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Serum Asprosin as a Potential Biomarker for Dental Caries and Periodontal Disease in Polycystic Ovary Syndrome: A Case-Control Study

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Abstract

Background/Objectives: Polycystic ovary syndrome (PCOS) is a prevalent endocrine condition in women. The impacts on oral and dental health have not been explicitly articulated. This study aimed to ascertain the association between serum asprosin levels, DMFT, and periodontitis in women with polycystic ovary syndrome (PCOS). Methods: The study included 100 participants: 60 women with PCOS and 40 healthy individuals. Clinical attachment loss (CAL), probing pocket depth (PPD), plaque index (PI), and gingival bleeding index (GBI) were documented. An enzyme-linked immunosorbent assay (ELISA) was used to measure serum asprosin and HOMA-IR concentrations. Results: An individual diagnosed with DMFT and periodontitis in women with PCOS had a markedly elevated concentration of asprosin (5.67 $\pm$ 3.55 ng/mL) compared to the control group (3.23 $\pm$ 2.25 ng/mL; $p < 0.0001$). The correlation between serum asprosin levels and clinical periodontal markers was found to be significantly positive. Conclusions: This case-control study suggests that elevated serum asprosin levels in women with polycystic ovary syndrome (PCOS) may be associated with poor periodontal sensitivity to proinflammatory stimuli.

Keywords: asprosin; PCOS; periodontitis; DMFT; dental caries; oral pH

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1. Introduction

Polycystic ovary syndrome (PCOS) is a hormonal condition that affects many women of reproductive age. The aetiology is unidentified. Polycystic ovary syndrome (PCOS) affects approximately 6–8 percent of women and is characterised by repeated anovulation, clinical and/or biochemical hyperandrogenism, and ovaries exhibiting multiple cystic

formations [1]. This syndrome impacts several systems and is characterised by menstrual abnormalities (oligoamenorrhea and dysfunctional uterine haemorrhage), manifestations of hyperandrogenism (hirsutism, acne, and seborrhoeic skin), obesity, and metabolic syndrome [2]. Insulin resistance (IR) is fundamental to the pathogenesis of polycystic ovary syndrome (PCOS) and refers to an inability to provide a sufficient physiologic response despite normal insulin concentrations. Insulin resistance and hyperinsulinemia are prevalent disorders in people with polycystic ovary syndrome (PCOS) [3]. Insulin resistance (IR) is not a diagnostic criterion for polycystic ovary syndrome (PCOS), although it is seen in 50–80% of women with the condition, irrespective of their body mass index (BMI), and it exacerbates the progression to diabetes by increasing susceptibility to prediabetes [4]. In recent years, the association between polycystic ovary syndrome (PCOS), which presents many problems, and periodontal disorders resulting from persistent low-grade inflammation has emerged as a significant concern in reproductive endocrinology [5]. The correlation between PCOS and inflammation, akin to periodontal illnesses, together with its enduring metabolic repercussions, has prompted the suggestion that it may be linked to periodontal parameters [6]. The precise biological mechanisms are not fully understood; however, hyperestrogenic and hyperandrogenic conditions in PCOS are believed to induce structural alterations in the gums and gingival inflammation, facilitating bacterial colonisation and elevating the risk of periodontal disease [7]. Consequently, research has indicated that the early diagnosis of periodontal disorders in PCOS patients and the commencement of therapy are essential for mitigating potential long-term metabolic consequences [8]. If chronic inflammation in periodontal tissues remains untreated, the supporting structures of the teeth will deteriorate over time, leading to dental caries and tooth loss [9–12]. Adipose tissue has been regarded as a passive energy reservoir that offers insulation against cold temperatures. It is also an endocrine organ since it synthesises adipokines or hormones generated from adipose tissue [13]. A novel glucogenic adipokine called asprosin is encoded by two exons (exon 65 and exon 66) of the Fibrillin 1 (FBN1) gene. White adipose tissue makes a majority of it when one does not eat. Asprosin has a diverse role within the central nervous system (CNS), peripheral tissues, and organs [14]. Asprosin levels in circulation are heightened in many cardio-metabolic disorders, such as obesity and type 2 diabetes [15]. A limited number of studies in the literature have examined its association with periodontal tissues, and no studies have investigated dental hard tissues or asprosin levels. In the few studies examining the association between PCOS and periodontal health as well as carious lesions, it remains unclear when oral health begins to decline in PCOS, since the studies have included patients of all ages. This study sought to examine higher asprosin levels associated with dental caries and periodontitis in adult women diagnosed with PCOS during their early reproductive phase.

