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Estimation of Serum Level of Omentin-1 In Hirsute Female: A Single Center Study

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ABSTRACT

Background: Hirsutism in females is defined as the presence of an androgen-dependent pattern of profuse terminal hair. The specific effect of Omentin-1 on hair development is not yet established. Omentin-1, on the other hand, has the potential to stimulate hair growth by either causing vasodilation or activating phosphatidylinositol 3-kinase (PI3K).

Objective: This study aimed to evaluate serum level of omentin-1 in hirsute females in comparison with normal non hirsute.

Patients and methods: This study included 42 females with hirsutism (21 idiopathic, 21 with polycystic ovary syndrome (PCOS) and 42 healthy matched controls. The included patients were chosen randomly from the Laser Unit in Dermatology Department of Mansoura University Hospital's. All patients were subjected to history taking, dermatological and gynecological examination, diagnosis of hirsutism, diagnosis of PCO, hormonal profile, assessment of serum omentin level.

Results: Hirsutism with PCO subgroup showed significantly lower dehydroepiandrosterone sulphate (DHEA-S), significantly higher testosterone (TT), fasting blood glucose (FBG), fasting insulin, HOMA-IR when compared to those with idiopathic hirsutism subgroup ($p<0.001$ for each). Higher omentin-1 level was significantly associated with idiopathic hirsutism when compared to hirsutism with PCO subgroups (mean= 309.7 versus 184.6, $p<0.001$). Detection of omentin-1 level could be used as reliable indicator in discrimination between idiopathic hirsutism and those with PCO subgroups (AUC=0.912) with high sensitivity (95.2%) and specificity (95.2%).

Conclusion: In females with idiopathic hirsutism, serum omentin-1 is increased, and this rise may contribute to excessive hair growth.

Keywords: Hirsutism, Omentin-1 level, Polycystic ovary syndrome.

INTRODUCTION

Hirsutism is a disorder in which excessive terminal hair is distributed in an androgen-dependent pattern in females [(1)].

Between 5% and 10% of women of reproductive age experience this prevalent ailment [(2)].

The quality of life of hirsute women can be negatively influenced in a number of ways, including the female identity and self-image. Untreated hirsutism can cause significant self-esteem loss and psychological morbidity [(2)].

This aberrant hair growth may be brought on by the pilosebaceous unit's sensitivity to a normal serum level of androgen, an increase in circulating androgen, or a combination of both causes [(3)]. Androgens alter the type of hair that is present while also boosting sebum production, hair follicle size, hair fiber diameter, and the percentage of time that terminal hairs are in the anagen (growth) phase [(4)].

Idiopathic hirsutism (IH) and polycystic ovarian syndrome are the two most typical causes of hirsutism [(5)].

About 6% of women of reproductive age have polycystic ovarian syndrome. It is the most prevalent endocrine and metabolic condition characterised by excess testosterone and ovulatory failure [(6)]. In women with polycystic ovarian syndrome (PCOS), both commonness and incidence of the metabolic syndrome are extremely high. The final PCOS phenotype is mostly determined by obesity and increased visceral fat. All of the characteristics of the metabolic syndrome may have developed as a result of androgen excess and insulin resistance [(7)].

Indicative of idiopathic hirsutism is the occurrence of substantial hirsutism in the absence of both ovulatory failure and hyperandrogenemia, which occurs in 50% to 70% of all hirsute women [(4)].

Adipocytokines, which are produced by adipose tissue, help control insulin sensitivity and reproduction. Omentin-1 is an adipokine generated by adipocytes that has anti-inflammatory and insulin-sensitizing properties [(8)].

Omentin-1, a substantial visceral fat secretory adipokine, is mostly secreted by adipose tissue in the omental region and to a much lesser extent in the heart, lung, and intestines. Intestinal lactoferrin receptor, galactofuranose-binding lectin, and endothelial lectin HL-1 are some of its alternate names [(9)]. In human ovarian follicles, omentin-1 is expressed, particularly in granulosa cells [(10)].

