Kirkuk Journal of Science Vol. 19, Iss. 4, p. 16-28, 2024 doi:10.32894/kujss.2024.152183.1166

Evaluation of Adiponectin Hormone and its Gene Polymorphism in Obese Women with Polycystic Ovary Syndrome.

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Article Information

Article Type:
Research Article

Keywords:
adiponectin; adiponectin gene promoter (+276 T / G) polymorphism; Oxidative stress and antioxidant parameters; Biochemical parameters.

History:
Received: 22 July 2024
Reviced: 1 November 2024
Accepted: 2 November 2024
Published: 30 December 2024

Citation: Naznaz Hussein Othman, Kalthum Assaf Maulood and Suhaila Nafee Darogha, Evaluation of Adiponectin Hormone and its Gene Polymorphism in Obese Women with Polycystic Ovary Syndrome, Kirkuk Journal of Science, 19(4), p.16-28, 2024, https://doi.org/10.32894/kujss.2024.152183.1166

Abstract

Polycystic ovary syndrome (PCOS), is a complex condition that affects women of reproductive age, women with PCOS present a prevalence of obesity. Current study aims to evaluate the adiponectin level and its gene polymorphism (+276 T /G). The patients and control women samples were collected from May 2022 to March 2023. 140 samples were collected ages from 18 to 40 years old from the Maternity Teaching Hospital, they divided into two groups: Group I: 70 obese control and Group II: 70 obese PCOS. Using PCR for allele-specific amplification refractory mutation system (ARMS). Serum specimens were collected for the measurement of many biochemical parameters. Results showed that adiponectin levels in PCOS decreased significantly, while leptin increased significantly when compared to control. HOMA-IR, Glucose, Insulin, and Testosterone parameters increased significantly in PCOS, while SHBG increased non-significantly. MDA and vit. D parameters increased significantly in PCOS, while vit.C showed a significant decrease. Serum cholesterol level was increased non-significantly in patients with PCOS group as compared to the control group, HDL was decreased significantly in PCOS women, while non-significant increase was observed in LDL, TG, and VLDL as compared to control women. The participants with homozygous for the G allele of the adiponectin (+276 T/G) had a significant and increased risk of PCOS with a significant decrease in adiponectin in PCOS. This study concluded that the decreased serum adiponectin can serve as a valuable biomarker for diagnostic PCOS in obese women, and G allele of the adiponectin showed an increased risk for PCOS.

1. Introduction:

Polycystic ovary syndrome (PCOS) is a complex condition that affects women of reproductive age and is characterized by ovulatory dysfunction and androgen excess [1]. PCOS is a chronic endocrine disorder that affects 10 to 15% of women worldwide [2]. It has been linked to metabolic diseases, such as diabetes, obesity, and insulin resistance (IR). The prevalence of obesity in women with PCOS is estimated at 49% worldwide [3]. When a person has a body mass index (BMI) of 30 kg/m2 or more, they are considered obese [4]. It is linked to the buildup of body fat, which is linked, either directly or indirectly to detrimental health impacts (Heart et al., 1998). There is a consensus that, from an epidemiological genetic standpoint, obesity and PCOS are closely related. About the former, between 38 and 88% of women with PCOS were obese [5]. Several genetic pathways linked to steroid production, insulin signaling, cell communication, reproductive function, and carbohydrate metabolism might all have a

significant impact on the pathophysiology of PCOS [6].

Obese women with PCOS exhibit a more severe phenotype that includes infertility, glucose intolerance and/or type 2 diabetes, metabolic syndrome, induced hypertension, preterm, and biochemical and clinical hyperandrogenism [7]. Adipokines, a variety of bioactive compounds released by adipose tissue have been revealed, adipokines act widely on different parts of the human body, regulating and controlling glucose and fatty acid metabolism, energy expenditure, inflammatory response, cardiovascular function, reproduction, and other biological processes over the entire body or locally [8]. Lin et al 2021 revealed that non-obese PCOS patients have a notably reduced amount of circulating adiponectin in comparison to non-obese subjects [9]. Adiponectin in PCOS patients might be useful in diagnosing or treating this complicated reproductive disease [10]. Data indicates that women with PCOS may have excess androgen due to insufficient adiponectin [11].

Oxidants are controlled by antioxidant enzymes and scavenge free radicals [12]. The total antioxidant capacity (TAC) measures how well antioxidants can protect cells from damage caused by oxidative stress. People with PCOS tend to have lower TAC levels, which suggests they have more oxidative stress [13]. Both a deficiency in antioxidant defense mechanisms and a proliferation of reactive oxygen species are responsible for oxidative stress [14]. In several pathological states, it is known that the change in redox balance in impacted fluids, tissues, or organs results in variations in antioxidant activity [15] .Up to 70% of women with PCOS have dyslipidemia [16].

Women with PCOS have lower concentrations of HDL and higher levels of plasma triglycerides (TG) [17]. Dyslipidemia in PCOS linked to a high prevalence of insulin resistance [18]. Numerous studies have examined the role of leptin in PCOS by comparing the serum levels of this adipokine in women with and without PCOS and hyperandrogenism [19]. A meta-analysis revealed that the PCOS group had higher blood levels of leptin than the control group [20]. Both the thin and obese categories of PCOS patients had elevated blood levels of leptin [21]. The current study aimed to explore whether obese women with PCOS have any difference in adiponectin and leptin levels compared to control group. Also to study the genetic polymorphism of adiponectin gene (+276 T/G) in PCOS.

