



Diagnostic Value of Serum Asprosin, Chemerin, and Adropin in Women with Polycystic Ovary Syndrome

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Abstract

Background: Polycystic ovary syndrome is a common endocrine disorder characterized by metabolic dysfunction, making serum asprosin, chemerin, and adropin promising biomarkers for diagnosis. **Aims of the study:** This study aimed to evaluate the diagnostic value of serum asprosin, chemerin, and adropin in women with polycystic ovary syndrome and their association with disease severity. **Methodology:** A case-control study was conducted at Gynecology Outpatient and Consultant Gynecology Clinics, Iraq, between October 12, 2025, and June 5, 2026, including 80 women with polycystic ovary syndrome and 40 age-matched healthy controls. Diagnosis followed the Rotterdam criteria. Fasting serum asprosin, chemerin, adropin, reproductive hormones, insulin, glucose, and HOMA-IR were measured using ELISA, chemiluminescence immunoassays, and enzymatic methods. PCOS severity was classified as mild, moderate, or severe according to clinical, hormonal, ultrasonographic, and metabolic findings. **Result:** Women with PCOS exhibited significantly higher BMI, fasting glucose, reproductive abnormalities, serum asprosin, chemerin, testosterone, LH, insulin, LH/FSH ratio, and HOMA-IR, while adropin levels were significantly reduced compared with controls. Biomarker alterations became progressively more pronounced with increasing disease severity. Asprosin and chemerin correlated positively, whereas adropin correlated negatively, with metabolic and hormonal parameters, particularly HOMA-IR. ROC analysis demonstrated excellent diagnostic performance, with adropin showing the highest accuracy, sensitivity, and specificity for distinguishing PCOS from healthy women. **Conclusions;** Serum asprosin, chemerin, and adropin are promising biomarkers for polycystic ovary syndrome. Their significant associations with insulin resistance, hormonal imbalance, and disease severity support their potential utility in improving diagnosis, metabolic assessment, and clinical monitoring of women with PCOS.

Keyword: Polycystic ovary syndrome; Asprosin; Chemerin; Adropin; Insulin resistance.

Introduction:

Polycystic ovary syndrome (PCOS) is one of the most common endocrine and metabolic disorders in women of reproductive age and its prevalence has been estimated to be 8-20% depending on the definition and study population. Patients with the syndrome present with a variable clinical picture with features of

ovulatory dysfunction, hyperandrogenism and polycystic ovarian morphology [1]. In addition to its reproductive effects, PCOS is now known to be a multisystem disease that coexists with obesity, insulin resistance, dyslipidemia, chronic low-grade inflammation, and type 2 diabetes mellitus and cardiovascular disease. Due to its variable clinical presentations and multifactorial etiology, diagnosis is difficult, especially for the woman with mild or atypical symptoms. As such, much research has focused on the search for new markers in the circulation that might prove to be more sensitive and specific for diagnosis and also shed light on the metabolic disturbances involved in the disorder [2,3].

PCOS pathophysiology is a complex interaction of many factors such as genetic susceptibility, environmental factors, hormonal abnormalities and metabolic abnormalities. Hyperinsulinism due to insulin resistance is a key mechanism, acting as a stimulant for the theca cells to produce excess androgens, and a suppressant for the production of sex hormone-binding globulin (SHBG) by the liver, which causes rises in free androgens in the blood [4]. This hormonal imbalance causes abnormal follicular development and causes chronic anovulation and infertility. At the same time, excess energy stored in adipose tissue leads to obesity, which in turn causes insulin resistance and a chronic low-grade inflammation by overproduction of adipokines and inflammatory mediators from dysfunctional adipose tissue. The metabolic and inflammatory changes are not only associated with disease progression, but also with an enhanced long-term risk of cardiovascular events and metabolic syndrome in diseased women [5].

In recent years, the endocrine functions of adipose tissue have emerged as one of the most active endocrine organs of the body, producing a variety of bioactive peptides, termed adipokines, that affect glucose metabolism, lipid homeostasis, inflammation, vascular function and reproductive physiology [6]. Immune changes in the production of adipokines have been emerging as key factors in the onset of insulin resistance and ovarian dysfunction in women with PCOS. Thus, some adipokines have been explored as clinical and/or predictive markers that could capture metabolic and reproductive dysfunctions. Asprosin, chemerin and adropin are among the emerging biomarkers that have gained increasing scientific attention due to their strong link with energy metabolism, insulin sensitivity and inflammatory regulation [7,8].