Omentin-1's precise impact on hair development is yet unclear. Omentin-1, however, can promote vasodilation or activate a downstream action of phosphatidylinositol 3-kinase (PI3K) [(11)], both of which can stimulate hair growth [(12)].

Vasodilation of the blood vessels and PI3K play a part in promoting hair growth [(13)]. The aim of the study was to evaluate level of omentin-1 in hirsute female in comparison with normal non hirsute women.

Also, to find a possible relation between

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omentin-1 level, severity of hirsutism and PCO.

PATIENTS AND METHODS

Eighty-four persons were included in this study. They were recruited from the Laser Unit in Dermatology, Andrology & STDs Department, Mansoura University Hospitals and from those attending the Outpatient Clinic for causes other than hirsutism.

They were divided into two groups: Group1

that included 42 hirsute females, and **Group 2**, which included 42 healthy matched controls.

Exclusion criteria:

Pregnant or lactating females. Females who have liver, kidney, thyroid, or adrenal issues. Women who had recently started a dietary regimen or those who were cachectic. Females with drug-induced hirsutism as (androgens, minoxidil, danazol, progestins, and estrogen antagonist as clomiphene). Females with BMI > 30.

Methods:

The following procedures were performed on all patients:

A detailed history including age, the length of the condition, the start of puberty, the regularity and length of menstruation, previous gynecological procedures, a family history of PCOS, the distribution of body hair, how often it is removed, and the severity and course of hirsutism, if it is present. Blood pressure and the body mass index (BMI kg/m^2) were assessed.

Any signs of hyperandrogenism, such as female pattern hair loss, acne, seborrhea, and the distribution of terminal hair, were looked for during the dermatological examination.

Hirsutism was diagnosed clinically, and the modified Ferriman Gallwey scoring system was used to classify the severity of the condition [(14)]. Nine androgen dependent sites (lip, chin, chest, upper abdomen, lower abdomen, upper arm, thigh, upper back, and lower back) with scores ranging from zero (no excessive terminal hair growth visible) to four (extensive hair growth visible) for each body part evaluated. A score of up to 36 is achievable, although according to the 95th percentile of data originally gathered by **Ferriman *et al.* [(15)],** a score of less than 8 often denotes hirsutism.

In all instances, either trans-vaginally or trans-abdominally (for virgin females) a gynaecological examination was performed using pelvic abdominal sonography (non-virgin females). The ovarian morphology was carefully evaluated, and the presence, number, and size of oocyte follicles were assessed.

According to Rotterdam criteria, the diagnosis of PCO was made when two out of the following three characteristics were present: (1) Oligoovulation and/or anovulation. (2) Clinical and/or biochemical signs of hyperandrogenism. (3) Polycystic ovaries on ultrasound examination (the presence of 12 or more follicles measuring 2-9 mm in diameter and/or ovarian volume >10 cm^3) [(16)]. Serum total testosterone (TT), 17OH progesterone, dehydroepiandrosterone sulfate (DHEA-S), insulin resistance (HOMA IR & fasting blood glucose), Serum prolactin, serum cortisol, thyroid profile was done in selected cases.

Assessment of serum omentin level:

In the morning, 5 mm venous blood samples were collected from each participant under complete aseptic conditions from the 2nd to the fifth day of the menstrual cycle. Blood was allowed to clot then centrifuged at 480 g for 10 minutes to separate the serum. The separated serum was preserved at −20 °C until the time of the run. Measurement of serum omentin-1 and total testosterone were done by enzyme linked immunosorbent assay (ELISA) using available kits (R & D systems for omentin-1 and Chemux Bioscience for total testosterone) Cat. No E3770HU.

Ethical consent:

The Ethics Committee of Faculty of Medicine, Mansoura University gave its approval for this study (IRB no. MS.20.08.1223), which was carried out in accordance with the guidelines outlined in the Declaration of Helsinki. All study participants gave their informed consents.