2. Methods and Materials

2.1. Individual Selection

This case-control study included patients aged 20 to 40 who were admitted to the Consulting Department from 20 June to 12 December 2025, at the Women and Children Hospital in Al-Anbar Province, Iraq. This research received approval from the Scientific Research Evaluation Ethical Committee of the Ministry of Health, Al-Anbar Directorate of Health, Iraq (no. 2025015, date: 22 April 2025). Informed written permission was acquired from all patients before their inclusion in the trial. A total of 100 individuals were enrolled: 60 women with PCOS and 40 control acting as periodontally healthy women. A professional dentist evaluated the clinical periodontal parameters.

2.2. Exclusion and Inclusion Criteria

The exclusion criteria for the study were: (1) patients with systemic diseases (liver disease, kidney disease, diabetes, thyroid disease, tumor, congestive heart disease, heart failure, and coronary heart disease), (2) history of ovarian cancer, (3) medications affecting hormones (within the preceding 2 months), and (4) oral infections or recent antibiotic therapy (within 2 months). Furthermore, the study had the following inclusion criteria: (1) female patients aged 20–40, (2) a clinical diagnosis of PCOS by a gynecologist (based on WHO criteria), (3) systemically healthy individuals, and (4) patients who did not receive periodontal treatment for at least the preceding two months.

2.3. Dental Visit and Oral Saliva Collection

Dental and periodontal examinations were performed by the same dentist (blinded to PCOS status). All patients were interviewed using a standardised, structured questionnaire to collect their demographic and oral health habit data. The participants' heights and weights were measured and recorded to determine their BMIs at the same time in the morning. Underweight was defined as having a low BMI. Oral saliva collection was conducted at 9:00 a.m. The participant occupied the dentist's chair and was instructed to clean her mouth with water to eliminate dirt. The person was instructed to swallow any residual saliva in her oral cavity, following which the timer was started. Unstimulated saliva was collected every minute into a beaker. The collected saliva was transferred to plastic Eppendorf tubes and kept at −20 °C before pH analysis.

2.4. Measurement of Periodontal Parameters

The specialist dentist conducted a periodontal index assessment. Clinical factors, including plaque index, gingival bleeding index, clinical attachment loss, and probing pocket depth, were assessed. An examination of clinical parameters included the third molars for all existing teeth. PPD, CAL, GBI, and PI values were gathered from six tooth surfaces (midbuccal, midlingual, buccal, lingual, mesial, and distal) and documented in the participant's file. The Clinical Attachment Level (CAL) was assessed from the cement–enamel junction to the periodontal pocket's base. GBI was evaluated based on the occurrence of bleeding for ten seconds after probing (0 or 1) [16]. PI assessed a value ranging from 0 to 3 [17]. The periodontal parameter evaluations were performed manually using the Williams Periodontal Probe, which was used for patient diagnosis [18].

2.5. Determination of Serum Asprosin and Insulin Levels

Five milliliters of blood were drawn from a peripheral vein between 8:30 and 11:30 in the morning after an overnight fast. After leaving the sample for approximately 30 min, the samples were centrifugated at 10,000 rpm for 5 min in order to extract the sera. An enzyme-linked immunosorbent assay (ELISA) was used to quantify asprosin and insulin concentrations in the serum using the Human ELISA kit from Elabscience, Texas, USA. An assay was conducted in accordance with the manufacturer's guidelines.

2.6. The Sample Size Calculation

The study's sample size analysis, which looked at two patient cohorts, had an effect size of 0.26 on the CAL and an expected SD of 0.5. [19], with a statistical power of 80% and a two-tailed significance criterion of 0.05. Approximately 57 participants per group were deemed necessary to achieve sufficient statistical power. A total of roughly sixty people were recruited in the patient group, yielding a power score of 80%.

2.7. Statistical Data

Statistical analysis was used in this research (Medical Calculations (MEDCALC)). The statistics are shown herein as means ± standard deviations (SDs), with a 95% confidence interval (CI) added to the mean. The data distribution was checked for normality using the Shapiro–Wilk test or an equivalent method. For continuous variables, an independent sample *t*-test was used to perform group comparisons (e.g., PCOS vs. control). A Pearson's correlation analysis was used to identify relationships between the continuous variables. The prediction model's or metric's diagnostic performance was assessed with the use of ROC curve analysis, which reported the AUC, ideal cut-off threshold, sensitivity, and specificity. Statistical significance was determined in all analyses when the *p*-value was less than 0.01. All analyses were processed using GraphPad Prism (version 10).

3. Results

3.1. The Population of the Study

A total of 100 women, 60 with polycystic ovary syndrome (PCOS) and 40 controls, participated in the study. The mean ages of the control and PCOS participants were 29 and 30 years, respectively. As shown in Table 1, there was a difference between the groups in terms of body mass index ($p < 0.01$), but there was no difference between the groups in terms of place of residence, tooth brushing frequency, and frequency of dental visits ($p > 0.01$). However, it was found that proximal tooth cleaning was less frequent and the frequency of sugar intake between meals was higher in those with PCOS compared to the controls ($p = 0.023$ and $p = 0.037$, respectively).

