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Serum Asprosin Levels in Women with Polycystic Ovary Syndrome in Duhok City, Kurdistan Region of Iraq

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Abstract

Asprosin is a novel peptide hormone produced and secreted by white adipose tissues. Asprosin associated with insulin resistance and promotes hepatic glucose production. Previous studies showed that serum asprosin was raised in the general population with polycystic ovary syndrome (PCOS). However, there were studies supporting the opposite. Also, there were studies that showed the highest levels of asprosin was due to insulin resistance, as well as in type 2 diabetes patients. PCOS is one of the metabolic disorders related to insulin resistance. Therefore, the current study aims to evaluate the levels of asprosin in the blood serum of women with PCOS compared to the healthy women who resident in Duhok in the Kurdistan Region of Iraq. A cross-sectional study was conducted from 20th of June, 2020 to 11th of January, 2021 at Obstetrics and Gynecology Hospital and Mazi medical clinics. Serum asprosin level was determined in 75 women with PCOS (18-44 years) and 96 healthy women. SPSS software was utilized for analyzing the study data. The (means ± SD) of demographic parameters (body mass index (BMI) and waist circumference (WC)) in women with PCOS were significantly highest in comparison to healthy women. The biochemical parameters (serum asprosin, fasting blood sugar (FBS), fasting insulin (FI), total cholesterol (TC), and triglyceride (TG)) in women with PCOS also were remarkably higher compared to healthy women with the exception of high-density lipoprotein- cholesterol (HDL-C). The current data show that serum asprosin variance significantly between WC, BMI, FBS, FI, TC, TG and HDL-C. The study confirms that serum asprosin in women with PCOS was higher than in the healthy women. In addition in women with PCOS it was found that serum asprosin was positively correlated with BMI, WC, FBS, FI, HOMA-IR, TC and TG (P<0.05). Except, HDL-C was negatively correlated with serum asprosin (P<0.01).

Keywords: *Asprosin, PCOS, Insulin resistance, Metabolic disorders, Duhok.*

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I. INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine and metabolic disease in women with an incidence of up to 17.8%, with hyperandrogenism, abnormal menstrual periods and polycystic ovaries (Witchel *et al.*, 2019).

In women with polycystic ovary syndrome, obesity and an aberrant metabolic profile are prevalent and about 50-70 % of them are insulin resistant (Dumesic *et al.*, 2015). While the majority of women with PCOS compensate for their insulin resistance (IR), a significant proportion of them have impaired beta-cell function, resulting in glucose sensitivity, which increases their risk of rising type two diabetes mellitus, regardless of their body mass index (BMI) or age (Alan *et al.*, 2019).

Additionally, women with PCOS are at an increased risk of rising dyslipidemia and hypertension, which are associated with an increased prevalence of metabolic syndrome (Amiri *et al.*, 2020; Özkan *et al.*, 2020).

The PCOS etiology remains unknown; although it is believed to be multifactorial. Hyperinsulinemia and hyperandrogenism in PCOS are strongly linked, but the processes underlying their relationship with polycystic ovary syndrome are not well understood (Rosenfield and Ehrmann, 2016; Escobar-Morreale, 2018).

Asprosin is a novel identified peptide hormone produced and secreted by white adipose tissue. Asprosin enhances the release of glucose from the

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liver by stimulating the guanine nucleotide-binding protein-Adenosine 3',5'-cyclic monophosphate - protein kinase A (G protein-cAMP-PKA) pathway (Romere *et al.,* 2016; Duerrschmidi *et al.*, 2017).

Recently, researchers have conducted several studies on asprosin and they were observed that asprosin levels were rises relative to controls in subjects with type two diabetes. Also, asprosin levels were correlated directly with insulin resistance (Wang *et al.*, 2018; Zhang *et al*., 2019).

Obesity, PCOS, diabetes, and cardiovascular disease (CVD) are all metabolic diseases that are caused by a breakdown to natural metabolic processes; pose a significant risk to human health. Thus, the discovery of asprosin was a key field of research for the treatment of multiple metabolic diseases associated with insulin resistance. Recent research has established that asprosin has a critical and complex function in metabolism and metabolic diseases (Yuan *et al.,* 2020).

To conduct more studies on asprosin and its effect on metabolic disorders, which include insulin resistance and PCOS, this study was conducted to evaluate serum asprosin levels in women with PCOS in the Kurdistan Region of Iraq. So this study considered as one of the new approaches for studying women with PCOS in our country.