Statistical analysis

Statistical package for Social Science (SPSS) was used to update, code, tabulate, and introduce the acquired data to a computer (Armonk, New York: IBM Corp., IBM SPSS Statistics for Windows) version 25.0. Data were given, and the type of data gathered for each parameter was appropriately analyzed. Data normality, the Shapiro test was used to determine whether the data distribution was normal. For parametric numerical data, the mean and standard deviation (SD) and proportion and frequency for non-numerical information.

The statistical significance of the difference between the means of the two research groups was evaluated using the Student t Test. ANOVA was employed to compare the means of the three groups in one direction. To investigate the connection between two qualitative variables, the Chi-Square test was performed. When the predicted count is less than 5 in more than 20% of the cells, the exact test was applied to investigate the association between two qualitative variables. Pearson's correlation was used to determine how strongly two quantitative variables were related.

The degree and direction of the linear link between two variables are determined by the correlation coefficient. A valuable tool for assessing the sensitivity and specificity of quantitative diagnostic measures that divide patients into two groups is the receiver operating characteristic (ROC) curve. The cutoff point that maximised the AUC value was deemed to be the ideal one. According to AUC, a test with an area of more than

0.9 has high accuracy, 0.7 to 0.9 has moderate accuracy, 0.5 to 0.7 has low accuracy, and 0.5 is a result that may have happened by chance. Regression analysis: Using generalised linear models, logistic and ordinal regression analyses were done to predict risk variables. Calculated were the odds ratio and 95% confidence interval. A p-value is deemed significant if it falls within the confidence interval of 0.05.

RESULTS

Table (1) demonstrated that there were no significant differences regarding weight, height and BMI between cases and controls, as well as between idiopathic hirsutism and hirsutism with PCO cases.

Table (1): Comparison of demographic and anthropometric data between studied groups and subgroups

		Control N=42	Hirsutism			P[1]	P[2]
			Total N=42	Idiopathic n=21	with PCO n=21		
Age (years)	**Mean ± SD**	26.8 ± 7.6	25.9 ± 7	26.1 ± 8.4	25.7 ± 5.3	0.591	0.862
Weight (kg)	**Mean ± SD**	77.5 ± 16.1	79.4± 12.6	77 ± 14.5	81.9 ± 10.2	0.543	0.213
Height (cm)	**Mean ± SD**	161.7 ± 9.4	164.1 ±8.6	165 ± 7.4	163.3 ± 9.8	0.220	0.548
BMI (kg/m^2)	**Mean ± SD**	29.7 ± 6.3	29.6 ± 4.8	28.3 ± 5.1	30.8 ± 4.2	0.925	0.087

SD, standard deviation; student t test was used for numerical parameters; Chi square test was used for categorical parameters; p1, comparison between all studied cases and control groups; p2, comparison between idiopathic and PCO associated hirsutism cases.

Among studied hirsutism cases, 16 cases had positive family history (38.1%), 50% had regular menses and 50% had irregular menses. Positive FH and irregular menses were significantly associated with hirsutism with PCO when compared to idiopathic hirsutism (p=0.001, <0.001 respectively) (Table 2).

Table (2): Comparison of family history and menstrual regularity between studied subgroups

		Hirsutism						
		Total N=42		Idiopathic n=21		with PCO n=21		P
		N	%	N	%	N	%	
FH	**Negative**	26	61.9%	18	85.7%	8	38.1%	**0.001**
	Positive	16	38.1%	3	14.3%	13	61.9%	
Menses	**Regular**	21	50%	18	85.7%	3	14.3%	**<0.001**
	Irregular	21	50%	3	14.3%	18	85.7%	

Chi square test was used for categorical parameters.