Table 1. Characteristics of the sociodemographic data for the control and polycystic ovary syndrome (PCOS) women.

Variable	Control (n = 40)	PCOS (n = 60)	*p*-Value
Age, mean ± SD	29.85 ± 6.35	30.20 ± 5.99	0.751
Place of residence n, %			
City	22 (55)	33 (55)	0.862
Town	10 (25)	15 (25)	
Village	8 (20)	12 (20)	
BMI, mean ± SD	24.92 ± 2.49	29.08 ± 3.81	0.001 *
Toothbrushing frequency n, %			
>2 times/day	10 (25)	15 (25)	0.345
Twice a day	20 (50)	30 (50)	
≤1 time/day	10 (25)	15 (25)	
Proximal tooth cleaning			
Once a day	6 (15)	12 (20)	0.023
Once a week	24 (60)	33 (55)	
Never	10 (25)	15 (25)	
Dental visit			
Twice a year	10 (25)	18 (30)	0.468
Once a year	26 (65)	30 (50)	
Never	4 (10)	12 (20)	

Table 1. *Cont.*

Variable	Control (n = 40)	PCOS (n = 60)	*p*-Value
Sugar intake between meals			
1 or 2/day	10 (25)	18 (30)	0.037
3 or 4/day	22 (55)	24 (40)	
5 or more/day	8 (20)	18 (30)	

p-values calculated with Student *t*-tests for continuous variables and differences between the categorical variables (shown by count (n) and rate (%)) were assessed by χ^2-tests; *: significant at the $p < 0.01$ level; BMI was derived from the weights and heights of the patients (kg/m^2).

3.2. Periodontal Parameters, DMFT, and Oral pH

The comparison results of the two groups in terms of periodontal health status and oral pH are shown in Figure 1 and Table 2. GBI percentage and mean CAL, PPD, and PI values were found to be lower in the control group compared to the PCOS group ($p < 0.0001$, $p < 0.000$ and $p = 0.0005$, and $p < 0.0001$, respectively). The mean decayed, missing, and filled teeth (DMFT) number of the control group was 6.65, whereas it was 9.06 in the PCOS group ($p = 0.0005$). The PCOS cases demonstrated significantly lower oral pH and were thus more acidic, and it was an obvious biochemical change. Integrated dental care is mandatory in Iraq for holistic PCOS management, as these data pointed towards an increased risk of periodontitis, caries, and an acidic oral environment in the women with PCOS.