II. MATERIALS AND METHODS

A. Subjects and study design

To achieve the aims of the study, a cross-sectional method was used. This study was performed at Obstetrics and Gynecology Hospital, Mazi Private Laboratory, and Mazi medical clinics in Duhok, Kurdistan Region-Iraq. The study was established from June 20th, 2020 to 11th January 2021. A total of 171 women (with ages ranging from (18-44) years) were selected throughout the period of performing this study. The study recruited 75 women from the study population who fulfilled the PCOS criteria, and 96 women as healthy women.

B. Data collection and research tool

A questionnaire was designed to elicit the necessary information from participants. After informing each participant about the study and obtaining their consent, the standardised questionnaire was administered via direct interview. Personal information (phone number, age, place of employment, and address), pregnancy history, menstrual history, birth control history, height, weight, BMI, waist circumference, prior gynaecological surgery, chronic diseases related to exclusion criteria, oral hypoglycemic drug history, and insulin therapeutic drug history were included in the questionnaire type.

C. Inclusion criteria

1. Polycystic ovary syndrome group

PCOS patients were diagnosed according to Rotterdam criteria (The Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group, 2004), after excluding other causes of hyperandrogenism and menstrual cycle irregularities. In this study a gynecologist uses Rotterdam criteria in the selection PCOS patients, which states the presence 2 of 3 the following criteria; Firstly, oligo and/or anovulation. Secondly, symptoms of hyperandrogenism (biochemical: total testosterone > 70ng/100ml, androstenedione > 245ng/100ml, dhea-s > 248ng/100ml, clinical signs: acne, hirsutism, acanthosis nigrans). Finally, polycystic ovaries on ultrasonography (ovarian volume 10 cm3 and / or >12 follicles in the antral region in one ovary). In addition, ultrasonography was done by the radiologist to exclude the other causes of hyperandrogenism and menstrual abnormality.

2. Healthy women group

The participants in this category were selected among the women who have regular menstrual cycles (25–35 days). Also, there were no endocrine anomalies, no physiological or biochemical signs of androgen excess and normal ultrasonic ovarian morphology.

D. Exclusion criteria

The exclusion criteria were attended to subject with the following conditions; Hypothyroidism, renal / liver disease, tumor of androgen-secreting, cushing's syndrome, congenital adrenal hyperplasia, hyperprolactinemia, congestive heart diseases, insulin resistance circumstances like acromegaly, use of medications for decreasing hypertension, dyslipidemia, hyperglycemia, insulin resistance, or obesity also were excluded, as well as the non-fasting patient was excluded in the present study.

E. Anthropometric assessment

Anthropometric measurements were assessed that include; age, height (cm), weight (kg), and waist circumference (centimeter). Additionally, all subjects' blood pressures were measured after 15-minutes of rest time in the seated position. The BMI value was calculated using the following formula: BMI = weight (kg)/square metre of height (m^2) (Deore *et al*., 2012).

F. Blood samples collection

Participants were given directives to attend the Mazi privet laboratory in the morning after overnight fasting for 10 hours. Around 9:30 and 11:30 a.m., samples of blood were obtained. Approximately 10 ml of blood was withdrawn and collected by venipuncture in the BD vacutainer device (in two serum separator tubes). The first tube was allowed for 15 minutes after blood collection, then serum was separated for 8 minutes using a centrifuge at 1000 x g, the obtained serum samples were prepared promptly so as to measure lipid profile, FBS, and fasting insulin following standard laboratory procedures using clinical chemistry analyzer Cobas 6000 Roche. The blood from the second tube was held at room temperature for two hours. Then serum was separated for 20 minutes using a centrifuge at 1000 x g. The obtained serum samples from the second tube were stored in deep freeze (-25) for later analysis of serum asprosin using the ELISA technique

G. Biochemical assessment

Serum samples were biochemically analyzed using the Autoanalyzer biochemical machine named COBASS series 6000 and ELISA technique in order to obtain sensitive and precise findings. A glucose HK kit was used to determine the level of serum (Reference number: 04404483 190 Roche/Germany). According to the manufacturer protocol instructions, in this case, cholesterol gen 2 (Reference number: 03039773 190, Roche, Germany) was used to detect the serum total cholesterol. Also, A triglyceride kit (Reference number: 20767107 322, Roche, Germany) was used to identify the serum triglyceride. Moreover, An HDL-Cholesterol gen.4 kit (Roche) was used to measure serum high-density lipoprotein-cholesterol. Reference number: 07528566 190. Furthermore, the insulin kit is used to monitor and evaluate fasting insulin levels (Reference number: 12017547 122) also, each test participant was assessed for insulin resistance using the (HOMA-IR) = fasting insulin (μU/ml) * fasting glucose (mg/dl)/405 (Matthews *et al.*, 1985).

