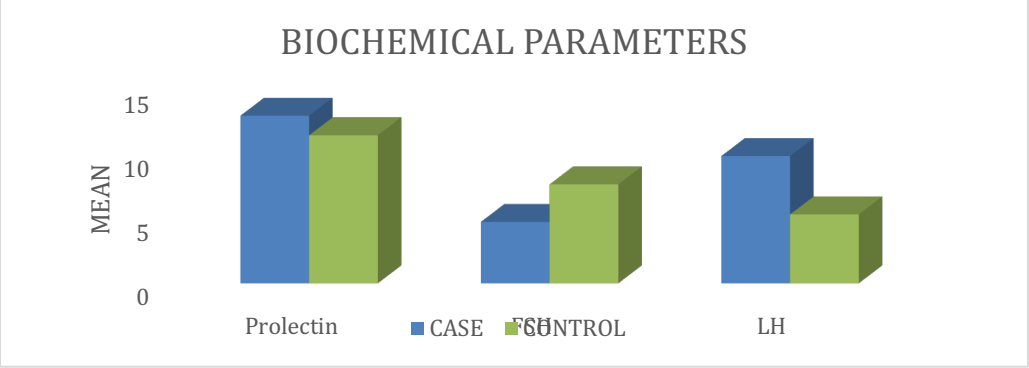

Figure 1a-: Comparison of Biochemical Parameters in Controls and Cases

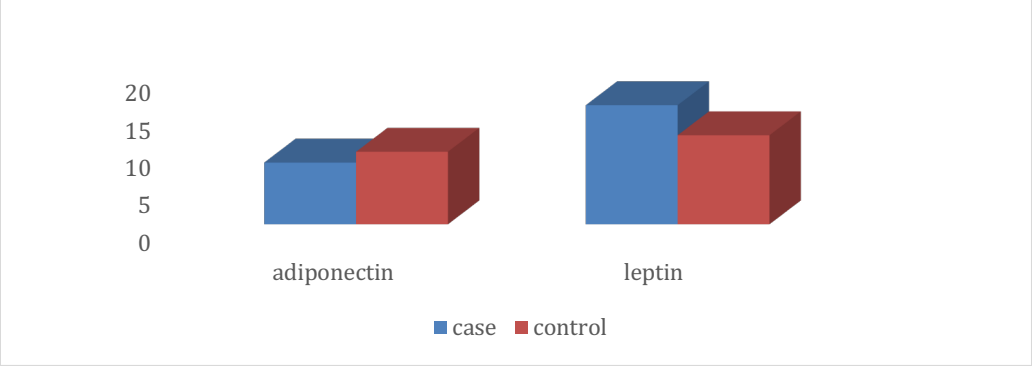

Figure 1b-: Comparison of Adiponectin & Leptin in Controls and Cases

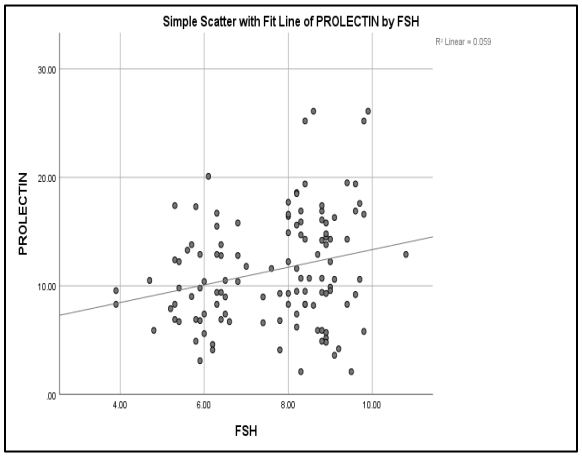

Figure 2A: Correlation Between Prolactin and FSH

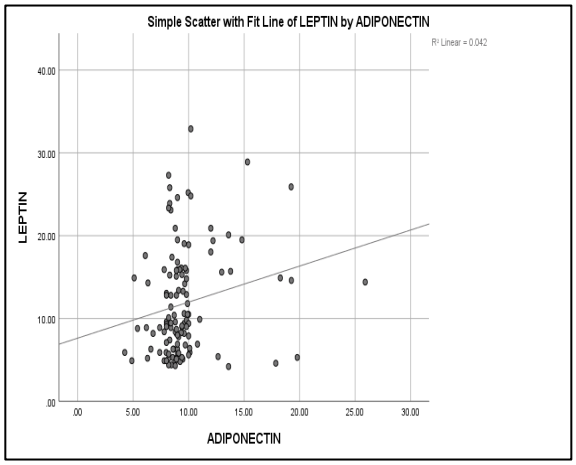

Figure 2B: Correlation Between Leptin and Adiponectin

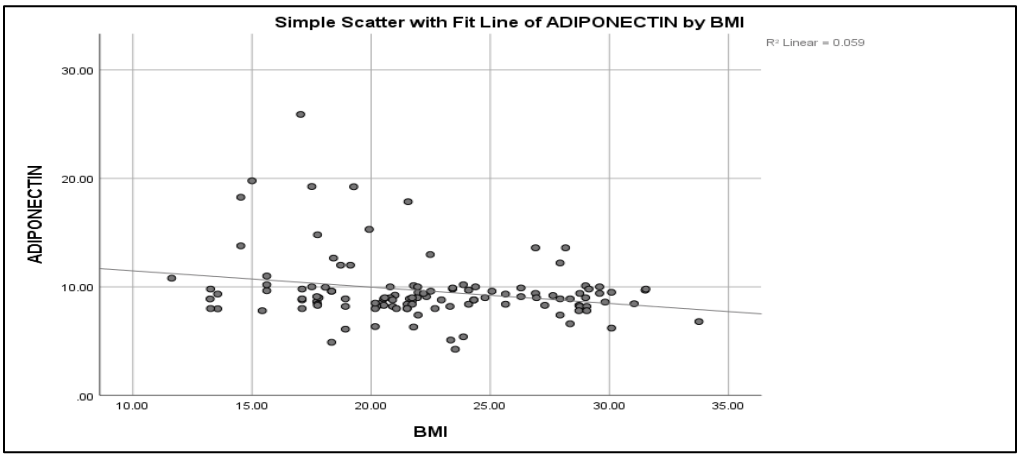

Figure 2 c: correlation between adiponectin and BMI

DISCUSSION

This research indicated that FSH and adiponectin concentrations were reduced, while LH, Prolactin, leptin, and BMI levels were elevated in PCOS patients compare to healthy controls. These results align with other studies indicating that a deficiency in adiponectin may have beneficial implications for antidiabetic effects and enhancing insulin sensitivity. Significant insulin resistance has been seen in conjunction with obesity in women diagnosed with PCOS. Numerous studies in the literature indicate that decreased adiponectin levels in women with PCOS correlate negatively with insulin resistance and positively with obesity [9,10]. Conversely, some research did not find a statistically significant correlation between adiponectin levels and PCOS [11,12,13]. Limited research has shown that the relationship between adiponectin levels and PCOS is independent of BMI and the degree of insulin resistance [14,15].

The current investigation could not find a standalone correlation between adiponectin levels and women diagnosed with PCOS. Consequently, it seems that reduced serum adiponectin levels are primarily linked to obesity rather than to PCOS. The hormone adiponectin, produced by adipose tissue, significantly contributes to the development of PCOS. Adiponectin has

antiatherogenic, antidiabetic, anti-inflammatory, and insulin-sensitizing properties, and its levels are negatively correlated with obesity in healthy persons.

Adiponectin exhibits several beneficial metabolic activities, including anti-atherogenic, antidiabetic, anti-inflammatory, and insulin-sensitizing effects. Furthermore, its circulating concentrations demonstrate an inverse association with obesity among metabolically healthy individuals.

The investigation of women diagnosed with polycystic ovary syndrome (PCOS) provides an important framework for elucidating the complex regulatory interactions between leptin metabolism and ovarian physiology. Several clinical investigations have documented a statistically significant increase in circulating serum leptin concentrations among women with PCOS [16,17,18]. Nevertheless, contradictory findings have also been reported, with certain studies failing to demonstrate a comparable association [19,20].