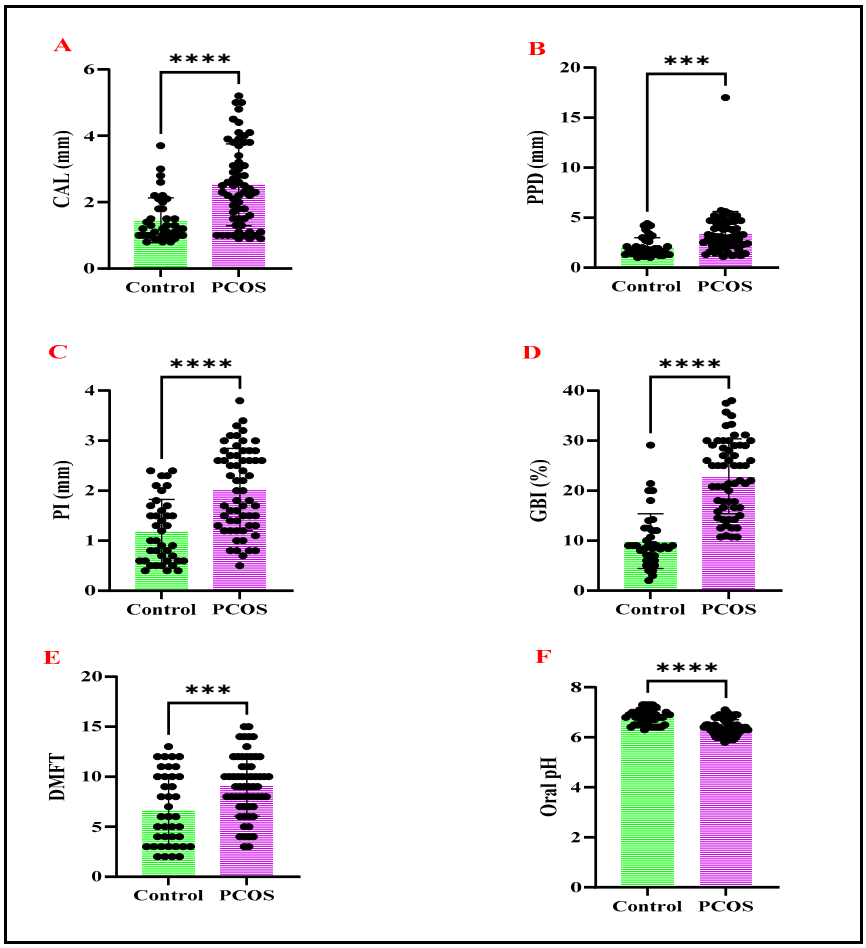

Figure 1. Clinical parameters compared between the PCOS and control groups included: (**A**) clinical attachment loss (CAL); (**B**) probing pocket depth (PPD); (**C**) plaque index (PI); (**D**) gingival bleeding index (GBI); (**E**) decayed, missing, and filled teeth (DMFT); and (**F**) oral pH. ****: $p < 0.0001$, ***: $p < 0.0005$, statistically significant at $p < 0.01$, *t*-test.

Table 2. Periodontal parameters, DMFT, and oral pH in all subjects.

Variables	Control	PCOS	95% Confidence Interval	*p*-Value
	Mean ± SD	Mean ± SD		
CAL (mm)	1.45 ± 0.21	2.52 ± 0.35	0.647–1.496	<0.0001 *
PPD (mm)	2.04 ± 1.12	3.39 ± 2.54	0.609–2.080	0.0005 *
PI (mm)	1.18 ± 1.33	2.02 ± 2.65	0.530–1.143	<0.0001 *
GBI (%)	9.90 ± 4.31	22.80 ± 6.32	10.23–15.71	<0.0001 *
DMFT	6.65 ± 2.76	9.06 ± 4.66	1.102–3.732	0.0005 *
Oral pH	6.81 ± 0.29	6.38 ± 0.94	0.555–0.302	<0.0001 *

CAL: clinical attachment loss; PPD: probing pocket depth; PI: plaque index; GBI: gingival bleeding index; DMFT: decayed, missing, and filled teeth; *: significant at ($p < 0.01$) *t*-test.

3.3. Stage and Grade Indicated for Periodontitis

Eleven patients (18.33%) had stage II periodontitis, twenty-one patients (35.10%) had stage III periodontitis, and twenty-eight patients (46.66%) had stage IV periodontitis among the patients with PCOS with grade C periodontitis, which impacted 60 patients (100%). The staging and grading of periodontitis variances were statistically significant at $p = 0.0001$, as shown in Table 3.

Table 3. Classification of periodontitis in PCOS cases: categorisation based on stage and grade.

Variables		PCOS Cases	*p*-Value
Stage of periodontitis, n (%)	Stage I	0 (0.0)	-
	Stage II	11 (18.33)	0.0001 *
	Stage III	21 (35.10)	
	Stage IV	28 (46.66)	
Grade of periodontitis, n (%)	Grade A	0 (0.0)	-
	Grade B	0 (0.0)	-
	Grade C	60 (100.0)	-

PCOS: polycystic ovary syndrome; *: statistically significant at $p < 0.01$.

3.4. Serum Levels of Asprosin, HOMA-IR, Insulin, and FBS Were Highly Correlated with PCOS Activity

The assessment of serum asprosin level was significantly higher in the PCOS patients (5.67 ± 3.55 ng/mL) than in the control group (3.23 ± 2.25 ng/mL; $p < 0.0001$) (95% CI, 1.936–2.941). The mean of the HOMA-IR levels was significantly higher in the PCOS patients than in the healthy individuals (3.13 ± 2.67 ng/mL vs. 1.85 ± 1.43 ng/mL; $p < 0.0001$) (95% CI, 0.811–1.756). The findings showed an increase in insulin level for the PCOS group against the controls (13.30 ± 5.31 ng/mL vs. 8.79 ± 3.54 ng/mL; $p < 0.0001$) (95% CI, 2.971–6.042). Furthermore, the results showed an increase in fasting blood sugar (FBS) levels in the PCOS group compared to the controls (134.5 ± 7.22 mg/dL vs. 85.83 ± 4.39 mg/dL; $p < 0.0001$) (95% CI, 2.971–6.042), as seen in Figure 2 and Table 4.

