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The Value of Serum Adiponectin in Patients with Polycystic Ovary Syndrome

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common condition in women at reproductive age associated with reproductive and metabolic dysfunction. It may be the most common cause of anovulation, early pregnancy loss, and later pregnancy complications. Adiponectin is the most abundant adipokine and is mainly secreted from visceral fat cells. It might be responsible for the metabolic and neuroendocrine derangements characteristic of obesity and obesity-related disease, such as PCOS. We aimed to evaluate the level of serum adiponectin in PCOS and the potential use of adiponectin as a biomarker for PCOS.
Methods: This case control study was carried on 100 patients, aged between 20–35 years, who were equally divided into four groups based on the diagnosis of PCOS; 2 case groups and 2 control groups. Group 1 were non-obese PCOS subjects with body mass index (BMI) <25 kg/m2. Group 2 were obese PCOS subjects with BMI >25 kg/m^2. Control groups were selected as; group 3 were non-obese control group with BMI <25 kg/m^2. Group 4 were obese control group with BMI >25 kg/m^2.
Results: Adiponectin was significantly lower in group 1 than group 3 and 4 (P2 and P3 <0.001). While it was significantly lower in group 2 than group 1, 3 and 4 and was significantly lower in group 4 than group 3 (P1 = 0.021, P4 and P5 <0.001).

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Conclusion: Serum adiponectin level may be taken into consideration as a biomarker for confirmation of PCOS diagnosis. The relationship between adiponectin and BMI suggests that adiponectin could serve as a marker for disease risk and provide opportunity for earlier intervention.

Keywords: Serum adiponectin; polycystic ovary syndrome; obesity; biomarker.

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common condition in women at reproductive age associated with reproductive and metabolic dysfunction. It is a frequently encountered as an endocrinopathy that occurs in 15–20% of reproductive age women [1].

PCOS is a complex disorder with multisystem involvement, in addition to ovulatory dysfunction and dysregulated androgen biosynthesis. This syndrome is also characterized by obesity, increased central adiposity, insulin resistance (IR), and glucose intolerance [2].

Polycystic ovary syndrome may be the most common cause of anovulation, early pregnancy loss, and later pregnancy complications. All have been implicated in the low fecundity of women with this disorder [3].

PCOS is associated with an increased risk of metabolic syndrome (MS) and type 2 diabetes mellitus (T2DM) [4].

It has become clear that adipose tissue is not merely an inert reserve of triglycerides, but rather an active endocrine organ that oversees energy metabolism. This seems to be accomplished among other means by the action of so-called adipocytokines biological molecules which are secreted and most likely contribute to peripheral insulin sensitivity [5].

In recent years, role of adipose tissue hormones, particularly adiponectin has been implicated in the pathogenesis of PCOS [6].

Adiponectin is the most abundant adipokine and is mainly secreted from visceral fat cells [7].

Adiponectin is a protein of 247 amino acids consisting of four domains, with a molecular weight of 30 kDa, and it has insulin-sensitizing effect [8]. Adiponectin has antiatherogenic, antidiabetic, anti-inflammatory and insulin sensitizing effects, and is negatively related to the degree of adiposity in healthy individuals [9].

Because adiponectin is a fat cell product, secreted into the circulating blood, it might be responsible for the metabolic and neuroendocrine derangements characteristic of obesity and obesity-related disease, such as PCOS. Studies have also shown that both insulin action and circulating levels of adiponectin are lower in women with PCOS [10].

Also, adiponectin modulates a number of metabolic processes, including glucose regulation and fatty acid oxidation [11].

In vitro and in vivo studies have shown that adiponectin has beneficial effects on the reproductive processes and an important relationship with the gonadotropins and other hormones [12].

The aim of this study was to evaluate the level of serum adiponectin in polycystic ovarian syndrome (PCOS) and the potential use of adiponectin as a biomarker for PCOS and to compare adiponectin levels in women with PCOS to those of non PCOS women.

2. PATIENTS AND METHODS

After research ethical committee approval from research center of Tanta University and informed written consent from all participants in this research, this case control study was carried on 100 patients at Tanta University Hospital – Gynecology department from August 2019 to August 2020.