2. Methodology:

2.1 Sample and Study Design:

Seventy women with PCOS patients conducted in maternity and teaching hospital in Erbil city were diagnosed by physicians, sonography and the history as well as a clinical laboratory assessment. Seventy healthy women who did not exhibit any evident indications of hirsutism or acne and had normal menstrual periods made up the control group. Every PCOS patient fulfilled the 1990 National Institute of Health (NIH) requirements [22] and thus had persistent oligoovulation, hyperandrogenism (hirsutism), and/or hyperandrogenemia.

The patients and controls women samples were collected during period May 2022 to March 2023, which consists of 140 participate women, the ages of patients and control women ranged from 18 to 40 years old. These were divided into two groups: Group I: 70 obese women without PCOS (control group) and Group II: 70 obese women with PCOS (PCOS group). The recruited women were counseled and written informed consent such as (age, body mass index (BMI) and waist circumference).

2.2 Blood Sample Collection:

Using a disposable syringe, 10 ml of venous blood was extracted from each participant and divided into two aliquots. A simple tube was used to pour the initial portion (7 ml). After 15 minutes at 3000 rpm, it was centrifuged. The second aliquot (3 ml) was transferred to EDTA tube for gene polymorphism.

2.3 Hormonal Assay and Biochemical Analysis:

1- Determination of Serum Adiponectin and Leptin

Sera of patients and controls was assessed for the level of adiponectin and leptin using commercially available kits (Elabscieence Biotechnology, USA) by ELISA.

2- Determination of Glucose and Insulin Resistance

Serum glucose and insulin were analyzed by using of Cobas 6000 (Roche, Switzerland) immunoassay analyzers, Insulin resistance was evaluated using the usual formula for HOMA-IR calculations: (Glucose (mg/dL) x Insulin (μu/L))/405 [23].

3- Determination of Oxidative Stress and Antioxidant

Before starting the procedure, ELISA kit components were left at room temperature. Sera of patients and controls were assessed for the levels of malondialdehyde (MDA) and vitamin C (Vit. C) using commercially available kits (Elabscieence Biotechnology, USA) by ELISA.

4- Determination of Lipid Profile

The serum was used for assessment of lipid profile of cholesterol, triglyceride (TG), low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol by using of Cobas 6000 (Roche, Switzerland) immunoassay analyzers.

5- Determination of Thyroid Hormones

Serum levels of thyroid-stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4), sex hormone binding

globulin (SHBG) and vit. D by using Cobas 6000 (Roche, Switzerland) immunoassay analyzers by ELISA.

2.4 Molecular Analysis DNA Extraction:

DNA from whole blood was extracted with PureLink^{TM} Genomic DNA Mini Kit from Thermo Fisher, USA. The following steps showed the DNA extraction steps according to the manufacturer's instructions.

2.5 Quantification and Qualification:

Quantification and qualification of total DNA concentration was performed by using One Drop TOUCH Nano Drop with absorbance wavelengths of A 260/280 ratio and a ratio of 1.7 is generally accepted as "pure" for DNA.

2.6 ARMS PCR:

A straightforward technique for identifying any known mutations involving single base changes is the amplification refractory mutation system (ARMS), also known as allele-specific PCR for ADIPO gene polymorphism. It is based on the use of sequence-specific PCR primers, which permit amplification of the DNA only when the target allele is present in the sample. All that is required for the ARMS approach is PCR amplification and amplicon gel electrophoresis. By using ARMS-PCR, the targeted areas of ADIPOQ were amplified. The PCR (ADIPOQ), primers were employed and the protocols are listed in the Tables 1, 2 below.

Table 1. Thermocycler Condition.

Components	Volume(μL)
Master Mix	10
Forward Primer(N)	1
Forward Primer(M)	1
Reverse Primer	1
d H_2O	4
Template (DNA)	3
Total Volume	20

2.7 Adiponectine (+276 T/G) Genotype:

Agarose gel electrophoresis (2%) was employed to check the efficiency of PCR reactions and stained with ethidium bromide (EtBr) to make the DNA visible under UV light. The power supply condition was set at 100V for 40 minutes. The expected fragment sizes of the bands are indicated in Figure 1.

2.8 Statistical Analysis:

Graph Pad Prism version 9 and MedCalc version 18 were used for the data analysis. Chi-square statistics analyzed the demographic characteristics. The Kruskal Wallis test calculated Mean±SD, p-value0.05 was considered as significant also, the predictive significance of the study determined severity via Receiver Operator Characteristic (ROC) Curve analysis. The Hardy-Weinberg equilibrium was estimated using the H-W calculator for two alleles. Using stepwise multiple regression modeling to assess factors affecting serum adiponectin and leptin levels.

3. Results:

3.1 Baseline Patient Characteristics:

Mean age of the all cases 18-40 years and the mean age of patients and control group were 28.20±5.755 and 30.71± 6.181 respectively the mean baseline of BMI was 31.82±5.110 and 30.78± 5.843 kg/m^2, and WHR 0.84±0.053 and 0.84±0.048 respectively. As in Table 3.