H. Serum asprosin assessment

Human Asprosin ELISA kit with the catalog No.:MBS7606420, sensitivity: 0.938ng/ml (from MyBioSource, Inc. San Diego, USA) was used for quantitative detection of Asprosin in serum in accordance with the instructions of manufactures.

I. Statistical analysis

The data were analysed and represented as (mean ± standard division) using the SPSS programme version 25. The T-test was performed to compare proportions. In addition, to compare various groups, one-way variance analysis (ANOVA) was utilised. When comparing categorical variables between groups, the Chi-square test was used. The Pearson correlation coefficient was employed to estimate the relationship between variables. ROC curve analysis was used to determine the serum asprosin cut-off point for PCOS prediction. In all experiments, a $p<0.05$ was statistically significant.

J. Ethical considerations

The Scientific Committee of Duhok Polytechnic University / Shekhan Technical College and the Ethical Committee of the General Directorate of Health in Duhok have reviewed and approved the study protocols (Code of Ethics:22062020-2).

III. RESULTS

A. Demographic and biochemical characteristic features of study population according to PCOS and healthy women

Demographic characteristics and Biochemical parameters of the population of the study are elucidated in Table 1. Women with PCOS presented significantly higher mean values of BMI, waist circumference (WC), serum asprosin, total cholesterol, serum levels of TG, FBS, fasting insulin, and HOMA-IR in comparison with healthy women ($P < 0.05$). Except for HDL-C which was lower in women with PCOS in comparison to healthy women ($P<0.05$). On the contrary, there was no significant variation in DBP, SBP and age across the groups ($P > 0.05$).

Table 1. The laboratory and demographic characteristics comparison in the study population

Variables	Mean ± SD		*P- value*
	PCOS (n=75)	healthy women(n=96)	
Age (years)	27.05 ± 7.50	25.88 ± 7.87	NS
BMI (kg/m2)	29.94 ± 6.82	24.84 ± 5.62	< 0.001*
WC(cm)	84.55 ± 18.01	78.97 ± 11.49	< 0.05*
SBP. (mmHg)	113.35 ± 14.82	114.81 ± 9.71	NS
DBP. (mmHg)	78.43 ± 11.21	76.40± 5.58	NS
TC (mg/dl)	154.73 ± 34.40	139.72 ± 25.21	< 0.05*
TG (mg/dl)	122.47 ± 65.39	84.57 ± 29.78	< 0.01*
HDL- C (mg/dl)	41.91 ± 9.96	56.25 ± 8.01	< 0.05*
FBS (mg/dl)	104.61 ± 22.52	97.91 ± 12.64	< 0.05*
Insulin (μU/mL)	19.26 ± 9.33	11.26 ± 4.75	< 0.001*
HOMA-IR	5.12 ± 3.28	2.75 ± 1.34	< 0.001*
Serum asprosin (ng/ml)	14.80±6.67	5.49±3.42	< 0.001*

t-test was used. A p value of <0.05 was considered significant (*). NS: non-significant a p value was >0.05.Key:BMI = Bodys MasssIndex, WC = WaistsCircumference,SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, TC= Total cholesterol, TG= Triglyceride ,HDL-C = High Density Lipoprotein -Cholesterol, FBS = Fasting Blood Sugar**,** and HOMA-IR= Homeostatic Model Assessment for Insulin Resistance.

B. Association of asprosin concentration with clinical parameters

Accordance to the Pearson correlation coefficient (r), there was a remarkable positive relation ($P <0.001$) between serum asprosin and age, BMI, Waist circumference, TC, TG, FBS, insulin, and HOMA-IR ($P <0.05$) in both groups. Also, there was a significant direct correlation ($P<0.001$) between serum asprosin and age, SBP only in the healthy women group ($P <0.001$). On contrary, serum asprosin was negatively correlated with HDL-C in both the groups ($P <0.05$) (Table 2).

Table 2. The correlations between serum asprosin level and possible metabolic risk factors in the study population.