The study included patients aged between 20–35 years that administered no PCOS treatment for the last 3 months, had normal thyroid function, normal prolactin serum levels and with no other diseases causing ovulatory dysfunction.

While pregnant PCOS patients and those with diabetes, chronic liver diseases and impaired renal function were excluded.

The women studied were equally divided into four groups based on the diagnosis of PCOS; 2 case groups and 2 control groups. **Group 1** were non-obese PCOS subjects with body mass index

(BMI) <25 kg/m^2. **Group 2** were obese PCOS subjects with BMI >25 kg/m^2. Control groups were selected as; **group 3** were non-obese control group with BMI <25 kg/m^2. **Group 4** were obese control group with BMI >25 kg/m^2.

All women included in the study provided personal, menstrual, obstetric and medical illness history.

General examination as well as anthropometric measurements (weight, height, BMI, waist circumference, hip circumference waist-hi ratio, blood pressure) were done.

Serum adiponectin was measured using human adiponectin (ADP) ELISA Kit (catalogue number 201-12-1551, China).

Transvaginal ultrasound, laboratory and other routine investigations (complete blood count, fasting blood sugar, renal and liver functions and coagulation profile) were performed.

Statistical Analysis: Statistical analysis was performed using the Statistical Package for the Social Sciences version 25 (IBM Inc., Chicago, IL, USA). Quantitative variables were expressed as mean, standard deviation (SD) and range and were compared using F test among the three groups with post hoc (Tukey test) test to compare each two groups. Qualitative variables were expressed as frequency and percentage and were statistically analyzed by Chi-square test. A two-tailed P value ≤ 0.05 was considered statistically significant.

3. RESULTS

Parity and Menses were significantly different among four groups (P <0.001). But age and marital were insignificantly different among four groups Table 1.

Parity was significantly lower in group 1 than group 4 (P3 = 0.001) and was significantly lower in group 2 than group 3 and 4. (P4 = 0.001. P5 = 0.001). But was insignificantly different between group 1 and 2, group 1 and group 3 and group 3 and 4 Table 1.

Menstrual irregularities were significantly higher in group 1 than group 3 and group 4 and were significantly higher in group 2 than group 3 and group 4 (P2, P3, P4 and P5 <0.001). But were insignificantly different between group 1 and 2 and between group 3 and 4 Table 1.

All anthropometric measurements were significantly different among four groups (P <0.001) except height was insignificantly different Table 2.

Table 1. Demographic data among four groups

		Group 1 (n=25)	Group 2 (n=25)	Group 3 (n=25)	Group 4 (n=25)	p-value		
Age (years)	Mean ± SD	2 ± 8.24 3.71	2 ± 7.68 3.79	2 ± 7.68 3.33	2 ± 7.92 2.81	0.930	---	
	Range	2 35 – 22	2 35 – 22	2 33 – 32	2 32 – 42	0.979	---	
Marital Status	Married	0 (80%)	0 (80%)	1 (84%)	2 (80%)			
	Not Married	5 (20%) 1	5 (20%) 1	4 (16%) 2	5 (20%) 3	>0.001*		
	Nulligravida (NG)	0 (40%)	3 (52%)	2 (8%)	3 (12%)		1	0.658
	Virgin	5 (20%)	5 (20%)	5 (20%)	5 (20%)		2	1
	Para 1	8 (32%)	5 (20%)	3 (12%)	1 (4%)		3	*0.001
	Para 2	2 (8%)	2 (8%)	11 (44%)	8 (32%)		4	*0.001
	Para 3	0 (0%)	0 (0%)	3 (12%)	8 (32%)		5	*0.001
	Para 4	0 (0%)	0 (0%)	1 (4%)	0 (0%)		6	0.790
	Normal	0 (0%)	0 (0%)	18 (72%)	2 (80%) 0	>0.001*	1	0.603
	Oligomenorrhea	16 (64%)	15 (60%)	0 (0%)	2 (8%)		2	>0.001*
	Polymenorrhea	3 (12%)	1 (4%)	7 (28%)	3 (12%)		3	>0.001*
	Amenorrhea	6 (24%)	9 (36%)	0 (0%)	0 (0%)		4	>0.001* 0.404