3.2 Adiponectin and Leptin:

Serum adiponectin levels in the PCOS group decreased significantly when compared to the control group at values 1.997±0.199 and 3.044±0.178 respectively. The level of serum leptin increased significantly from 625.9±152.7 in the control group to 724.3±184.4 in the PCOS group as shown in Table 4. According to area under the curve (AUC) values, serum adiponectin and leptin exhibit good markers for PCOS patients. The AUC of serum adiponectin and leptin were 0.784, p<0.001 and 0.631, p<0.078, sensitivity% were 52.9, 86.9 and specificity% were 90.0, and 40.0 respectively as shown in Figure 2.

3.3 Glucose and Hormones:

Table 5 demonstrated that Insulin, HOMA-IR, and SHBG increased non-significantly in PCOS patient as value (156.7 ± 158.4), (39.72±56.93) and (1.608±0.066) respectively, when compared to control women while, Glucose, testosterone, TSH, T3, and T4 parameters increased significantly in PCOS patient as value (95.85±32.44), (0.440±0.020), (2.446±1.459), (3.044±1.494) and (108.4±23.71) than control group respectively. According to the area under the curve values, HOMA-IR, Glucose, and Insulin exhibit a good marker for PCOS patients. The AUC of HOMA-IR was 0.597, Glucose was 0.603, Insulin was 0.563, Testosterone was 0.576 and SHBG was 0.511 and TSH was 0.576, T4 was 0.563 and T3 was 0.563 respectively and sensitivity% of glucose, HOMA-IR, TSH, and T4 were 94.3, 90, 68.6, 66.1 and specificity % were 39.4, 31.8, 71.2, 47 respectively as showed in Figure 3 and Table 6.

3.4 Oxidative Stress and Anti-Oxidant:

Level of MDA and vit. D increased significantly in the PCOS group (938.7±75.10) and (25.16±1.054) compared with the control group (511.8±115.6) and (22.41±1.018).

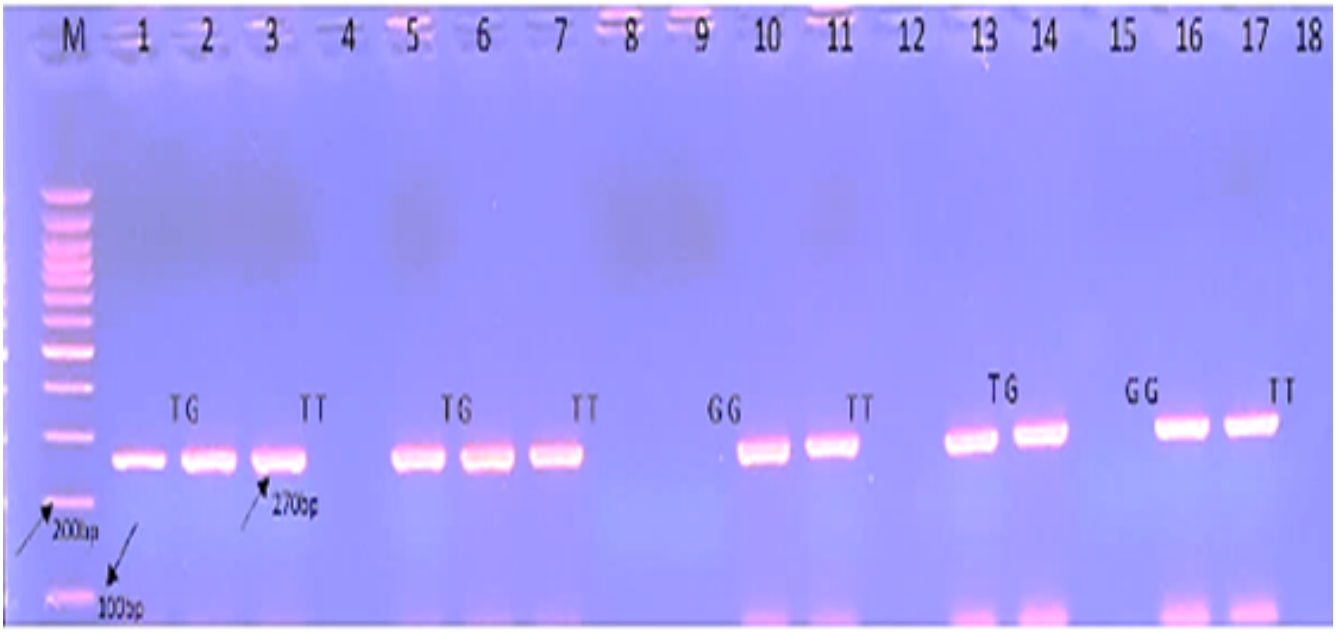

Figure 1. Agarose gel electrophoresis illustrating PCR products for the ADIPOQ (+276 T/G). Ethidium bromide used to make the DNA visible under UV light with PCR products of 270bps.M stands for DNA Marker (Ladder) 100bps, T/T: Wild type homozygous, T/G: Mutant heterozygous, G/G: Mutant homozygous.