Variables	PCOS (n=75)		healthy women(n=96)	
	r	*p-value*	*r*	*p-value*
Age (years)	.164	0.161	.590**	<0.001
BMI (kg/m2)	.322**	0.005	.671**	<0.001
WC(cm)	.369**	0.001	.611**	<0.001
SBP (mmHg)	.149	0.203	.243*	0.017
DBP (mmHg)	.079	0.499	.166	0.105
TC (mg/dl)	.319**	0.005	.402**	<0.001
TG (mg/dl)	.310**	0.007	.596**	<0.001
HDL- C (mg/dl)	-.250-*	0.031	-.376-**	<0.001
FBS (mg/dl)	.311**	0.007	.699**	<0.001
Insulin (μU/mL)	.362**	0.001	.249*	0.014
HOMA-IR	.405**	<0.001	.427**	<0.001

Correlations between variables were analyzed by Pearson analysis. (**) Correlation is remarkable at the 0.01 level (2-tailed). (*) Correlation is remarkable at the 0.05 level (2-tailed). Key:BMI = Bodys MasssIndex, WC = WaistsCircumference,SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, TC= Total cholesterol, TG= Triglyceride ,HDL-C = High Density Lipoprotein -Cholesterol, FBS = Fasting Blood Sugar**,** and HOMA-IR= Homeostatic Model Assessment for Insulin Resistance.

C. The concentration of serum asprosin and PCOS

Based on concentrations of serum asprosin, all subjects were divided into tertiles (T1: < 4 ng/mL, T2: 4–13.5 ng/ mL and T3: > 13.5 ng/mL). The Demographic characteristics and Biochemical parameters of each category are listed in Table 3. In correspondence to tertiles the parameters like age, waist circumference, BMI, FBG, fasting insulin, T. Cholesterol, TG, and HOMA-IR were increased. Moreover, parameter such as HDL-C decreased in correspondence to tertiles. The number of PCOS patients increased as asprosin concentrations in the tertiles increased, according to the trending of the Chi-square test (Figure 1). Besides that, the major patients with PCOS placed in the third tertile. The receiver operating characteristic (ROC) analysis showed that the cut-off value for serum asprosin to predict PCOS was (7.476 ng/ml) (p >0.001). It had an area under the curve (AUC) (0.909), with sensitivity (0.906), and specificity (0.750) (Figure 2).

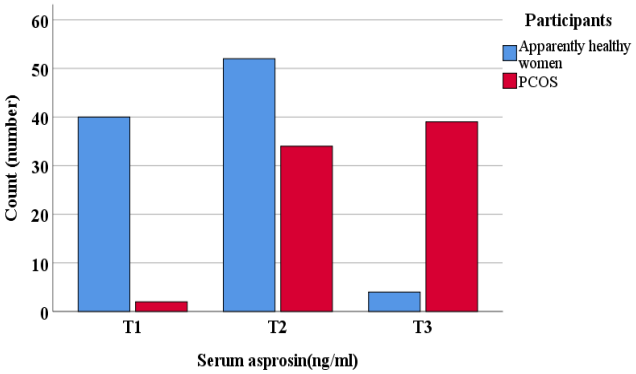

Figure 1.Number of individuals present in each tertile. Most of healthy people were situated in T1 and T2s. However, the number of PCOS women was higher in T2 and T3 in comparison to T1. The most interesting result is that the major number of participants who had PCOS on the T3.

Table 3. Distribution of clinical parameters of participants in different tertiles based on serum concentrations of asprosin in all subjects

Variables	(T1) N=42	(T2) N=86	(T3) N=43	*p-value*
Serum asprosin (ng/ml)	2.385 ± 0.769	8.275 ± 2.666	19.194 ± 5.189	<0.001
Age (years)	21.905 ± 4.023	27.233 ± 8.141	29.093 ± 7.855	<0.001
BMI (kg/m2)	22.264 ± 3.522	26.602 ± 5.599	32.714 ± 6.912	<0.001
WC(cm)	74.679 ± 8.677	77.994 ± 12.365	94.837 ± 16.274	<0.001
SBP (mmHg)	111.952 ± 10.007	114.419 ± 11.708	115.837± 14.808	0.330
DBP (mmHg)	75.643 ± 6.362	76.500 ± 7.295	80.465 ± 11.583	0.015
TC (mg/dl)	131.452 ± 22.541	142.581 ± 25.344	168.256 ± 34.767	<0.001
TG (mg/dl)	67.952 ± 21.123	94.791± 37.106	146.465± 66.854	<0.001
HDL- C (mg/dl)	57.976 ± 6.848	51.593 ± 10.753	38.860 ± 7.220	<0.001
FBS (mg/dl)	91.643± 5.136	100.000 ±13.358	111.535 ± 26.647	<0.001
Insulin (µU/mL)	9.844± 3.002	14.194 ± 7.256	20.497 ± 9.818	<0.001
HOMA-IR	2.231 ± 0.703	3.546 ± 1.954	5.794 ± 3.706	<0.001