** Significant as P value < 0.05; P1, P value between group 1 and group 2; P2, P value between group 1 and group 3; P3, P value between group 1 and group 4; P4, P value between group 2 and group 3; P5, P value between group 2 and group 4; P6, P value between group 3 and group 4*

Table 2. Anthropometric measurements among four groups

		Group 1 (n=25)	Group 2 (n=25)	Group 3 (n=25)	Group 4 (n=25)	p-value		
Weight (kg)	Mean ± SD	59.5.51 ± 88	8 ± 8.56 15.15	5 ± 9.99 9.14	83 ± .24 11.08	0.001*	1	0.001*
	Range	50 – 71	6 140 – 2	2 74 – 3.8	61 – 106		2	
							3	0.001*
							4	0.001*
							5	0.307
							6	0.001*
Height (cm)	Mean ± SD	162 4.75 ± .12	1 ± 60.84 5.61	1 ± 61.48 5.68	16 ± 1.00 5.03	0.828	---	
	Range	153 – 171	1 175 – 51	1 – 52 173	15 171 – 0			
BMI (kg/m²)	Mean ± SD	22. 1.03 ± 80	3 ± 4.14 4.71	2 ± 3.40 1.02	32 ± .11 3.60	0.001*	1	0.001*
	Range	20. 24.7 – 8	2 – 6.5 45.7	2 – 1.4 24.8	26 37.1 – .3		2	0.884
							3	0.001*
							4	0.001*
							5	0.071
							6	0.001*
Waist circumference (cm)	Mean ± SD	80. 3.85 ± 88	9 ± 3.44 7.44	8 ± 1.40 4.47	93 ± .88 5.50	0.001*	1	0.001*
	Range	70 – 87	8 121 – 5	7 88 – 0	86 – 105		2	0.987
							3	0.001*
							4	0.001*
							5	0.992
							6	0.001*
Hip circumference (cm)	Mean ± SD	97. 3.40 ± 56	1 ± 05.44 8.75	1 ± 00.64 3.59	10 ± 6.68 5.12	0.001*	1	0.001*
	Range	90 – 104	9 140 – 5	9 107 – 5	99 – 116		2	0.222
							3	0.001*
							4	0.017*
							5	0.865
							6	0.002*
Waist-Hip Ratio	Mean ± SD	0.8 0.06 ± 3	0. 0.03 ± 89	0 ± .82 0.04	0. 0.03 ± 87	0.001*	1	0.001*
	Range	0.6 0.92 – 2	0.097 – 84	0 – 0.75 0.88	0.092 – 81		2	0.076

	Group 1 (n=25)	Group 2 (n=25)	Group 3 (n=25)	Group 4 (n=25)	p-value		
						3	0.001*
						4	0.014*
						5	0.759
						6	0.001*

**, Significant as P value < 0.05; P1, P value between group 1 and group 2; P2, P value between group 1 and group 3; P3, P value between group 1 and group 4; P4, P value between group 2 and group 3; P5, P value between group 2 and group 4; P6, P value between group 3 and group 4*

Weight, BMI, waist circumference and waist-hip ratio were significantly lower in group 1 than group 2 and group 4, and were significantly higher in group 2 than group 3, and were significantly lower in group 3 than group 4 (P1, P3, P4 and P6 <0.001). But were insignificantly different between group 1 and group 3 and between group 2 and group 4 Table 2.

Hip circumference was significantly lower in group 1 than group 2 and group 4 and was significantly lower in group 3 than group 4 (P1 and P3<0.001. P6 = 0.002) But was insignificantly different between group 1 and group 3 and between group 2 and group 4 Table 2.

LH, serum Estradiol and total testosterone were significantly different among four groups. (P <0.001). FSH, prolactin, TSH, free thyroxine and fasting blood glucose insignificantly different among four groups Table 3.

LH was significantly lower in group 1 than group 2 and higher than group 3 and 4. (P1 = 0.001, P2=0.004 and P3 = 0.001). And was higher in group 2 than group 3 and 4 (P4 and P5 <0.001). But was insignificantly different between group 3 and four Table 3.