Among studied hirsutism cases, 20 cases had acne (47.6%), 50% had acanthosis nigricans (AN) and 52.4% had female pattern hair loss (FPHL). Acne, AN, and FPHL were significantly associated with hirsutism with PCO when compared to idiopathic hirsutism (p=0.013, 0.031, 0.002 respectively) (Table 3).

Table (3): Comparison of clinical examination between studied subgroups.

	Control N=42		Hirsutism							
			Total N=42		Idiopathic n=21		with PCO n=21		*P1*	*P2*
	N	%	N	%	N	%	N	%		
Acne	8	19%	20	47.6%	6	28.6%	14	66.7%	**0.005**	**0.013**
AN	-	-	21	50.0%	7	33.3%	14	66.7%	-	**0.031**
FPHL	-	-	22	52.4%	6	28.6%	16	76.2%	-	**0.002**

Chi square test was used for categorical parameters.

Among studied hirsutism cases, 12 cases had mild degree (28.6%), 21 had moderate degree (50%), and 9 cases had severe degree (21.4%). Higher degrees were significantly associated with hirsutism with PCO when compared to idiopathic hirsutism (p<0.001) (Table 4).

Table (4): Comparison of severity of hirsutism between studied subgroups according to modified Ferriman Gallwey scoring system

		Hirsutism						
		Total N=42		Idiopathic n=21		with PCO n=21		p
		N	%	N	%	N	%	
Degree	**Mild**	12	28.6%	12	57.1%	0	0%	**<0.001**
	Moderate	21	50.0%	9	42.9%	12	57.1%	
	Severe	9	21.4%	0	0%	9	42.9%	

Chi square test was used for categorical parameters.

Hirsutism with PCO subgroup showed significantly lower DHEAS, significantly higher TT, FBG, insulin, HOMA-IR when compared to idiopathic hirsutism subgroup (p<0.001 for each) (Table 5).

Table (5): Comparison of laboratory data between studied subgroups.

	Hirsutism						
	Total N=42		Idiopathic n=21		With PCO n=21		p
	Mean	±SD	Mean	±SD	Mean	±SD	
DHEAS (ng/mL)	168.7	40.2	186.2	44.1	151.2	36.2	**<0.001**
TT (ng/mL)	1.0	0.21	0.6	0.11	1.4	0.3	**<0.001**
17 OH (ng/mL)	1.5	0.31	1.4	0.31	1.6	0.4	0.111
FBS (mg/dL)	125.7	30.2	95.1	21.8	156.4	23.6	**<0.001**
Insulin (mμ/mL)	20.2	4.9	16.5	3.7	24.0	5.3	**<0.001**
HOMA-IR	6.5	1.51	3.9	0.9	9.2	1.7	**<0.001**

SD, standard deviation; student t test was used for numerical parameters.

Significant lower omentin-1 level in hirsutism cases when compared to control group (mean=247.1 versus 271.5, p=0.087). While higher omentin-1 level was significantly associated with idiopathic hirsutism when compared to hirsutism with PCO subgroups (mean= 309.7 versus 184.6, p<0.001) (Table 6).

Table (6): Comparison of omentin-1 level among studied subjects.

		Control N=42	Hirsutism			P^1	P^2
			Total N=42	Idiopathic n=21	With PCO n=21		
Omentin-1 (ng/mL)	**Mean**	271.5	247.1	309.7	184.6	0.087	**<0.001**
	± SD	47.5	60.3	63.9	45.4		

Student t test was used for comparison of numerical parameters; p1, comparison between all studied cases versus control groups; p2, comparison between idiopathic versus PCO associated hirsutism cases.

Receiver operating characteristic curve (ROC) was conducted for discrimination between hirsutism and control groups. Poor AUC was found (AUC=0.608) for discrimination between cases and controls. At best cut off value was 283 pg/mL, sensitivity was 66.7%, specificity was 52.4%, PPV was 58.4%, NPV was 61.1% and accuracy was 59.6% (Table 7).