One way ANOVA test was used. Data are presented as mean ± S.D. Key**:** BMI = Bodys MasssIndex, WC = WaistsCircumference,SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, TC= Total cholesterol, TG= Triglyceride ,HDL-C = High Density Lipoprotein -Cholesterol, FBS = Fasting Blood Sugar**,** and HOMA-IR= Homeostatic Model Assessment for Insulin Resistance.

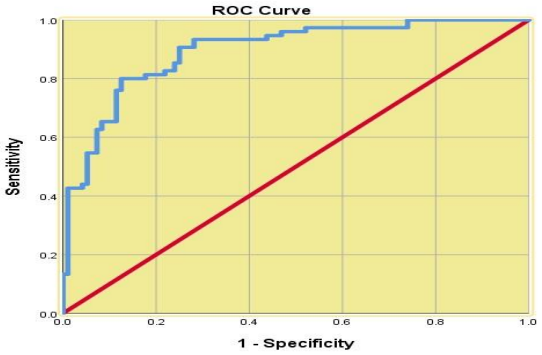

Figure 2. PCOS prediction by utilizing ROC curve analyses on serum asprosin.

IV. Discussions

Adipose tissue secretes a large number of adipokines that regulate glucose, lipid metabolism and insulin resistance (Coelho *et al*., 2013). Numerous adipokines such as leptin, ghrelin, asprosin, and irisin have been documented to have modified and influenced insulin secretion and insulin effect in women with PCOS (Polak *et al.*, 2017). Asprosin is a newly known adipokine (Romere *et al*., 2016). Four independent study groups investigated whether asprosin plays a role in women with PCOS, but their findings were conflicting (Li *et a*l., 2018; Alan *et al.*, 2019; Chang *et al*., 2019; Deniz *et al*., 2020). Although Li *et al.*, Alan *et al*., and Deniz *et al*. reported that women with PCOS had higher circulating asprosin levels than controls (Li *et al*., 2018; Alan *et a*l., 2019; Deniz *et al.*, 2020), Chang *et al*. reported that it was not correlated with the aetiology of PCOS (Chang *et al*., 2019). Despite this interest, no one has researched the evaluation levels of asprosin in patients with PCOS in the Kurdistan region of Iraq to our knowledge. Therefore, this study was designed to clarify conflicting asprosin results in PCOS. In the current study, our hypothesis was the evaluation of serum asprosin in women with PCOS and if the asprosin can use as a biomarker for diagnosis of PCOS. In this study, we found out that the

asprosin levels of women with PCOS were significantly higher than in healthy women (P-value <0.001).

Obesity has been associated with abnormal hypothalamic-pituitary-ovarian (HPO) axis function through a variety of mechanisms that lead to the development of PCOS (Legro, 2012). Although many people with PCOS are overweight, obese, or centrally obese the impact of obesity on PCOS outcomes is ambiguous (Lim *et al.*, 2013). In the current study, the serum asprosin was positively correlated with BMI and waist circumference in women PCOS and healthy women. As well as, results proved the presence of a significant variation in the mean of serum asprosin according to the BMI and waist circumference in the women with PCOS. Similarly, results show that the presence of a significant difference in the mean of serum asprosin according to the BMI and waist circumference in healthy women.

All the above results illustrated the risk of elevation of serum asprosin in the study population elevated by increasing the BMI and waist circumference. An increase in BMI and waist circumference indicated there were fatty tissues excessive in the body, especially in the abdominal area. Consequently, Romere *et al*. (2019), claimed that asprosin secretion elevates since the accumulation of these fats in the abdominal area. The results of our study supported by many researchers, they found a positive correlation between serum asprosin and BMI in women with PCOS (Li *et al.,* 2018; Alan *et al.,* 2019; Ugur and Aydin, 2019; Deniz *et al.,* 2020). In contrast with these findings, a cross-sectional study by Jiang *et al.* (2019) revealed that in PCOS women, serum asprosin was negatively correlated with body mass index.