Estradiol was significantly lower in group 1 than group 2 and higher than group 3 and 4. (P1 < 0.001, P2 < 0.001 and P3 > 0.001). And was higher in group 2 than group 3 and 4 (P4 and P5 <0.001). But was insignificantly different between group 3 and four Table 3.

Total testosterone was significantly lower in group 1 than group 2 and higher than group 3 and 4. (P1 = 0.001, P2 < 0.001 and P3 > 0.001). And was higher in group 2 than group 3 and 4 (P4 and P5 <0.001). But was insignificantly different between group 3 and 4 Table 3.

Adiponectin ranged between 0.81 and 4.85 with a mean value of 1.99 ± 0.86 at group 1, ranged between 0.42 and 1.5 with a mean value of 0.82 ± 0.27 at group 2. ranged between 3.71 and 19.96 with a mean value of 10.14 ± 3.80 at group 3 And ranged between 3.06 and 7.93 with a mean value of 5.16 ± 1.40. Adiponectin was significantly lower in group 1 than group 3 and group 4 (P2 and P3 <0.001) and was significantly lower in group 2 than group 1, 3 and 4. (P1 = 0.021, P4 and P5 <0.001). And was significantly lower in group 4 than group 3 (P6 <0.001) Table 4 and Fig. 1.

There was negative significant correlation between adiponectin and BMI, LH and E2 (P <0.001, 0.032 and 0.006 respectively). But there was insignificant correlation between adiponectin and age, FSH, Total testosterone and Prolactin Table 5.

The ROC curve revealed that the cutoff value of ≤2.7 adiponectin for prediction of PCO yielded a sensitivity of 96%, a specificity of 100%, a PPV of 100% and a NPV of 96.2% with AUC of 0.993 and P <0.001 Fig. 2.