Sustained elevation of circulating leptin concentrations, commonly referred to as hyperleptinemia, may potentially induce leptin receptor desensitization and impair downstream intracellular signaling mechanisms. Such alterations could contribute to a disruption in the physiological relationship between circulating leptin levels and adipose tissue mass in women affected by PCOS. Moreover, androgen excess has been suggested to exert an inhibitory influence on leptin secretion from adipose tissue, indicating a possible interaction between reproductive hormones and adipokine regulation.

Leptin is an important adipocyte-derived hormone that participates in the regulation of energy homeostasis and modulates several aspects of reproductive physiology. Polycystic ovary syndrome (PCOS), a prevalent endocrine and metabolic disorder associated with ovulatory dysfunction and infertility, is primarily characterized by persistent anovulation, hyperandrogenism, insulin resistance, and a substantial prevalence of obesity. Consequently, alterations in leptin secretion and signaling may contribute to the complex pathophysiological mechanisms underlying PCOS (21,22).

Furthermore, previous research has emphasized the necessity of conducting additional investigations to establish the independent associations between circulating leptin concentrations and other biochemical as well as clinical parameters in women with PCOS. Such investigations may provide a more comprehensive understanding of the potential involvement of leptin in the metabolic and reproductive abnormalities associated with this syndrome (23).

CONCLUSION

In this study, FSH and adiponectin levels were shown lower while LH, Prolactin and Leptin levels were higher in PCOS patients compared to healthy controls. Leptin concentrations were higher in obese subjects in both the PCOS and control groups. Leptin is involved in metabolic and reproductive regulation and may contribute to PCOS through its association with obesity, insulin resistance, and altered adipose tissue function. Increased leptin levels and leptin resistance may influence the progression of metabolic and hormonal disturbances associated with PCOS. Further studies are required to clarify its exact role and therapeutic significance. Adiponectin levels were significantly lower in the PCOS group compared with healthy controls. Adiponectin contributes to insulin sensitivity, inflammatory regulation, and androgen metabolism. Reduced adiponectin levels may therefore be associated with metabolic and reproductive abnormalities in PCOS. Further research is needed to understand its underlying mechanisms and potential role as a therapeutic target.

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