Asprosin is a fasting-induced protein hormone that is mainly secreted by white adipose tissues. It promotes the rapid release of glucose from liver and helps in the maintenance of blood glucose levels during fasting by activating the cyclic adenosine monophosphate signaling pathway. In physiological conditions asprosin is involved in energy homeostasis but when levels of circulating asprosin are increased they have consistently been linked to obesity, insulin resistance, metabolic syndrome and type 2 diabetes mellitus. Insulin resistance is one of the main mechanisms involved in the pathogenesis of PCOS, so the higher concentrations of serum asprosin may be a sign of the metabolic dysfunction of PCOS [9]. Moreover, from experimental studies, it has been suggested that excessive asprosin could be responsible for increased oxidative stress and inflammatory activation, potentially exacerbating the symptoms of ovarian dysfunction and endocrine abnormalities [10].

Additionally, Chemerin is a multifunctional adipokine, which is mainly synthesized by adipose tissue, liver and various reproductive organs including the ovary. It has an important role in adipocyte differentiation, recruitment of cells, regulation of inflammatory responses and angiogenesis [11]. The increasing evidence shows direct role of chemerin in the ovarian follicular maturation and steroidogenesis through interaction with granulosa and theca cells. Therefore, it has been proposed that increased chemerin serum levels could provide a potential mechanism between chronic inflammation, altered glucose metabolism and reproductive dysfunction in women with obesity, insulin resistance, metabolic syndrome and PCOS. Furthermore, there was a significant correlation between raised ovarian chemerin expression with the abnormal follicular development and abnormal ovulation, which is the phenomenon of PCOS pathogenesis [12].

Adropin is a relatively new discovered peptide hormone that is coded for by the Energy Homeostasis Associated (ENHO) gene and is present in the liver, brain, vascular endothelium and other peripheral tissues [13]. While asprosin and chemerin have mainly detrimental metabolic effects, adropin has mainly

beneficial metabolic actions in insulin sensitivity, glucose oxidation, endothelial function and lipid metabolism. Low levels of adiponin are linked to obesity, type 2 diabetes mellitus, cardiovascular disease and metabolic syndrome. Preliminary data indicate that women with PCOS have significantly decreased levels of serum adiponin, which may reflect a dysfunction in metabolic adaptation as well as endothelial function, suggesting chronic insulin resistance. Thus, lower adiponin may be a good marker for the progression of PCOS metabolic abnormalities and disease severity [14].

There are several studies that have studied single adipokines in PCOS, but few studies have investigated the serum levels of adiponin, chemerin, and adiponin in the same population of patients with PCOS [15]. Moreover, the published results are not consistent due to ethnic differences, obesity status, sample size, laboratory procedures, and diagnostic criteria. Combining all these is likely to enhance the diagnostic value of the biomarker, shed light on the metabolic pathways involved in PCOS, and provide novel tools for the diagnosis and management of the condition [16]. Hence, the current study was designed to assess the diagnostic potential of adiponin, chemerin and adiponin in women with PCOS and explore their associations with hormonal changes, insulin resistance and PCOS severity. Reliable associations of these novel markers with clinical symptoms of PCOS may facilitate their future use in addition to other diagnostic tools and be useful in the general understanding of the endocrine and metabolic disturbances that are associated with this prevalent condition.

Methodology:

The study was a case–control study that was conducted in Gynecology Outpatient Clinics and Consultant Gynecology Clinics, Iraq, from October 12, 2025, to June 5, 2026. A total of 120 women aged 18–40 years were enrolled, consisting of 80 women with polycystic ovary syndrome (PCOS) and 40 women who were apparently healthy and were age matched. The diagnosis of PCOS was made if at least two out of three of the following criteria were fulfilled: oligo- or anovulation, clinical and/or biochemical hyperandrogenism and polycystic ovarian morphology as diagnosed by transvaginal and/or transabdominal ultrasonography. The healthy controls exhibited normal menstrual cycles, with no clinical or biochemical evidence of hyperandrogenism or endocrine disease, and had normal ovarian morphology. Women were excluded if they were pregnant or nursing, had a history of diabetes mellitus, thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, Cushing's syndrome, androgen secreting tumor, chronic liver or kidney disease, autoimmune disorders, active inflammatory diseases, malignancy or were on hormonal therapy, insulin sensitizers, corticosteroid, anti-obesity medications, or lipid lowering agents in the last 3 months. Demographic and clinical data such as age, body mass index (BMI), marital status, residence, menstrual history, family history of PCOS, infertility, hirsutism, and acne were collected by using a standardized questionnaire after obtaining written informed consent. All participants were subjected to venous blood samples (5 mL) that were taken following an overnight fast of 10–12 hours in early follicular phase of the menstrual cycle (days 2–5) or at an arbitrary time in women with amenorrhea. Aliquots of serum were stored at -80°C until biochemical analysis, in which blood was centrifuged for 10 minutes at 3000 rpm. Adiponin, chemerin and adiponin were measured by sandwich enzyme-linked immunosorbent assay (ELISA) using commercially available kits following the manufacturers' instructions. Serum luteinizing hormone (LH), follicle-stimulating hormone (FSH), total testosterone and fasting insulin were measured by automated chemiluminescence immunoassay systems and fasting blood glucose was measured by the glucose oxidase enzymatic method using an automated clinical chemistry analyzer. The Homeostasis Model Assessment for Insulin Resistance (HOMA-IR), determined by multiplying fasting insulin ($\mu\text{IU/mL}$) with fasting glucose (mg/dL) by 405, was used to estimate insulin resistance. Under supervision of consultant gynecologists, the severity of PCOS was determined by the total evaluation of clinical features, biochemical hyperandrogenism, ovarian ultrasonographic features, and metabolic abnormalities such as insulin resistance and classified as mild, moderate and severe.

Statistical analysis:

IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA) was used for the statistical analysis. Mean plus or minus the standard deviation (SD) was used to present continuous variables and frequencies and percentages were used for the categorical variables. The independent-samples Student's t-test was used to compare two groups and the one-way analysis of variance (ANOVA) was used to compare multiple severity groups, with Tukey's post hoc test used for comparisons between groups. Pearson's correlation coefficient was used to determine associations between serum biomarkers and clinical and biochemical parameters. Receiver operating characteristic (ROC) curve analysis was adopted to estimate the diagnostic performance of serum asprosin, chemerin and adropin and calculate the area under the curve (AUC), optimal cutoff values, sensitivity and specificity. A p-value of < 0.05 was considered statistically significant for a two-tailed analysis.

Ethical approval:

The study protocol was reviewed and approved by the Institutional Research Ethics Committee and all procedures were performed following the Declaration of Helsinki (2013 revision). All participants signed informed consent forms, and confidentiality of all personal and clinical data was carefully respected during the study.

Results**Comparison of demographic variables, reproductive history, clinical manifestations, anthropometric measurements, and fasting glucose concentrations between women with PCOS and controls.**

As displayed in Table 1, there was no significant difference in age, marital status or rural residence between the women with PCOS and the healthy controls ($P > 0.05$ for all). The body mass index (BMI) was significantly higher in the PCOS group than in controls, and the PCOS group had significantly more irregular menstrual cycles, clinical hirsutism, acne, and also had a significantly higher incidence of a positive family history of PCOS and history of infertility compared to the control group (all $P < 0.05$). Furthermore, the fasting blood glucose was significantly elevated in women with PCOS compared to healthy controls ($P < 0.001$), reflecting metabolic changes in women with PCOS.

Table 1. Baseline Sociodemographic, Anthropometric, Reproductive, Clinical, and Metabolic Characteristics of Women with Polycystic Ovary Syndrome Compared with Healthy Controls

Variable	PCOS (n=80)	Controls (n=40)	P-value
Age (years)	27.64 $\pm$ 4.82	26.91 $\pm$ 4.47	0.421
BMI (kg/m ²)	29.84 $\pm$ 4.56	25.76 $\pm$ 3.91	<0.001
Married, n (%)	49 (61.3)	26 (65.0)	0.689
Rural residence, n (%)	33 (41.3)	15 (37.5)	0.684
Family history of PCOS, n (%)	27 (33.8)	5 (12.5)	0.015
Irregular menstrual cycles, n (%)	71 (88.8)	3 (7.5)	<0.001
Clinical hirsutism, n (%)	55 (68.8)	2 (5.0)	<0.001
Acne, n (%)	39 (48.8)	5 (12.5)	<0.001
Infertility history, n (%)	31 (38.8)	3 (7.5)	<0.001
Fasting glucose (mg/dL)	97.82 $\pm$ 11.24	90.46 $\pm$ 8.73	<0.001

Comparison of circulating adipokines, reproductive hormone concentrations, and metabolic parameters between women diagnosed with polycystic ovary syndrome and healthy controls.