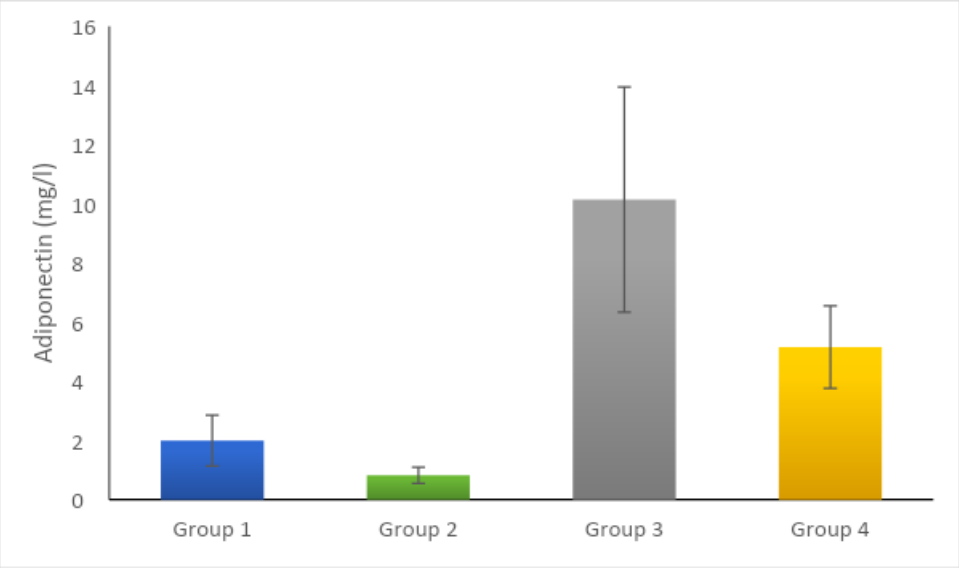

Fig. 1. Adiponectin among four groups

Table 3. Laboratory Investigations among four groups

		Group 1 (n=25)	Group 2 (n=25)	Group 3 (n=25)	Group 4 (n=25)	p-value		
FSH (mIU/ml)	Mean ± SD Range	5.5 1.71 ± 2 1.7 – 8.5	5. 1.60 ± 11 2. 9.3 – 4	5 ± .62 1.63 3 9.1 – .6	5. ± 06 1.36 3. 8.6 – 19	0.434	---	
LH (ml) (U/ml)	Mean ± SD Range	10. 3.39 ± 34 4.1 16.8 – 6	13 ± .62 3.84 6. 21.5 – 42	7 ± .25 2.46 3 11 – .8	7. ± 09 1.97 4. 11.2 – 2	0.001*	1 2 3 4 5 6	*0.001 *0.004 *0.001 *0.001 *0.001 0.992
Prolactin (ng/ml)	Mean ± SD Range	13. 5.30 ± 49 6.2 – 22.6	11 ± .44 4.40 3. 19.7 – 4	1 4.5 ± 1.5 2 19.6 – 0.6	1 ± 3.15 4.12 5. 20.5 – 3	0.258	---	
Serum Estradiol (pg/ml)	Mean ± SD Range	45. 21.08 ± 51 11. 86 – 6	68 ± .31 16.80 33 100 – .6	2 ± 5.66 9.43 1 – 2.6 48.2	2 ± 5.43 8.51 1 45 - 4.3	0.001*	1 2 3 4 5 6	0.001* 0.001* 0.001* 0.001* 0.001* -
TSH (µIU/ml)	Mean ± SD Range	2.3 0.79 ± 1 0.8 3.5 – 1	2. 1.01 ± 24 0. 3.9 – 4	2 ± .22 0.75 0 3.4 – .41	2. ± 42 0.65 1. 3.6 – 24	0.884	---	
Free Thyroxine (ng/dl)	Mean ± SD Range	1.3 0.13 ± 6 1.1 1.58 – 6	1. 0.73 ± 42 1 – 4.8	1 ± .32 0.17 1 – .02 1.58	1. ± 35 0.12 0. 1.7 – 9	0.857	---	
Fasting blood glucose (mg/dL)	Mean ± SD Range	87. 7.12 ± 44 76 – 98	87 ± .40 5.82 78 – 97	8 ± 4.60 5.73 7 95 – 5	8 ± 7.80 6.06 7 97 - 6	0.239	---	
Total testosterone (ng/ml)	Mean ± SD	0.9 0.33 ± 2	1. 0.75 ± 51	0 ± .29 0.16	0. ± 31 0.19	0.001*	1 2 3 4 5	*0.001 *0.001 *0.001 *0.001 *0.001
	Range	0.2 1.67 – 6	0. 3.78 – 48	0 – .12 0.65	0. – 08 0.81		6	-

*FSH: Follicular stimulating hormone, LH: Luteinizing hormone, TSH: Thyroid stimulating hormone, * significant as P value < 0.05; P1, P value between group 1 and group 2; P2, P value between group 1 and group 3; P3, P value between group 1 and group 4; P4, P value between group 2 and group 3; P5, P value between group 2 and group 4; P6, P value between group 3 and group 4*

Table 4. Adiponectin levels among four groups

		Group 1 (n=25)	Group 2 (n=25)	Group 3 (n=25)	Group 4 (n=25)	p-value		
Adiponectin (mg/l)	Mean ± SD	1. 0.86 ± 99	0.8 0.27 ± 2	10. 3.80 ± 14	5.1 1.40 ± 6	0.001*	1	*0.021
	Range	0. 4.85 – 81	0.4 1.5 – 2	3.7 19.96 – 1	3.0 7.93 – 6		2	0.001*
							3	0.001*
							4	0.001*
							5	0.001*
							6	0.001*

Significant as P value < 0.05; P1, P value between group 1 and group 2; P2, P value between group 1 and group 3; P3, P value between group 1 and group 4; P4, P value between group 2 and group 3; P5, P value between group 2 and group 4; P6, P value between group 3 and group 4

Table 5. Correlation between adiponectin and different parameter in all PCOS cases

	Adiponectin	
	r	**p-value**
Age (years)	0.184	0.201
BMI (kg/m2)	0.642-	*0.001>
FSH (mIU/ml)	0.118	0.416
LH (mIU/ml)	0.303-	*0.032
E2	0.383-	*0.006
TSH (µIU/ml)	0.279	0.050
Total testosterone (ng/ml)	0.237-	0.097
Prolactin (ng/ml)	0.131	0.364