Table (7): Validity of Omentin-1 level for discrimination between hirsutism cases and control groups

	Omentin-1
AUC	0.608
Cut off (pg/mL)	283
Sensitivity (%)	66.7
Specificity (%)	52.4
PPV (%)	58.4
NPV (%)	61.1
Accuracy (%)	59.6

AUC, area under ROC, ROC, receiver operating characteristic curve; PPV, positive predictive value; NPV, negative predictive value.

ROC was conducted for discrimination between idiopathic hirsutism and those with PCO subgroups. High accuracy AUC was found (AUC=0.912) for discrimination between both subgroups. At best cut off value was 224.5 pg/mL, sensitivity was 95.2%, specificity was 95.2%, PPV was 95.2%, NPV was 95.2% and accuracy was 95.2% (Table 8).

Table (8): Validity of Omentin-1 level for discrimination between idiopathic and PCO associated hirsutism subgroups

	Omentin-1
AUC	0.912
Cut off (pg/mL)	224.5
Sensitivity (%)	95.2
Specificity (%)	95.2
PPV (%)	95.2
NPV (%)	95.2
Accuracy (%)	95.2

AUC, area under ROC, ROC, receiver operating curve; PPV, positive predictive value; NPV, negative predictive value.

Lower omentin-1 level was significantly associated with irregular menses, acne, lower grades of severity (p<0.001, =0.009, 0.001 respectively). Moreover, lower omentin-1 level was non- significantly associated with positive FH, FPHL, AN (p>0.05) (Table 9).

Table (9): Association of omentin-1 level according to studied parameters in hirsutism group.

		Hirsutism N=42			
		Omentin-1 (pg/mL)			**p**
		Mean	**±**	**SD**	
Menses	**Regular**	295.2	±	73.2	**<0.001**
	Irregular	199.1	±	47.4	
Acne	**Absent**	279.5	±	68.32	**0.009**
	Present	211.5	±	50.9	
FH	**Negative**	262.7	±	63.4	0.138
	Positive	221.8	±	52.9	
FPHL	**Absent**	262.6	±	64.4	0.275
	Present	233.1	±	57.3	
AN	**Absent**	255.5	±	62.6	0.539
	Present	238.8	±	57.4	
Grade of hirsutism	**Mild**	302	±	75.1	**0.001**
	Moderate	251.5	±	61.2	
	Severe	163.8	±	35.4	

Student t test was used for comparison of Omentin-1 level according to menses, acne, FH, FPHL, AN. ANOVA test was used for comparison of omentin-1level according to severity.

DISCUSSION

Hirsutism causes a lot of discomfort in women. Additionally to being an aesthetic issue, it can also negatively impact a patient's quality of life and be accompanied by extreme anxiety and sadness [(17)]. The two most frequent causes of hirsutism are PCOS and idiopathic hirsutism (IH) [(18)]. Further hormone testing should be done in women who have hirsutism, menstrual problems, infertility, or other physical examination results that point to endocrine issues [(19)]. The aim of the present study was to evaluate serum level of omentin-1 in hirsute females in comparison with normal non hirsute women and to find a possible relation between omentin-1 level and severity of hirsutism and PCOS. This study included 42 patients diagnosed with hirsutism (21 idiopathic, 21 PCO associated hirsutism) and 42 healthy matched controls. They were chosen randomly from the Laser Unit in Dermatology Department of Mansoura University Hospitals.

In the present study, positive family history (FH) and irregular menses were significantly associated with hirsutism with PCO when compared to idiopathic hirsutism ($p < 0.001$). These findings are in line with those of **Kenawi *et al.*** [(20)] who found substantial differences in the positive FH in hirsute females with PCOS and idiopathic hirsutism compared to the control group. This might be attributed to the genetic susceptibility and comparable ethnic backgrounds of the study's female participants [(21)]. Additionally, they discovered that hirsute females with PCOS experienced higher menstrual cycle irregularity than did controls and women with idiopathic hirsutism (P = 0.0005, 0.00001, respectively).