3.5. Receiver Operating Curve Characteristic (ROC)

When comparing the PCOS patients with the control individuals, asprosin showed excellent diagnostic performance (AUC > 0.9). The test had a strong specificity of 82.5% and a high sensitivity of 86.7% when used with the recommended cut-off of ~4.00 ng/mL. In the current study, the crude AUC of the asprosin ROC curve for PCOS detection was 0.915, indicating excellent significance. Therefore, it could be used as an ideal indicator

for a PCOS diagnosis. Furthermore, the findings showed a good diagnostic performance (AUC of >0.8). The test had a strong specificity of 75.0% and a high sensitivity of 71.7% when used with the recommended cut-off of 2.261 ng/mL. The study showed that the crude AUC of the HOMA-IR ROC curve for PCOS detection was 0.806, indicating good significance. Therefore, both biomarkers could be ideal indicators of a PCOS diagnosis, as seen in Figure 3 and Table 5.

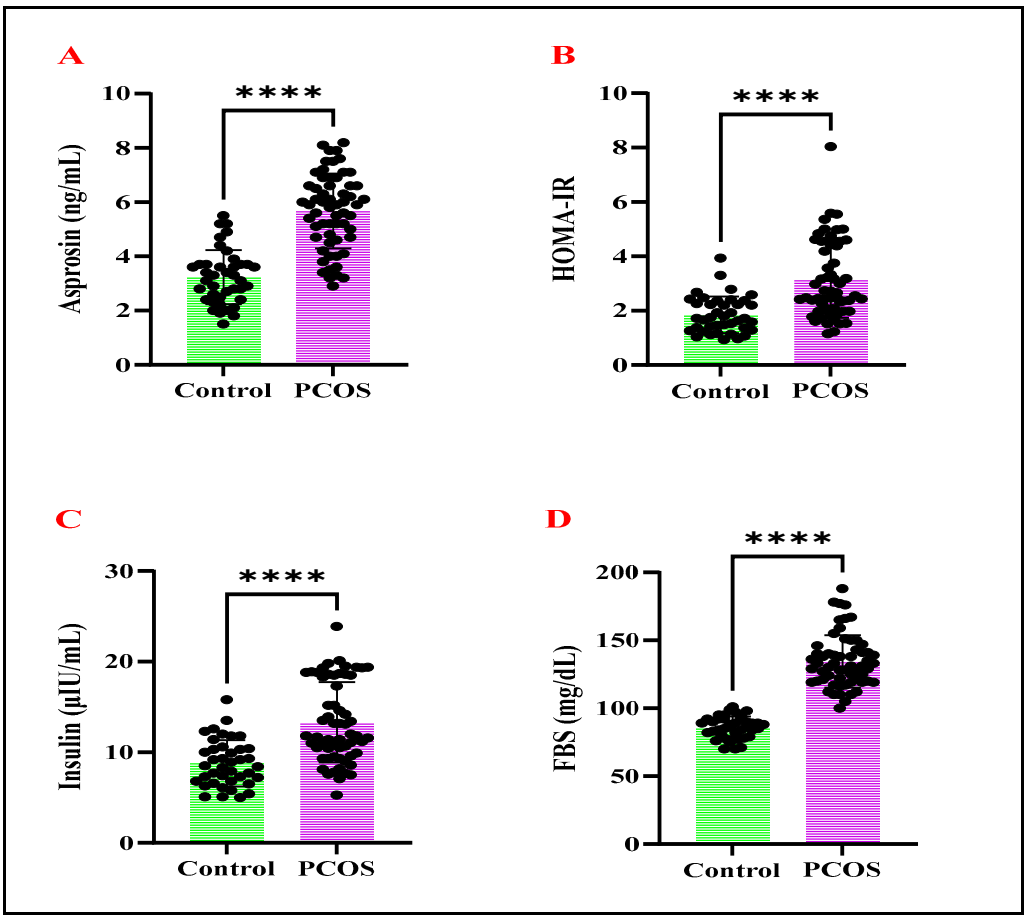

Figure 2. Serum levels of: (**A**) asprosin; (**B**) HOMA-IR; (**C**) Insulin, and (**D**) Fasting blood sugar (FBS) in the women with PCOS compared to the controls (****: significant at $p < 0.01$, *t*-test).

Table 4. The means of the asprosin, HOMA-IR, insulin, and fasting blood sugar (FBS) levels in the PCOS group compared to the controls.

Variables	Control	PCOS	95% Confidence Interval	*p*-Value
	Mean ± SD	Mean ± SD		
Asprosin (ng/mL)	3.23 ± 2.25	5.67 ± 3.55	1.936–2.941	<0.0001 *
HOMA-IR	1.85 ± 1.43	3.13 ± 2.67	0.811–1.756	<0.0001 *
Insulin (µIU/mL)	8.79 ± 3.54	13.30 ± 5.31	2.971–6.042	<0.0001 *
FBS (mg/ dL)	85.83 ± 4.39	134.5 ± 7.22	42.23–55.02	<0.0001 *

HOMA-IR: (homeostatic model assessment for insulin resistance); PCOS: polycystic ovary syndrome; FBS: fasting blood sugar; *: statistically significant at $p < 0.01$, *t*-test.