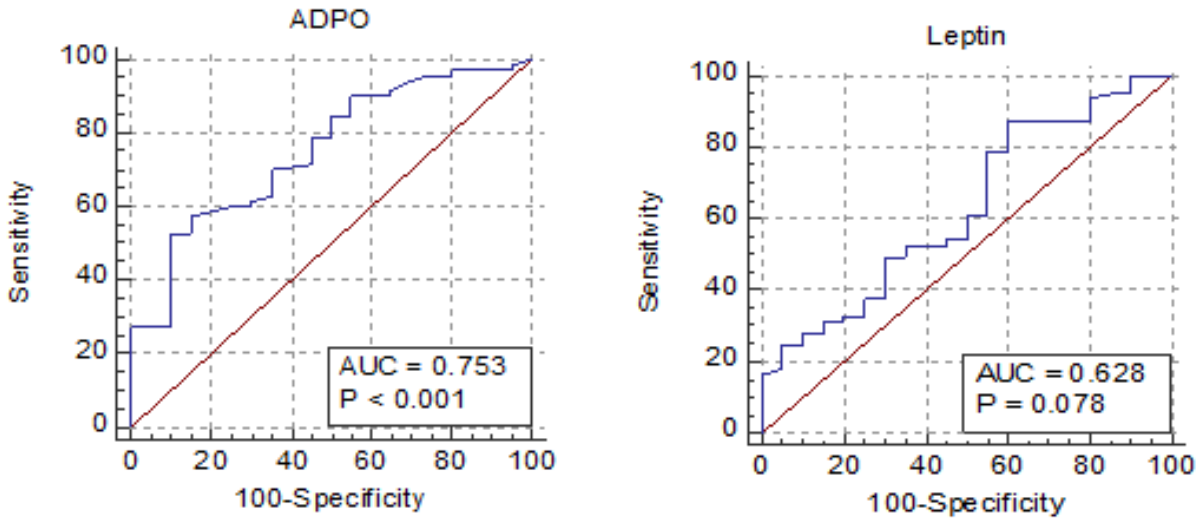

Figure 2. Agarose gel electrophoresis illustrating PCR products for the ADIPOQ (+276 T/G). Ethidium bromide used to make the DNA visible under UV light with PCR products of 270bps.M stands for DNA Marker (Ladder) 100bps, T/T: Wild type homozygous, T/G: Mutant heterozygous, G/G: Mutant homozygous.

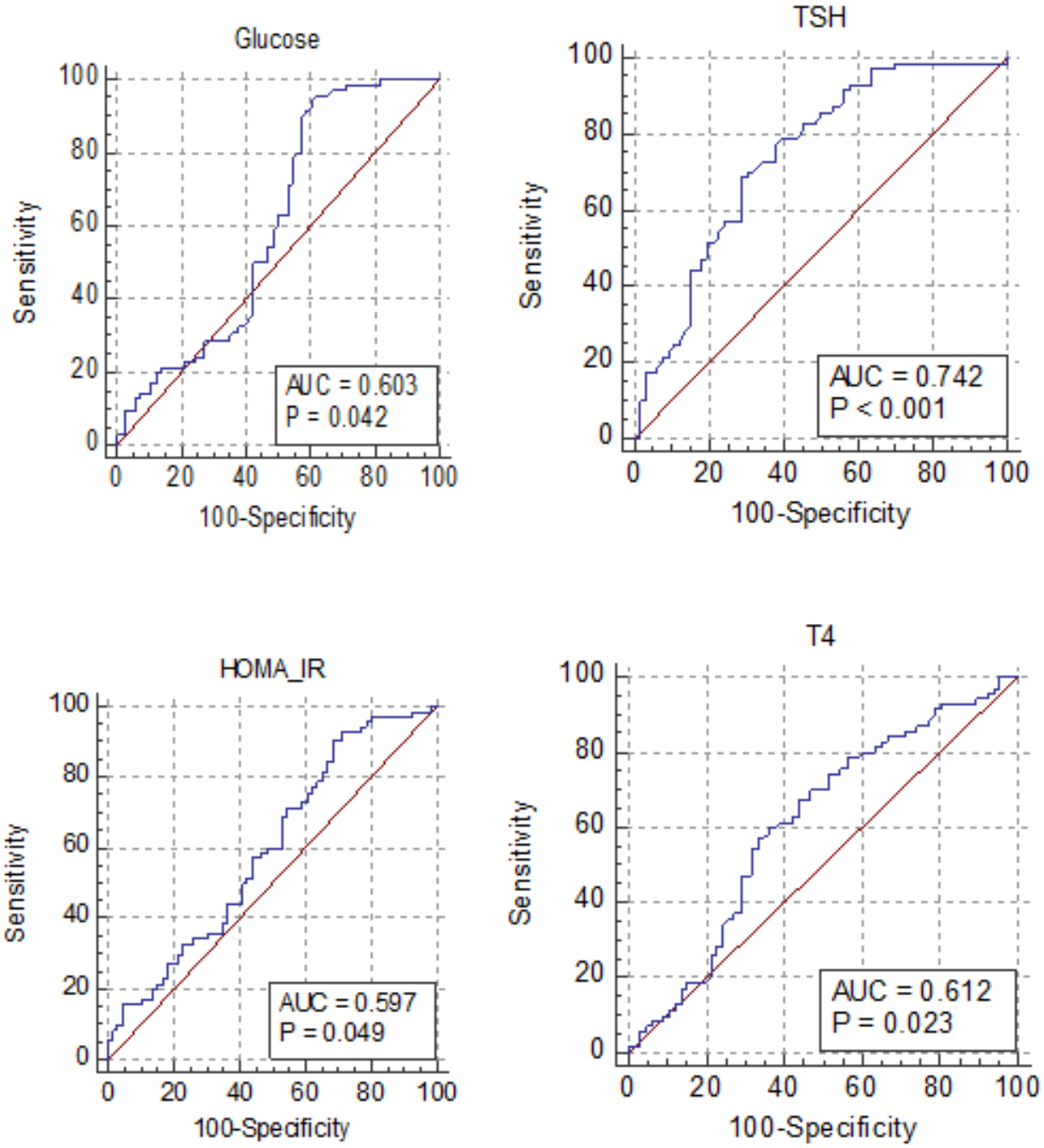

Figure 3. AUC value of glucose, HOMA-IR, TSH, and T4.