*r: Pearson correlation; *: significant as p value < 0.05*

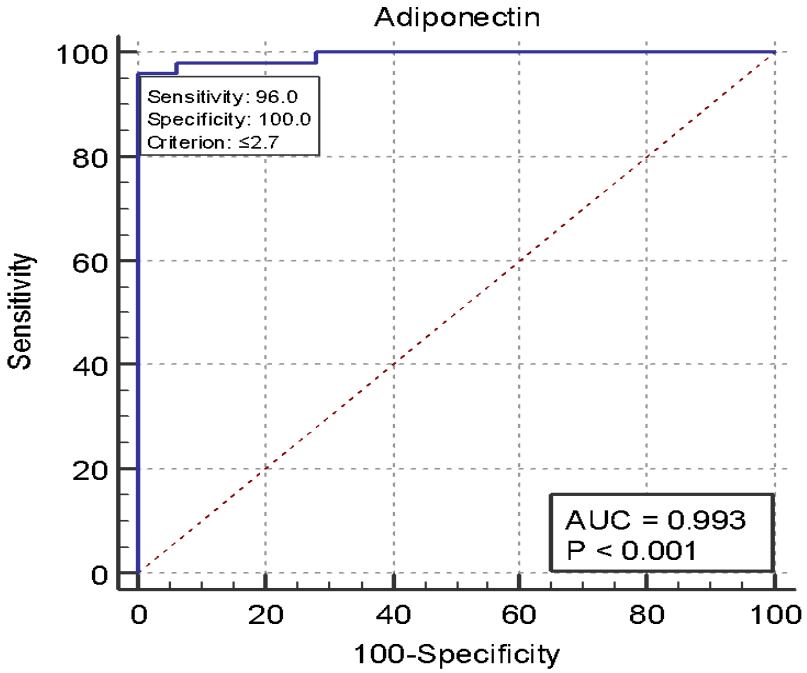

Fig. 2. ROC curve of adiponectin for prediction of PCOS

4. DISCUSSION

Adipose tissue hormones, notably adiponectin, have been implicated in the etiology of PCOS in recent years. The most abundant adipokine is adiponectin, which is mostly released by visceral fat cells [6].

Regarding anthropometric measurements among study groups; statistical analysis of current study stated that weight, BMI, waist circumference, hip circumference and waist-hip ratio were significantly higher in PCOS than control groups except height that was insignificantly different. Panidis et al. [13] agreed with current study and stated that the mean BMI was significantly higher in group I (obese PCOS) compared with group II (P < 0.001) and group III (P < 0.001). Escobar-Morreale et al. [14] conducted a cross-sectional case–control study to evaluate the possible involvement of Adiponectin and Resistin in the pathogenesis of polycystic ovary syndrome (PCOS). They disagreed with our current study and stated that there was no interaction between PCOS or control status and degree of obesity, indicating that the influence of PCOS on the variables studied here was not different in lean women compared with overweight and obese women and that the influence of the degree of obesity on the dependent variables was the same in the PCOS and control groups. Alfaqih et al. [15] disagreed with current study and stated that no significant difference in BMI was observed between PCOS and normally menstruating women.

Regarding laboratory investigations among study groups; statistical analysis of current study stated that LH, serum estradiol and total testosterone were significantly different among study groups. LH, serum estradiol and total testosterone were significantly higher in PCOS than control groups and higher with obesity.

Panidis et al. [13] agreed with current study and that LH levels were significantly higher in women with PCOS and normal BMI (group 2) compared with women having PCOS + BMI >25 kg/m2 and controls. However, they disagreed with current study and noted that LH levels did not differ significantly between groups 1 and 3. Also, they agreed with us and stated that compared with controls, women with PCOS had significantly higher serum levels of testosterone, D4-androstenedione, 17a-OH-progesterone and DHEA-S (group 1 versus 3, group 2 versus 3 in all comparisons). But they disagreed with the finding the serum levels of the above hormones did not differ significantly between groups I and II as regard BMI.