3.6. Serum Asprosin Levels Exhibited a Substantial Correlation with Periodontal Parameters, DMFT, and Oral pH

The coefficient of Pearson's correlation indicated a positive statistical significance between the periodontal measurement scores (PI, GBI, PPD, and CAL), DMFT, and serum

asprosin level ($p = 0.0001$). Furthermore, oral pH exhibited a statistically significant negative correlation with serum asprosin level ($p = 0.0001$), as seen in Figure 4A–F.

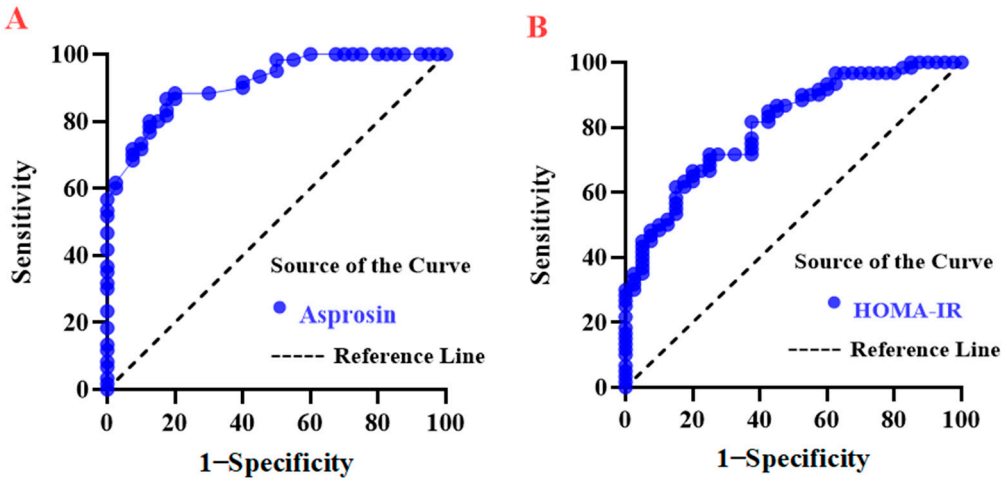

Figure 3. The (ROC) patterns of performance for identifying PCOS included: (**A**) asprosin and (**B**) HOMA-IR.

Table 5. The (ROC) analyses of asprosin levels for estimating clinical abatement.

Variable	AUC	Optimal Cut-Off	Sensitivity	Specificity	95% CI	*p*-Value
Asprosin	0.915	≈3.950 ng/mL	0.867 (86.7%)	0.825 (82.5%)	0.863–0.966	<0.0001 *
HOMA-IR	0.806	≈2.261 ng/mL	0.717 (71.7%)	0.750 (75.0%)	0.722–0.890	<0.0001 *

AUC: Area Under the Curve, *: Statistically significant at ($p < 0.01$).