Table 2. Designed primer sequences used in ARMS-PCR genotyping detection and interpretation.

No.	Primer Name	Primer Sequence 5'-3'	bp	Amplicon Size
1	ADIPOQ 1F	CTGCTATTAGCTCTGCCCGGT	21	270 bp
2	ADIPOQ 2F	CTGCTATTAGCTCTGCCCGGG	21	430 bp
3	ADIPOQ R	CATCACAGACCTCCTACACTGATA	24	

Table 3. Designed Primer Sequences used in ARMS-PCR Genotyping Detection and Interpretation.

Group of demographic	Patient	Control	p. value
Age (years)	28.20±5.755	30.71± 6.181	NS
BMI (kg/m^2)	31.82±5.110	30.78± 5.843	NS
WHR(cm)	0.84±0.053	0.84±0.048	NS

BMI=body mass index WHR=waist hip ratio

Table 4. The mean± SD of adiponectin and leptin level in the patients group and control group.

Parameters	PCOS	Control	P- value
ADIPO (μg/mL)	1.997±0.199	3.044±0.178	0.000
Leptin (ng/mL)	724.3±184.4	625.9±152.7	0.058

Data expressed as mean± standard Deviation; ADIPO, Adiponectin.

Table 5. The mean± SD of glucose, insulin resistance, HOMA-IR, Testosterone, SHBG, TSH, T3 and T4 levels in the patient and control group.

Parameters	PCOS	Control	P- value
Glucose (mmol/L)	95.85±32.44	84.09±20.88	0.038
Insulin (μU/mL)	156.7±158.4	107.2±71.75	NS
HOMA-IR	39.72±56.93	23.96±18.84	NS
Testosterone (ng/dL)	0.440±0.020	0.381±0.025	0.045
SHBG (nmol/L)	1.608±0.066	1.492±0.078	NS
TSH (mIU/L)	2.446±1.459	1.474±1.262	0.000
T3 (ng/dL)	3.044±1.494	1.997±0.846	0.002
T4 (ng/dL)	108.4±23.71	99.45±27.57	0.024

Table 6. Area under the curve of insulin, testosterone SHBG and T3.

Variable	AUC	Sensitivity%	Specificity%	Confidence interval	%95 P -value
Insulin	0.563	92.86	22.73	0.475 to 0.648	0.204
Testosterone	0.576	92.9	30.3	0.488 to 0.660	0.130
SHBG	0.512	98.6	15.0	0.404 to 0.618	0.882
T3	0.563	67.1	47	0.476 to 0.648	0.201

While vit. C decreased significantly (0.175±0.005), as compared to the control group (0.207±0.007). as shown in table 7. According to the area under the curve values, serum MDA, vit. D and Vit. C exhibits a good marker for PCOS patients. The AUC of serum MDA was 0.692, Vit. D was 0.655 and Vit. C was 0.721 with p value<0.996, p<0.001, and p <0.001 respectively as shown in Figure 4.

3.5 Lipid Profile:

Serum cholesterol level was increased non-significantly in patients with PCOS group (136.8+4.055) as compared to the control group, HDL was decreased significantly (37.67+1.106) in PCOS women, while non-significant increase observed in level of LDL (78.00+2.977), TG (135.5+7.392) and VLDL (27.35+0.811) as compared to control women Table 8. According to the area under the curve values, serum lipid profile levels were exhibiting a good marker for PCOS patients. The AUC of serum Cholesterol was 0.554, TG was 0.522, LDL was 0.532, HDL was 0.589, and VLDL was 0.522 respectively and HDL sensitivity% was 57.1 and specificity % was 62.1. Figure 5 and Table 9.

3.6 Molecular Analysis:

Our results showed that the numbers of individuals of TT, TG, and GG genotypes of ADIPOQ (+276T/G) (rs2241766) were 27(38.5%), 34(48.5%), and 9(12.8%) in the case PCOS group and 52(74.2%), 12(17.1%) and 3(4.2%) in the control group, respectively. Moreover, the frequencies of the T and G alleles were 88(125.7%) and 52(74.2%) in the case PCOS group and 116(165.7%) and 18(25.7%) in the control group, respectively. Table 10. The genotype frequencies of

Table 7. Oxidative Stress and Anti-Oxidant Parameters.