Though, even after adjusting for BMI, **Song *et al.*** [(22)] discovered that women with hirsutism had more menses each year (10 ± 0 vs 9 ± 2), which was statistically significant (P< 0.001). Amenorrhea or oligomenorrhea were not present in any of the hirsute ladies. 511 (25.4%) of the women who did not have hirsutism experienced oligomenorrhea.

In the instant study, acne, acanthosis nigricans (AN) and female pattern hair loss (FPHL) were significantly associated with hirsutism with PCO when compared to idiopathic hirsutism (p=0.013, 0.031, 0.002 respectively). According to **Kenawi *et al.*** [(20)], females with idiopathic hirsutism had significantly more seborrhea and female pattern hair loss (FPHL) than controls (P = 0.015, 0.0128, respectively), and hirsute females with PCOS had significantly more seborrhea and acne than controls (P = 0.043, 0.0002, respectively). Similar to this, **Khan *et al.*** [(23)] found that 70% of hirsute females had acne.

The current study demonstrated that hirsute females displayed indications of hyperandrogenism more frequently than controls, which is caused by either an increase in blood testosterone levels or a hypersensitivity to it. By directly affecting the hair follicle and pilosebaceous unit (PSU), androgen excess

causes hirsutism, FPHL, and acne [24]. Two pathways seem to be involved in how androgens help villus hair become terminal hair. First, androgens can directly affect the size and colour of the hair follicle by activating androgen receptors in the PSU of the dermal papilla, which is in charge of doing so through regional signaling elements. Second, the enzyme 5-reductase, which changes testosterone into the more powerful dihydrotestosterone (DHT), is present in the hair follicle, along with all the other enzymes required to produce and metabolise androgens. Acting through androgen receptors, testosterone and DHT lengthen the growth phase of the hair cycle and encourage terminal hair development, resulting in longer and thicker hair. Thus, even if circulating hormone levels are not increased, local androgen synthesis might nonetheless lead to hirsutism [25]. Circulating androgen levels, local androgen levels, and hair follicle androgen sensitivity all contribute to hirsutism [26].

In the current study, higher grades of hirsutism were significantly associated with PCO when compared to idiopathic hirsutism ($p<0.001$). This is in line with the findings of **Kiran *et al.*** [27] who discovered that more PCOS patients had severe hirsutism than non-PCOS individuals, albeit this difference was not statistically significant ($p=0.16$). The hormonal imbalance associated with PCOS might make hirsutism more severe. Nevertheless, **Kenawi *et al.*** [20] found no statistically significant difference in the grade of hirsutism between hirsute females with PCOS and hirsute females with idiopathic hirsutism ($P = 0.289$).

The results of this study showed significantly higher TT in hirsutism with PCO subgroup when compared to idiopathic hirsutism subgroup ($p<0.001$). This is in agreement with **Kenawi *et al.*** [20] research, which showed that hirsute girls with PCOS had higher serum testosterone levels than hirsute females with idiopathic hirsutism ($P < 0.001$).

In the present study, hirsutism with PCO subgroup showed significantly higher FBG, insulin, HOMA-IR when compared to idiopathic hirsutism subgroup ($p<0.001$). According to **Bakry *et al.*** [28] there was no significant difference in the level of fasting serum insulin between the PCO and IH groups, although fasting blood sugar (FBS) and HOMA-IR were considerably higher in the PCO group compared to the IH group ($P=0.025$ and 0.037, respectively). Insulin-like growth factor (IGF)-1 activation of theca-cell receptors by hyperinsulinemia increased visceral fat and resulted in ovarian hyperandrogenemia [29]. According to **Mahde *et al.*** [30], PCOS patients exhibited higher levels of testosterone, insulin, and HOMA-IR, which are consistent with the current findings.