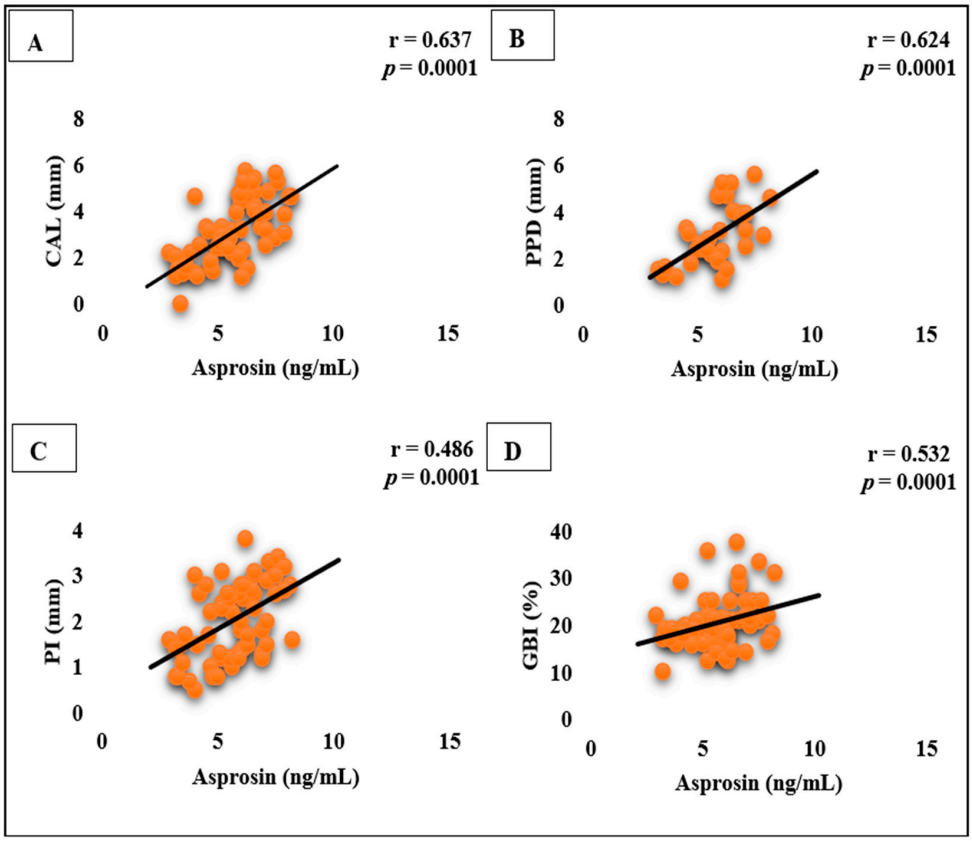

Figure 4. *Cont.*

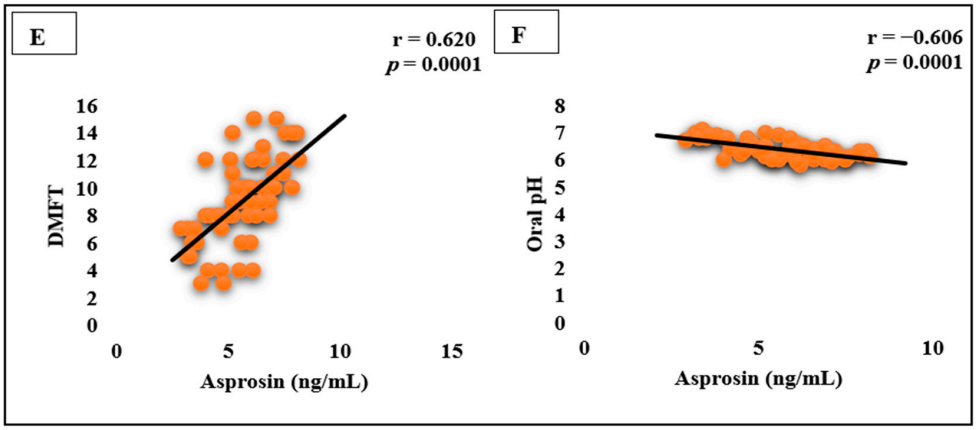

Figure 4. Serum level of asprosin (ng/mL) correlated with: (**A**) clinical attachment level (CAL), (**B**) probing pocket depth (PPD), (**C**) plaque index (PI), (**D**) gingival bleeding index (GBI), (**E**) decayed, missing, and filled teeth (DMFT), and (**F**) Oral pH.

4. Discussion

This study aimed to evaluate the relationship between blood levels of asprosin and clinical periodontal indicators in patients with polycystic ovary syndrome (PCOS). Consequently, we investigated the diagnostic ability and measurement of serum asprosin levels using ELISA for potential periodontal tissue deterioration induced by PCOS. To our knowledge, no previous studies have examined the serum levels of asprosin in periodontal patients with PCOS. Our findings corroborated and enhanced prior research. The participants with PCOS were younger and had higher BMIs in comparison to those without the syndrome. They had increased circulating values of asprosin, DMFT, and periodontal parameter levels as compared to the controls. Many of the hormonal and metabolic findings reported here fit well with what one could expect when comparing young women with and without PCOS [13,20]. PCOS is not an exclusive ovarian dysfunction, but rather, a total endocrine and metabolic disorder related to insulin resistance. The meta-analysed data from the PCOS patients showed increased insulin, glucose, HOMA-IR, triglyceride, and asprosin levels and reduced HDL-C levels. Asprosin regulates lipid and carbohydrate metabolism [21], stimulates the production of reactive oxygen species and inflammation-associated cytokines, and decreases insulin secretion [22,23]. In obese children and adult subjects, asprosin secretion is increased and related to increased waist circumference and triglyceride levels [24–26]. Increased asprosin levels have also been reported in patients with insulin resistance and T2DM [21,25]. Some experimental studies have proposed the use of antibodies against asprosin to neutralise metabolic syndrome [27]. Our results showed that asprosin secretion is altered in women with PCOS during reproductive years as compared to women without the syndrome. It remains to be determined what the roles of this newly identified hormone are in the development of periodontal disease in PCOS. The findings suggested that asprosin may be a metabolic biomarker for PCOS. Based on the consensus among researchers, it also seems to be linked to IR, suggesting it might represent a key link in a (possible) pathogenic chain ranging from adipokine dysregulation to hyperandrogenism and hyperinsulinemia in PCOS. However, the need for further studies on phenotype groups (stratified for BMI, IR, and androgen type) to determine whether asprosin is a cause of PCOS, caused by it, or just a surrogate is emphasised by the disparities [28]. Our results agreed with those of other studies, which creates a two-way street in that PCOS's systemic inflammation exacerbates periodontal disease and periodontitis, the chronic infection of the mouth caused by PD. Findings from studies such as that which described higher levels of inflammatory markers in teeth in PCOS