Parameters	PCOS	Control	P- value
MDA (μmol/l)	938.7±75.10	511.8±115.6	0.005
Vit. D (ng/mL)	25.16v±1.054	22.41±1.018	0.003
SHBG 0.404 to 0.618	0.512 0.882	98.6	15.0
T3 0.476 to 0.648	0.563 0.201	67.1	47
vit. C(mg/dL)	0.175±0.005	0.207±0.007	0.002

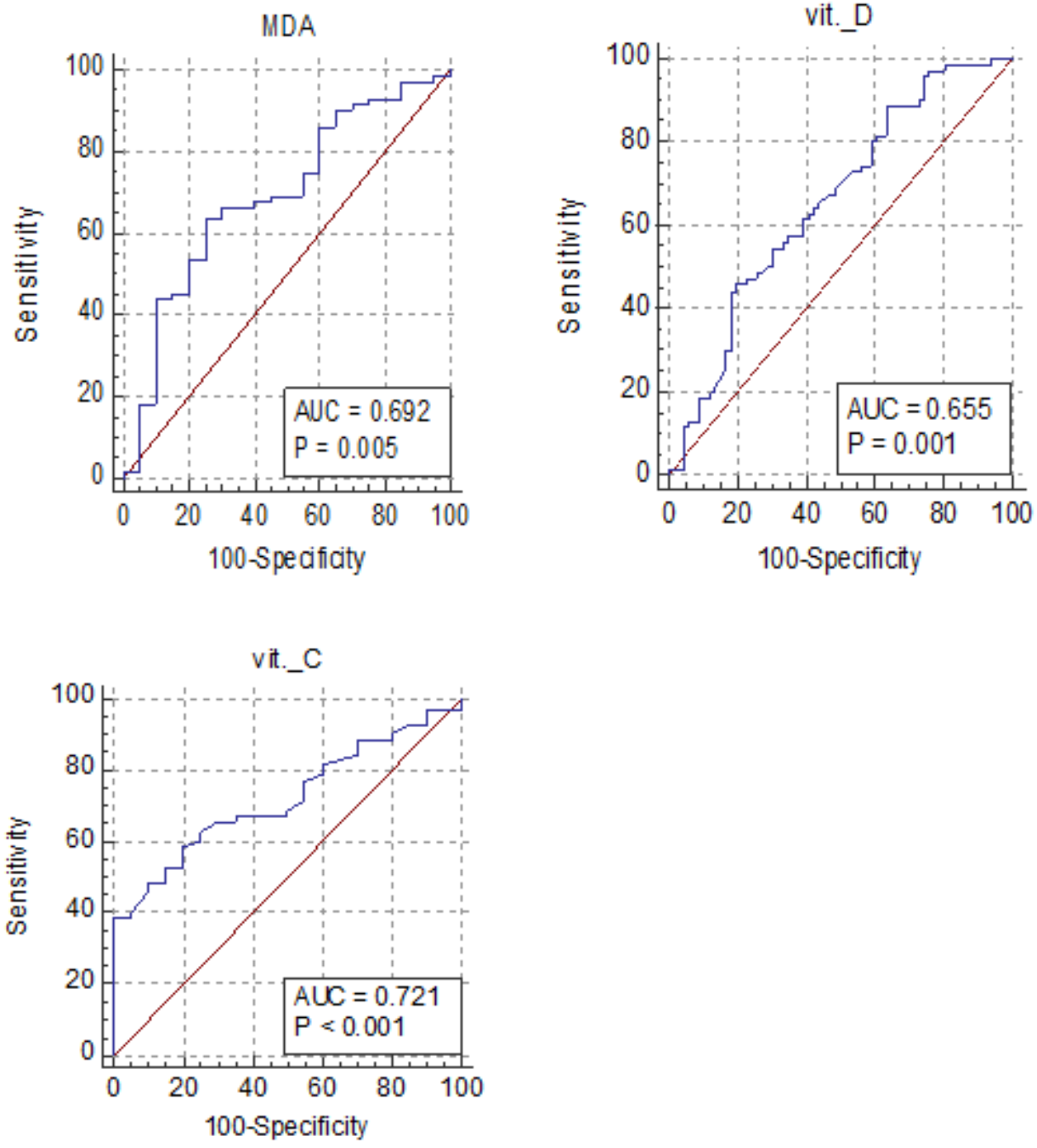

Figure 4. AUC value of serum Oxidative stress and anti-oxidant parameters in PCOS and control group.

Table 8. Oxidative Stress and Anti-Oxidant Parameters.

Parameters	PCOS	Control	P- value
Cholesterol (mg/dL)	136.8±4.055	132.1±3.039	NS
TG (mg/dL)	135.5±.392	119.8±5.408	NS
LDL (mg/dL)	78.00±2.977	72.78±2.240	NS
HDL (mg/dL)	37.67±1.106	40.93±1.263	0.050
VLDL(mg/dL)	27.35±0.811	26.43±0.607	NS

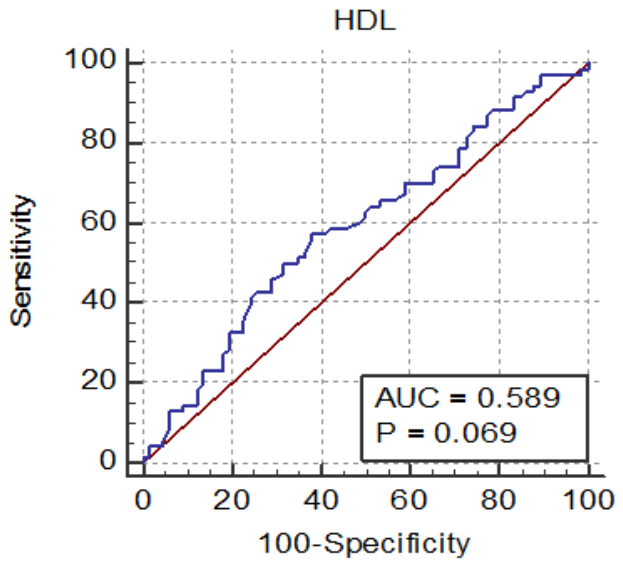

Figure 5. AUC value of Lipid profile (HDL) parameter in PCOS and control.