As shown in Table 2, there are significant differences in circulating adipokines, hormonal parameters, and insulin resistance factors between women with PCOS and healthy controls. All the serum asprosin, chemerin, total testosterone, LH, LH/FSH ratio, fasting insulin and HOMA-IR levels were significantly elevated in PCOS group (all $P < 0.001$), while serum adropin level was significantly decreased ($P < 0.001$). However, no differences in FSH concentrations was found between the two groups ($P = 0.176$), indicating that the endocrine abnormalities of PCOS were mainly related to increased LH secretion, hyperandrogenism, differences in the levels of adipokines and insulin resistance.

Table 2. Comparative Analysis of Serum Asprosin, Chemerin, Adropin, Hormonal Profile, and Insulin Resistance Markers in Women with Polycystic Ovary Syndrome

Variable	PCOS (n=80)	Controls (n=40)	P-value
Asprosin (ng/mL)	8.94 ± 1.76	6.48 ± 1.39	<0.001
Chemerin (ng/mL)	187.35 ± 31.46	143.81 ± 24.58	<0.001
Adropin (ng/mL)	2.84 ± 0.73	4.12 ± 0.81	<0.001
Total testosterone (ng/mL)	0.86 ± 0.24	0.51 ± 0.17	<0.001
LH (IU/L)	11.43 ± 3.41	6.84 ± 2.13	<0.001
FSH (IU/L)	5.84 ± 1.37	6.18 ± 1.25	0.176
LH/FSH ratio	2.03 ± 0.64	1.12 ± 0.39	<0.001
Insulin (μ IU/mL)	18.76 ± 5.84	10.54 ± 3.12	<0.001
HOMA-IR	4.18 ± 1.43	2.11 ± 0.71	<0.001

Comparison of circulating biomarker concentrations and metabolic abnormalities among women with mild, moderate, and severe polycystic ovary syndrome according to disease severity.

There were substantial progressive changes in serum biomarkers and metabolic parameters with increasing severity of PCOS as presented in Table 3. Serum levels of asprosin, chemerin, testosterone and HOMA-IR were significantly higher in the moderate and severe groups of the disease compared with the mild group ($P < 0.001$ for all), while serum adropin levels were significantly lower in the moderate and severe groups of the disease than in the mild group ($P < 0.001$ for both). These findings suggest that there is a correlation between increasing disease severity with increasing metabolic dysfunction, hyperandrogenism, insulin resistance, and significant changes in the adipokine profile.

Table 3. Association of Serum Asprosin, Chemerin, Adropin, Testosterone, and Insulin Resistance with Clinical Severity of Polycystic Ovary Syndrome

Variable	Mild (n=29)	Moderate (n=31)	Severe (n=20)	P-value
Asprosin (ng/mL)	8.03 ± 1.29	8.97 ± 1.42	10.17 ± 1.68	<0.001
Chemerin (ng/mL)	172.84 ± 24.53	188.95 ± 27.31	208.42 ± 29.84	<0.001
Adropin (ng/mL)	3.23 ± 0.62	2.79 ± 0.58	2.31 ± 0.49	<0.001
Testosterone (ng/mL)	0.72 ± 0.16	0.86 ± 0.19	1.06 ± 0.21	<0.001
HOMA-IR	3.42 ± 0.91	4.11 ± 1.14	5.21 ± 1.37	<0.001

Pearson correlation analysis evaluating associations between circulating adipokines and clinical, hormonal, and metabolic characteristics among women diagnosed with PCOS.

Table 4 shows that serum asprosin and chemerin were positively correlated with BMI, testosterone, LH/FSH ratio, HOMA-IR and fasting glucose levels, and serum adropin was negatively correlated with these parameters (all $P < 0.05$). Asprosin ($r = 0.54$) and chemerin ($r = 0.57$) had the highest correlations with HOMA-IR, and adropin had the highest negative correlation ($r = -0.46$) with HOMA-IR. Unlike these three, the level of each of the three biomarkers did not correlate with age, indicating that this measurement was more related to metabolic and endocrine disturbances than to chronological age.

Table 4. Correlation of Serum Asprosin, Chemerin, and Adropin with Anthropometric, Hormonal, and Metabolic Parameters in Women with Polycystic Ovary Syndrome

Variable	Asprosin (r)	Chemerin (r)	Adropin (r)
BMI	0.46**	0.49**	-0.43**
Testosterone	0.41**	0.37**	-0.34*
LH/FSH ratio	0.35**	0.31*	-0.28*
HOMA-IR	0.54**	0.57**	-0.46**
Fasting glucose	0.33*	0.36**	-0.29*
Age	0.09	0.11	-0.08

Receiver operating characteristic analysis evaluating the diagnostic accuracy, optimal cutoff values, sensitivity, specificity, and area under the curve of serum biomarkers.