women support this cause-and-effect relationship [29]. Findings have reported that women with PCOS had substantially higher concentrations of asprosin and significantly increased rates of CAL, PPD, PI, and GBI. Both elevated adipokines and degradation of periodontal tissue are induced by the metabolic disturbance related to PCOS. The pathophysiological association between hyper-asprosinemia and PCOS is in agreement with this connection, where systemic inflammation disrupts the local immuno-inflammatory equilibrium at the gingival crevice, motivating dysbiotic biofilm and tissue demise [30–32]. However, local bacterial plaque (indexed by PI) does contribute substantially to GBI. Likewise, in the PI correlation, the increased plaque accumulation, which stimulates asprosin, may serve to fully mediate the relationship between the two variables and GBI [33]. Asprosin levels were significantly positively correlated with DMFT score in the PCOS women; thus, it is possible to speculate on an association between this metabolic hormone and higher cumulative caries experience. There are several plausible side channels that would make this possible. First, the higher concentration of glucose in saliva found for IR, and perhaps hyperglycemia, may result in caries-promoting conditions in the oral cavity. Second, these patients may suffer from systemic inflammation that alters the composition and flow of saliva and thus it is less effective in remineralisation and buffering. Third, modifications in diet or medication use for the management of PCOS may influence the risk of cavities. The finding is compatible with the scarce but increasing evidence suggesting an association between some features of metabolic syndrome, namely insulin resistance and dysglycemia, and heightened dental caries risk [34]. Our results demonstrated that the metabolic hormone asprosin could be a candidate linked to a more acidic oral cavity, as there was a significant negative correlation between high levels of asprosin and low salivary pH in the PCOS group. Acid erosion and demineralisation of tooth enamel are both due to low pH, and so this finding has great implications. An excessive acidosis pH in the oral environment and an increased incidence of caries related to systemic metabolic disruption, as observed in diabetes, perfectly align with the principles of cariology. It sets up a possible biochemical path connecting PCOS metabolism to oral disease [35]. The receiver operating characteristic (ROC) analysis for asprosin indicated its potential for use in diagnostics in subjects with periodontal disease and PCOS. It was demonstrated that the sensitivity and specificity of asprosin in sera were useful indicators for distinguishing between periodontitis and PCOS outcome. Previously, a study found asprosin to be a more accurate biomarker for differentiating between gingivitis and healthy tissue, particularly in terms of sensitivity and specificity [36]. Nonetheless, our study's shortcomings include a restricted sample size, a brief duration, and the inability to entirely exclude several confounding variables, such as the type of therapies administered. Lastly, a substantial body of evidence has supported the correlation between periodontitis-related PCOS and the inflammatory components of each condition. Currently, there is little evidence available on the role of biomarkers in the proposed causal relationship between periodontitis (PD) and PCOS. The limitations arise from considerable variability in the investigation's design, participant demographics, assay methods, and evaluated biomarkers. In the future, a prospective study and a randomised controlled trial with biological data will be needed to establish the causal relationship between periodontal inflammation and PCOS. Further inquiry is necessary to elucidate the function of biomarkers in connecting these disorders.

5. Conclusions

In conclusion, asprosin levels were significantly elevated in individuals with PCOS and showed positive relationships with DMFT and the severity of periodontal disease. Increased asprosin levels were also associated with poor periodontal sensitivity to proinflammatory stimuli.

Author Contributions: M.S.J.: methodology, investigation, and project administration; C.S.-R.: resources, software, formal analysis, data curation, and funding acquisition; H.A.N.N.: conceptualisation, methodology, and investigation; G.T.K.: formal analysis, data curation, methodology, and investigation; S.A.A.: methodology, investigation, and project administration; E.R.A.: software, formal analysis, and data curation; C.T.Z.: writing—review and editing; A.A.A.-K.: conceptualisation, methodology, investigation, writing-original draft, project administration, and supervision. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Ministry of Health, Al-Anbar Directorate of Health, Iraq (protocol code 2025015; date of approval: 22 April 2025).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patients to publish this paper.

Data Availability Statement: Data are contained within the article.

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Conflicts of Interest: The authors declare that they have no known competing financial interests.

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