ADIPOQ among the category of PCOS were assessed by the Hardy-Weinberg equilibrium (HWE) calculation. The differences in frequency of heterozygous genotype (TG) between observed and expected in the PCOS group were 34(48.5%), 32.69(46.7%), 12(17.1%) and 15.58(22.2%), in the control group respectively. The variance between the observed and expected values of genotype frequencies was statistically non-significant, indicating that the distribution of this cohort was under HWE Table 11.

4. Discussion:

Polycystic ovary syndrome still does not have a clear cause, and subjective phenotypes make it hard to make a complete diagnosis. The main finding of the present study was the relationship between ADPO and Leptin in PCOS. Women with PCOS had significantly lower mean in serum ADIPOQ than control women, which is in agreement with [24]. It was hypothesized that this might be because to variations in the distribution of adipose tissue [25].

Excessive body fat and adipose tissue malfunction cause PCOS and reduced expression of adiponectin, it also found that PCOS women with average body mass indexes below 25 had lower levels of adiponectin [26]. [27] concluded that adiponectin may be a useful biomarker in the diagnosis of PCOS, which may corroborate the results of the current study however, the study analysis of serum adiponectin showed no differences between PCOS and control groups, however, there was a direct relationship between this hormone and body fat percentage values [28]. One possible mechanism by which obesity influences the development of insulin resistance is obesity-associated downregulation of adiponectin. After correcting for BMI, the findings of multivariate regression analysis support this link and show that insulin resistance as measured by HOMA-IR is an independent predictor of adiponectin [25].

In the current study level of leptin in the PCOS group increased significantly when compared with the control group, There is evidence that adipose tissue malfunction and increased body fat lead to an excess of leptin production in PCOS [26]. According to a study, leptin levels in obese PCOS women are not different from those in control subjects, indicating that leptin may be involved in a physiological feedback loop that prevents obesity [29]. Nevertheless, a number of lines of evidence point to a more comprehensive physiological role for leptin, one that has a major impact on the reproductive system and may be crucial in the pathophysiology of PCOS [30]. Present study showed that women with PCOS had a significantly higher mean level of testosterone, TSH,T3 and T4 than control women, these results were in line with [31].

The current findings showed that the patient group had much higher levels of TSH and low level of triiodothyronine indicated that PCOS women had hypothyroidism as evidenced by higher TSH level [32], A meta-analysis study of relationship between SHBG and PCOS revealed a significant correlation between low SHBG and obesity, glucose intolerance, insulin resistance and hyperandrogenism [33].

This result is in dis agreement with the present study. Current study revealed that women with PCOS had considerably higher levels of insulin and HOMA-IR, two markers of insulin resistance these findings align with [25]. Oxidative stress alters biological molecules, and over time, these alterations build up in the biological structures, which might harm cellular and tissue structures at the molecular level [34] and [35].

The antioxidant defense system may be weakened by the decrease of follicular function. Oxridative stress may therefore harm the ovarian cell. A proinflammatory condition brought on by oxidative stress may be linked to PCOS's hyperandrogenism and insulin resistanc. [34] and [35].The current result showed that the level of MDA increased significantly between PCOS patient and control group and this may increase

Table 9. Oxidative Stress and Anti-Oxidant Parameters.

Variable	AUC	Sensitivity%	Specificity%	Confidence interval %95	P- value
Cholesterol	0.554	71.43	43.94	0.466 to 0.639	0.281
TG	0.522	25.7	86.4	0.435 to 0.608	0.6582
LDL	0.532	62.86	53.03	0.445 to 0.618	0.521
VLDL	0.515	26.47	87.30	0.426 to 0.603	0.772

Table 10. The genotype and allele frequency of ADIPO in PCOS and control group.

ADIPOQ (rs2241766)	patient N%	Control N%	Relative Risk	Etiology or Preventive Fraction	Exact Fishers Probability	95% Confidence Intervals
Genotype						
TT	27(38.5%)	52(74.2%)	0.18	0.63	0.000	0.09-0.38
TG	34(48.5%)	12(17.1%)	4.33	0.37	0.000	1.99-9.40
GG	9(12.8%)	3(4.2%)	3.15	0.08	0.129	0.82-12.06
Allele						
T allele	88(125.7%)	116(165.7%)	0.26	0.63	0.000	0.14-0.48
G allele	52(74.2%)	18(25.7%)	3.81	0.27	0.000	2.09-6.95

RR: Relative risk,
CI: Confidence Intervals,
Exact Fishers Probability (P).

oxidative stress because of obesity. Current results showed that Patients with PCOS have significant increase in, vit. D level this result disagreement with [36], [37], which showed lower level of vit.

D in patient with PCOS while, [38] demonstrated that the level of vit. D not changed in PCOS and control group. Vitamin C decreased significantly in PCOS group in the present study its considered as antioxidant and play a protective role against PCOS [39]. Current results demonstrated that the lipid parameters which include, total cholesterol was non significantly increased in PCOS group but, HDL was decreased significantly in PCOS women while, no significance was seen in the levels of LDL, TG and VLDL, these results were found in study done by [24] and [40].