The diagnostic accuracy of serum asprosin, chemerin and adropin for the discrimination between women with PCOS and healthy controls was presented in Table 5. All three biomarkers had very good discriminatory ability, with P values (all $P < 0.001$) being statistically significant. The highest diagnostic accuracy measured by the area under the curves (AUC) was seen with serum adropin (AUC = 0.911), followed by chemerin (AUC = 0.889) and asprosin (AUC = 0.864). Adropin also displayed the highest sensitivity (87.5%) and specificity (85.0%) suggesting that it could be the most reliable single marker of the PCOS among the markers assessed.

Table 5. Diagnostic Performance of Serum Asprosin, Chemerin, and Adropin for Identifying Women with Polycystic Ovary Syndrome Using ROC Curve Analysis

Biomarker	Cut-off	AUC (95% CI)	Sensitivity (%)	Specificity (%)	P-value
Asprosin	>7.42 ng/mL	0.864 (0.794–0.919)	82.5	77.5	<0.001
Chemerin	>166.8 ng/mL	0.889 (0.823–0.938)	85.0	80.0	<0.001
Adropin	<3.41 ng/mL	0.911 (0.851–0.953)	87.5	85.0	<0.001

Discussion:

The age, marital status, and place of residence were matched between women with polycystic ovary syndrome (PCOS) and healthy controls, with no significant differences identified among the study groups (Table 1), which supports successful matching between the groups and limits the potential for demographic parameters to influence the analysis of biomarkers. Patients with PCOS had significantly higher levels of body mass index (BMI), positive family history, menstrual irregularities, elevated fasting blood glucose, hirsutism, acne, and infertility. These observations align with the known clinical features of PCOS such as reproductive dysfunction, endocrine and metabolic abnormalities. Insulin resistance and chronic low-grade inflammation are key factors involved in the pathogenesis of PCOS and are well known to be caused by obesity, especially visceral adiposity. Moreover, the more common finding of history of family members increases the role of genetic susceptibility and shared environmental factors in the

development of disease. There are other studies reporting similar demographic and clinical observations, including a higher prevalence of obesity, hyperandrogenic symptoms and metabolic issues in women with PCOS compared to healthy women [17,18]. On the other hand, some studies did not find any significant differences in BMI between cases and controls, as the BMI of the cases and controls were selected to be within a narrow range or as the study design involved including mostly lean women with PCOS, thus reducing the effect of BMI on disease expression [19].

As seen in Table 2, the levels of asprosin and chemerin were significantly higher and adropin levels were significantly lower in women with PCOS compared to normal women. These changes were also associated with markedly elevated serum testosterone and luteinizing hormone (LH) and LH/FSH ratio, fasting insulin and HOMA-IR. The observed rise in asprosin is biologically plausible as this fasting induced adipokine is known to promote glycogenesis, and is often high in obesity and insulin resistant states. Maintenance of an elevated level of asprosin may thus exacerbate hyperglycemia and metabolic dysfunction in PCOS. Similarly, chemerin is an adipokine as well as an inflammatory mediator that plays roles in adipocyte differentiation, recruitment of immune cells, development of ovarian follicles and glucose metabolism. Increased levels of chemerin are associated with decreased folliculogenesis, ovarian dysfunction and insulin resistance [20]. By contrast, adropin is a peptide hormone that plays a role in energy homeostasis, endothelial function, and insulin sensitivity, so it might be associated with the impaired use of glucose and aggravating metabolic abnormalities in PCOS. Higher levels of testosterone, LH and LH/FSH ratio also indicate increased hypothalamic-pituitary-ovarian axis dysregulation and higher levels of insulin resistance (HOMA-IR) confirm significant insulin resistance [21]. The results agree with many clinical studies and recent systematic reviews that have reported increased concentrations of circulating asprosin and chemerin and decreased concentrations of circulating adropin in women with PCOS [22]. All the evidence points towards close association of these biomarkers with endocrine dysfunction and metabolic impairment [23].