Amer et al. [16] agreed with current study and stated that no significant difference is found between cases and controls regarding their hormonal profile except for testosterone and insulin levels which were significantly higher among cases. However, our study didn't evaluate insulin levels. Also, regarding the hormonal profile, there is a significant difference between obese and non-obese concerning LH and testosterone. Olszanecka-Glinianowicz et al. [17] agreed with current results and stated that in both PCOS subgroups serum testosterone and free testosterone levels were significantly increased when compared to the controls.

On the other hand, statistical analysis of current study stated that FSH, prolactin, TSH, free thyroxine and fasting blood glucose were insignificantly different among study groups and there was an insignificant correlation between adiponectin and age, FSH, total testosterone and prolactin.

Amer et al. [16] agreed with current study and stated that there is no significant difference between obese and non-obese regarding FSH and E2.

Yildiz et al. [18] disagreed with current study and stated that significant reverse correlation was observed between adiponectin level and prolactin level.

Olszanecka-Glinianowicz et al. [17] disagreed with current results and stated that in the PCOS group positive linear correlations were found between circulating adiponectin concentrations and plasma FSH.

While Adiponectin was significantly lower in PCOS than control groups and lower with obesity. Also, there was a significant correlation between adiponectin and BMI, LH and E2. As regard to correlation between adiponectin and different parameter in all PCOS cases, there was a negative correlation between adiponectin and BMI, LH and E2.

Escobar-Morreale et al. [14] agreed with current study and stated that PCOS patients presented with reduced serum adiponectin levels compared with healthy controls. but they were against us and noted that serum adiponectin levels were independent of the degree of obesity. Amer et al. [16] agreed with current study and stated that serum adiponectin level is significantly lower among cases than control. The adiponectin is a significantly lower in the obese cases than non-obese PCOS cases.

Yildiz et al. [18] investigated the relationship between adiponectin, metabolic and hormonal parameters, and insulin resistance in patients with non-treated polycystic ovary syndrome. They agreed with current study and stated that there is a significant reverse correlation between serum adiponectin and BMI for patients with PCOS.

Alfaqih et al. [15] agreed with current study and stated that in normally menstruating women, the mean adiponectin levels were 7.916 ± 0.3970 mg/mL. The serum adiponectin levels in PCOS women were significantly lower.

Shirazi in 2021 [19] has showed in his cross-sectional study a high incidence of insulin resistance in PCOS patients independent of obesity, and determined BMI related lower level of high molecular weight adiponectin in obese PCOS individuals.

Olszanecka-Glinianowicz et al. [17] disagreed with current results and stated that the circulating concentration of adiponectin didn't differ between normal weight PCOS and controls. But they agreed with us and stated that the adiponectin was the lowest in the obese PCOS subgroup when compared with both the normal weight PCOS subgroup and the controls. They didn't

observe differences in these parameters between the normal weight PCOS subgroup and the controls. They also agreed with current results and stated that in the PCOS group positive linear correlations were found between circulating adiponectin concentrations and plasma LH levels.

In Groth's 2010 study [20], the link between adiponectin and PCOS as well as insulin resistance, sensitivity, metabolic syndrome, and BMI in female patients suggested that adiponectin potentially could serve as a marker for PCOS risk and provide opportunity for earlier intervention if knowledge is successfully translated from laboratory to clinical practice.

Chen et al. [21] agreed with current study and stated that adiponectin was negatively correlated with insulin resistance, body mass index (BMI), and total testosterone. The adiponectin was significantly lower in PCOS women than in those without PCOS.

Regarding accuracy of adiponectin for prediction of PCOS among study groups; the ROC curve revealed that the cutoff value of ≤2.7 adiponectin for prediction of PCOS yielded a sensitivity of 96%, a specificity of 100%, a PPV of 100% and a NPV of 96.2% with AUC of 0.993. According to our knowledge there were no previous studies assessed this result.

5. CONCLUSION

Serum adiponectin level may be taken into consideration as a biomarker for confirmation of PCOS diagnosis. The relationship between adiponectin and BMI suggests that adiponectin could serve as a marker for disease risk and provide opportunity for earlier intervention.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the authors.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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