According to molecular analysis, the current study showed that the TG genotype was relative risk for PCOS and G allele was heterozygous for PCOS, this result is in agreement with [41] and dis agreement with [42]. Zhang et al 2008 demonstrated that SNPs 276 (T/G) in the ADIPO gene was strongly associated with PCOS and conferred to IR and insulin action, which suggests that adiponectin genetic variation may play a common role in the pathogenesis of PCOS and other complex diseases such as type 2 diabetes and obesity [43].

5. Conclusion:

This study concluded that the decreased serum adiponectin can serve as valuable biomarker for detecting PCOS in obese women. According to thyroid stimulating hormone, T4 and T3 levels showed an important indicator in PCOS patients, an elevated level of MDA and decreased level of vitamin C among PCOS patients may improve the management of PCOS. Molecular analysis showed that the frequency of TG genotype was more relative risk of PCOS than GG and TT and G allele was an increased risk for PCOS.

Table 11. The genotype and allele frequency of ADIPO in PCOS and control group.

ADIPOQ gene at position +276 T/G (rs2241766)							
Case		Genotypes			HWE	Alleles	
Categories		TT	TG	GG	p. value	T	G
PCOS	Observed	27(38.5%)	34(48.5%)	9(12.8%)	0.736	88(125.7%)	52(74.2%)
patients	Expected	27.66(39.5%)	32.69(46.7%)	9.66(13.8%)		Not estimated	
Control	Observed	52(74.2%)	12(17.1%)	3(4.2%)	0.059	116(165.7%)	18(25.7%)
	Expected	50.21(71.7%)	15.58(22.2%)	1.21(1.7%)		Not estimated	

Funding: None.

Data Availability Statement: All of the data supporting the findings of the presented study are available from corresponding author on request.

Declarations:

Conflict of interest: The authors declare that they have no conflict of interest.

Ethical approval: The manuscript has not been published or submitted to another journal, nor is it under review.

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تقييم هرمون الأديبونيكتين وتعدد أشكاله الجينية لدى النساء البدينات المصابات بمتلازمة المبيض المتعدد الكيسات

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الخلاصة

متلازمة المبيض المتعدد الكيسات *PCOS* وهي حالة معقدة تؤثر على النساء في سن الإنجاب. الغرض من البحث الحالي هو دراسة العلاقة المحتملة بين متلازمة تكيس المبايض ومصل الأديبونيكتين مع تعدد الأشكال الجينية $+276G/T$ تم جمع عينات النساء من المرضى والسيطرة خلال الفترة من مايو 2022 إلى مارس 2023 . 140 شارك في هذه الدراسة امرأة وتراوحت أعمار من 18 إلى 40 سنة. تم تقسيم عددهم 140 شخصًا إلى مجموعتين: المجموعة الأولى: 70 مجموعة مراقبة يعانون من السمنة المفرطة والمجموعة الثانية: 70 مجموعة مصابة بمتلازمة تكيس المبايض. استخدام عثغ لنظام طفرة التضخيم الحراري للأليلاغيص. ، تم جمع عينات المصل لقياس العديد من العوامل البيوكيميائية. انخفض الأديبونيكتين في ، في حين ارتفع مستوى الليبتين في الدم بشكل ملحوظ. زادت مؤشرات $HOMA-IR$ والجلوكوز والأنسولين والتستوستيرون بشكل ملحوظ بينما زاد $SHBG$ بشكل غير ملحوظ. MDA وفيتامين. زادت مؤشرات D بشكل ملحوظ ، بينما بالنسبة لفيتامين ث انخفضت بشكل ملحوظ. ارتفع مستوى الكوليسترول بشكل غير ملحوظ، وانخفض حضى بشكل ملحوظ ، في حين لوحظت زيادة غير معنوية في مستوى LDL ، TG و $VLDL$ مقارنة بنساء السيطرة. كان المشاركون الذين لديهم متماثل الزيجوت في أليل G للأديبونيكتين $276+T/$ جم معنويين ويزيدون من خطر الإصابة بمتلازمة تكيس المبايض مع انخفاض كبير في مصل $ADPO$ في مجموعة متلازمة تكيس المبايض. خلصت هذه الدراسة إلى أن الانخفاض الكبير في مستويات الأديبونيكتين في المصل يمكن أن يكون مفيدًا لتشخيص متلازمة تكيس المبايض لدى النساء وأظهر أليل G المتماثل للأديبونيكتين زيادة خطر الإصابة بمتلازمة تكيس المبايض لدى النساء البدينات.

الكلمات الدالة : اديبونيكتين. تعدد الأشكال لمروج الجينات الأديبونيكتين $(+276G/T)$ ؛ الإجهاد التأكسدي والمعلمات المضادة للأكسدة؛ المعلمات البيوكيميائية.

التمويل: لايوجد.

بيان توفر البيانات: جميع البيانات الداعمة لنتائج الدراسة المقدمة يمكن طلبها من المؤلف المسؤول.

اقرارات:

تضارب المصالح: يقر المؤلفون أنه ليس لديهم تضارب في المصالح.

الموافقة الأخلاقية: لم يتم نشر المخطوطة أو تقديمها لمجلة أخرى، كما أنها ليست قيد المراجعة.