PCOS is a metabolic disorder that often manifests itself in insulin resistance. Up to 70% of women with PCOS have insulin resistance. Insulin resistance is a significant factor for the development of PCOS (Rojas *et al.,* 2014; Alan *et al.,* 2019). The current study found a significant difference in fasting insulin, FBS, and HOMA-IR between women with PCOS and healthy women. There was a significant positive association between serum asprosin and FI, FBS, and HOMA-IR in the current study's PCOS women. Similarly, serum asprosin had a significant positive association with FBS, FI, and HOMA-IR in healthy women. Insulin resistance in patients with PCOS may have been caused by a high level of asprosin, which mediates the release of glucose from the liver into circulation (Romere *et al.,* 2016), resulting in an accumulation of glucose in circulation and an excessive secretion of insulin to get the increased circulating glucose to physiologic levels (Rojas *et al.,* 2014). As a result, in current research, increasing the circulating asprosin levels of patients with PCOS could be in order to neutralise the hyperinsulinemia observed in these patients. The fact that patients with PCOS had elevated HOMA-IR values supported this. Another possible explanation for elevated circulating asprosin levels in PCOS patients is the production of asprosin resistance. In other words, high levels of asprosin in patients with PCOS were either not used by the cells or were not metabolised properly as a result of overproduction, and thus could not be removed. Hormones are metabolised either by the liver or the kidneys or by both. However, there is currently no empirical evidence indicating where asprosin is metabolised. Thus, the available evidence suggests that insulin resistance is a significant contributor to hormonal and biochemical defects in PCOS disease.

Additionally, the elevated glucose levels observed in this study may be a result of aprosin's role in the release of glucose from hepatic tissues into circulation, as asprosin is a molecule that facilitates the transport of glucose from the liver to circulation (Romere *et al.*, 2016). The current study's results corroborated those of Li *et al.* (2018), Alan *et al*. (2019), and Deniz *et al*. (2020), but contradicted Jiang *et al.* (2019), who demonstrated that asprosin was negatively associated with IR and HOMA-IR in women with PCOS and women with IR.

According to the results of this study, there was a significant difference in TC, TG, and HDL-C levels among women with PCOS and healthy women. Whereas, the results of this study are in agreement with the results of recent studies conducted by Pergialiotis *et al*. (2018), Tsouma *et al.* (2014), Ghaffarzad *et al.* (2016). In which they claimed that women with PCOS have lipid defects. Also, PCOS is associated with a variety of lipid forms, including low levels of HDL-C, elevated TG levels and TC levels.

In the present study, there was a strong positive association between serum asprosin and TC and TG levels in women with PCOS. However, the results demonstrated the existence of a significant negative association between HDL-C and serum asprosin in women in both study groups.

In this study, it was found that there was a significant relationship between dyslipidemia and increased asprosin in patients with PCOS. The effect on metabolic parameters was probably due to the effects of asprosin on appetite because it was reported in previous studies that increased asprosin in T2DM patients affected appetite and led to metabolic changes (Li *et al.,* 2018: Zhang *et al.,* 2019). The current study's results corroborated those of Li *et al.* (2018), Alan *et al*. (2019), and Deniz *et al*. (2020), but contradicted that with Jiang *et al.* (2019), who found that asprosin had a negative correlation with TG and a positive correlation with HDL-C in women with PCOS.

The crude AUC of the asprosin ROC curve for detecting PCOS was 0.909 in the current investigation, and it was regarded to be of good significance, which could be related to the small sample size and non-normal distribution of the examined population. As a result, it may not be an ideal predictor of PCOS diagnosis. So, a large-scale study is recommended to confirm the serum asprosin cut-off value for PCOS diagnosis.

Our study had several limitations; firstly, a cross-sectional design was performed in this study and it was done in a

single city, so that can be considered as a limitation. Secondly, our sample size was small and could be considered as insufficient for making a general conclusion. Finally, the present study contained only Iraqi-Kurdish women.

V. CONCLUSIONS

This study confirms that serum asprosin in women with PCOS was higher than in healthy women. Also, serum asprosin was significantly positively correlated with BMI, waist circumferences, fasting insulin, fasting blood sugar, HOMA-IR, total cholesterol and triglyceride in healthy women and women with PCOS. On contrary, high-density lipoprotein–cholesterol was negatively correlated with serum asprosin in both groups of the study population.

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