In our study, in comparing all studied hirsutism cases to control group revealed non-significant lower omentin-1 level in hirsutism cases when compared to control group ($p=0.08$). While higher omentin-1 level was significantly associated with idiopathic hirsutism when compared to hirsutism with PCO subgroups ($p<0.001$). This is in agreement with **Kenawi *et al.*** [20], who found that the serum omentin-1 level was considerably lower in hirsute women with PCOS than control women ($P = 0.003$) and significantly higher in women with idiopathic hirsutism than hirsute women without PCOS ($P <0.0001$). These differences in findings, even across two studies, may be the consequence of methodological quirks or other modifying elements within the examined groups.

When omentin-1 levels in the blood of women with PCOS were compared to those of matched control participants, **Mahde *et al.*** [30] study and **Tang *et al.*** [31] meta-analysis found that patients with PCOS had lower serum omentin-1 ($p<0.0001$), which is consistent with the present findings. Furthermore, **Mahde *et al.*** [30] discovered that there was a difference in omentin-1 levels between PCOS patients with regular and irregular menstrual cycles that was statistically significant ($p <0.001$). This may be because omentin-1 can affect the secretion of hormones that regulate ovarian and menstrual function or affect receptors for these hormones.

The present study demonstrated that detection of omentin-1 level could be used as reliable indicator in discrimination between idiopathic hirsutism and those with PCO subgroups (AUC=0.912) with high sensitivity (95.2%) and specificity (95.2%).

In the present study, lower omentin-1 level was significantly associated with irregular menses, acne and higher grades of hirsutism severity in hirsutism group ($p<0.001$, 0.009, 0.001 respectively). Omentin-1 level showed significant negative correlations with degree of hirsutism, TT, FBG, insulin levels and HOMA-IR ($r<0$, $p<0.05$ for each). In addition, omentin-1 level showed marginally significant negative correlations with BMI. According to **Kenawi *et al.*** [20], there was a statistically significant negative association between serum testosterone and omentin-1 in the hirsutism group but a non-significant inverse correlation between omentin-1 and BMI. Additionally, this was in line with the findings of **Mahde *et al.*** [30], who found a substantial negative relationship between the level of omentin-1 and fasting insulin, HOMA, and BMI. Omentin-1 levels in PCOS patients may be linked to elevated HOMA-IR ratios, according to **Tang *et al.*** [31]. Omentin-1 may have the ability to increase insulin sensitivity via activating the insulin signaling pathway by phosphorylating Akt [32].

The evaluation of the homeostasis model revealed an inverse relationship between plasma omentin-1 levels and obesity and insulin resistance. It offers potential benefits in the treatment of several endocrine illnesses, including type 2 diabetes and polycystic ovarian syndrome, and it is related to patients' levels of insulin resistance [33]. Given that it has been observed that obese insulin-resistant people had lower plasma omentin-1 levels, it is interesting that

women with PCOS, an insulin resistance and pro-diabetic condition, have lower amounts in their plasma and adipose tissue [34]. In conclusion, omentin-1 could have a protective effect against higher degree of hirsutism, acne, obesity, insulin resistance, hormonal abnormalities in hirsute females.

CONCLUSION

Omentin-1 level could be used as reliable indicator in discrimination between idiopathic hirsutism and those with PCO.

This study revealed significant lower omentin-1 level in hirsutism cases when compared to control group. Higher omentin-1 level was significantly associated with idiopathic hirsutism when compared to hirsutism with PCO subgroups.

Lower omentin-1 level was significantly associated with irregular menses, acne, higher grades of hirsutism severity. Moreover, lower omentin-1 level was non-significantly associated with positive FH, FPHL, AN.

Omentin-1 levels significantly correlated negatively with HOMA-IR, TT, FBG insulin levels, and degree of hirsutism. Additionally, it revealed weakly significant negative associations with DHEA-S and BMI. No significant correlations were found between omentin-1 level versus age and 17OH progesterone level. In conclusion, omentin-1 may protect hirsute ladies against more severe hirsutism, acne, obesity, insulin resistance, and hormonal irregularities.

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