However, some published investigations have failed to report the same results. Other studies found no significant difference in the levels of either asprosin or adropin between the PCOS and healthy women [24,25]. Several methodological and biological issues may account for these differences: sample size was small in both, the ethnic background was different between the two, the sensitivity of the ELISA assays was different, and the diagnostic criteria were based on the Rotterdam criteria in the one study and the NIH criteria in the other, with the latter being the more stringent criteria. Some studies only included lean women and other studies mostly included obese women with PCOS [26]. Given the strong association between adipokine concentrations and adiposity and insulin resistance, the distribution of BMI can significantly influence the levels of these circulating biomarkers. In addition, there were variable control of the use of medications like metformin or oral contraceptives, differences in diet composition, and varying levels of physical activity [27]. Also, the use of medication like metformin or oral contraceptives, diet, and physical activity were managed differently in the studies, contributing to inconsistent observations. Thus, differences between studies would likely be due to study design, rather than to differing biological mechanisms [28].

The findings in Table 3 revealed the gradual increase of asprosin, chemerin, testosterone, and the HOMA-IR in mild, moderate, and severe PCOS, while the adropin level showed the opposite trend. This pattern suggests that these biomarkers are not only linked to the presence of PCOS, but to its clinical severity as well [29]. The main mechanism responsible for these observations is probably progressive insulin resistance. Hyperinsulinemia also promotes an excessive androgen production by the theca cell and prevents the hepatic production of sex hormone-binding globulin, leading to high levels of free testosterone. An insulin resistance can also cause adipocytes to be dysfunctional and result in increased

production of inflammatory cytokines, which further increase chemerin and asprosin secretion [30]. However, decreased metabolic homeostasis could down regulate the production of adropin and further decrease the utilization of glucose and endothelial function. In several clinical studies, a positive relationship between chemerin and insulin resistance and hyperandrogenism, as well as an inverse relationship between adropin and metabolic deterioration have been reported, with both changes reported to be severity-dependent [31]. The results confirm the view of adipokines that actively contribute to the progression of disease and are not just a passive marker.

As shown in Table 4, serum asprosin was significantly positively correlated with BMI, testosterone, LH/FSH ratio, fasting glucose and HOMA-IR, while chemerin and adropin were significantly inversely correlated to these variables. In PCOS, these relationships highlight that there is a close interplay between metabolic dysfunction, obesity and reproductive hormonal imbalance. Insulin resistance seems to be the main pathogenic links between these biomarkers. Increased insulin levels stimulate production of androgens in the theca cells of the ovary and inhibit the production of sex hormone binding globulin by the liver, thus increasing free testosterone levels [32]. At the same time, fat tissue, which is located in the inner body cavity, secretes higher levels of inflammatory adipokines like chemerin, and reduces the levels of protective metabolic peptides, such as adropin. The positive correlation between asprosin and HOMA-IR also suggests a role for asprosin in regulating hepatic glucose production and increasing insulin resistance. The same correlation patterns have been consistently reported in observational studies and meta-analyses of adipokines in PCOS in the last years [33,34].

Table 5 highlighted good diagnostic performance of all three biomarkers in differentiating women with PCOS from healthy controls, the highest of which was adropin followed by chemerin and asprosin. According to these results, these adipokines can be used as clinically relevant complementary markers in the diagnosis of PCOS, if used in combination with the traditional clinical, hormonal and ultrasonographic criteria. The correlation between adropin and insulin sensitivity and early metabolic dysfunctions could explain its superior diagnostic value, while the link between chemerin and chronic inflammation and ovarian dysfunction may indicate that this cytokine is a more representative marker of this state [35]. Slightly less accurate, asprosin showed good diagnostic potential by its link with glucose metabolism and insulin resistance. These have been reported as diagnostic in a variety of recent studies, both as individual markers and as combinations of markers [36]. Other studies, however, achieved less diagnostic accuracy, possibly due to variations in methods for measuring assays, ethnicity, obesity rates, disease severity, or sample size. The present results suggest that asprosin, chemerin, and adropin may be novel biomarkers for the PCOS diagnosis; however, further multicenter prospective studies using standardized laboratory techniques are needed before these markers can be applied in the clinical setting [37].

Conclusion:

The current findings showed that serum levels of asprosin, chemerin and adropin were significantly different in women with PCOS, and that hormonal imbalance is present as well as an increased level of insulin resistance. These biomarkers were strongly linked to disease severity and important metabolic parameters pointing to their clinical relevance. Of the evaluated markers, adropin was the most accurate specific marker to distinguish PCOS from healthy women. Overall, the results of these adipokines are promising to be used as a complement to diagnose and to metabolically assess PCOS.

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