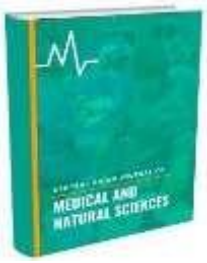

CENTRAL ASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES
https://cajmns.casjournal.org/index.php/CAJMNS
Volume: 07 Issue: 03 | July 2026 ISSN: 2660-4159

Article

Evaluation of Adiponectin and Resistin in Early Diagnosis and Prediction of Pre-eclampsia

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Citation: Hassan, H. A.,, Evaluation of Adiponectin and Resistin in Early Diagnosis and Prediction of Pre-eclampsia. Central Asian Journal of Medical and Natural Science 2026, 7(3), 389-398.

Received: 10th Feb 2026
Revised: 21th Mar 2026
Accepted: 08th Apr 2026
Published: 5th June 2026

Abstract: Background: Inflammation, organ damage, and endothelial dysfunction characterize preeclampsia, a pregnancy-specific multisystem hypertension disease. Adipokines, particularly resistin and adiponectin, have been identified as possible participants in the metabolic and inflammatory pathways that may be involved in the development of disease. **Objective:** The purpose of this research was to examine the relationship between preeclampsia serum adiponectin and resistin levels and their function as early diagnostic and disease severity predictive markers. **Methods:** There were 88 pregnant women in this case-control research; 30 were healthy controls, 29 had mild preeclampsia, and 29 had severe preeclampsia. The guidelines set out by the American College of Obstetricians and Gynecologists were used to define preeclampsia. An ELISA was used to ascertain the amounts of resistin and serum adiponectin. On automated analyzers, routine parameters related to renal and hepatic biochemistry were measured. For group comparisons, either Kruskal-Wallis or ANOVA tests were utilized. The use of Spearman coefficients allowed for the evaluation of correlations. In order to find independent factors that could predict the presence and severity of the disease, logistic regression models were developed. In order to evaluate the efficacy of the diagnostic, ROC curve analysis was performed. **Results:** The levels of resistin and adiponectin were found to be significantly higher in the mild and severe preeclampsia groups compared to the control group ($p < 0.001$). Strong associations were seen with systolic blood pressure ($r = 0•816$, $p < 0•0001$), uric acid ($r = 0•49$, $p = 0•0001$), and gestational age ($r = .477$, $p = 0•0002$), and resistin had a clear progressive rise (8.55 ± 1.43 vs 10.98 ± 1.06 vs 14.16 ± 1.04 mg/L). Even while adiponectin increased (5.05 ± 1.19 vs 10.93 ± 2.43 vs 16.83 ± 4.19 mg/L), it was not as effective in differentiating between the groups. As compared to adiponectin, resistin performed better in ROC analysis for severe preeclampsia (AUC = 0.980 vs. 0.866). Resistin was found to be a significant predictor of both preeclampsia and the severity of the condition, according to logistic regression (OR = 4.93, 95% CI: 2.31-10.54, $p < 0.001$). **Conclusions:** Serum resistin outperforms adiponectin in both diagnostic and prognostic tests, and its rise is linked to the severity of preeclampsia. For the purpose of risk stratification and severity evaluation in preeclampsia, resistin could be a clinically valuable supplementary biomarker.

Keywords: *Preeclampsia, Adiponectin, Resistin, Uric acid, Traditional renal markers.*

1. Introduction

Preeclampsia is a complex, pregnancy-specific hypertensive disorder characterized by new-onset hypertension after 20 weeks of gestation accompanied by proteinuria and/or maternal organ dysfunction. It remains a leading cause of maternal and perinatal morbidity and mortality worldwide, particularly in low- and middle-income countries [1][2][3]. The global burden of the disease is further compounded by its unpredictable onset and variable clinical course, which can range from mild hypertension to life-

threatening complications affecting multiple organ systems. Preeclampsia is now understood as a complex illness including aberrant placentation, systemic endothelial dysfunction, oxidative stress, and excessive inflammation [4],[5].

At the molecular level, abnormal trophoblastic invasion and spiral artery remodeling cause placental hypoxia and the release of antiangiogenic and pro-inflammatory mediators into the maternal circulation, damaging endothelium. Inflammatory cytokines and soluble fms-like tyrosine kinase-1 (sFlt-1) impair vascular homeostasis and cause systemic vasoconstriction. Placental pathology and maternal systemic disease are linked by endothelial dysfunction [1],[6],[7].

In preeclampsia, bioactive peptides generated by adipose tissue, called adipokines, regulate metabolic, inflammatory, and vascular pathways [8,9]. Resistin and adiponectin are notable for their opposite biological effects. Endothelial activation, increased vascular stiffness, and increased production of inflammatory mediators including tumor necrosis factor-alpha and interleukin-6 are associated to resistin [10][11][12]. Resistin levels are often elevated in preeclampsia and correlate with illness severity, suggesting it may be a biomarker and mediator of vascular dysfunction [10,11].

By increasing endothelial nitric oxide generation and insulin sensitivity, adiponectin is anti-inflammatory and vasculoprotective [8,13]. It also reduces oxidative stress and vascular smooth muscle development. Research on adiponectin levels in preeclampsia ranges from raised to decreased, depending on gestational age, maternal BMI, and disease phenotype [14][15][16]. These contradictions may suggest preeclampsia's heterogeneity, which comprises pathophysiological endotypes with distinct metabolic and inflammatory features [4,17].

Finding reliable biomarkers for early diagnosis and illness severity prediction remains a clinical challenge. Modern diagnosis procedures rely on late-stage clinical characteristics, limiting early intervention [18]. Thus, biomarkers of core pathophysiological mechanisms such inflammation, angiogenic imbalance, and endothelial dysfunction are therapeutically relevant [19][20][21].

Biological relevance, stability in circulation, and ease of measurement make adipokines ideal candidates. In early prediction models, they may work with biochemical and angiogenic indicators such sFlt-1 and PlGF [22],[23]. Though its diagnostic and prognostic usefulness, especially for disease severity, is unclear and need further study.

This study measured resistin and adiponectin levels in preeclampsia, assessed their diagnostic and severity potential, and examined their correlations with clinical and biochemical data.

2. Materials and Methods

2.1 Study design and participants

The College of Medicine at Al-Nahrain University and the Department of Obstetrics and Gynecology collaborated to carry out this case-control study. All participants were asked to sign a written informed consent form. Of the 88 pregnant women who participated, 30 were assigned to the control group, 29 to moderate preeclampsia, and 29 to severe preeclampsia.

The American College of Obstetricians and Gynecologists used the following criteria to define preeclampsia: (ACOG., 2020) newly-discovered hypertension (≥140/90 mmHg) after 20 weeks of gestation along with either proteinuria (≥300 mg/24 h) or signs of end-organ involvement (such as thrombocytopenia, renal insufficiency, or hepatic dysfunction) [24]. Exclusion criteria included multiple gestation, chronic hypertension predating pregnancy, pregestational diabetes mellitus, renal disease, autoimmune disease, acute infection, and known chronic inflammatory disorders.

2.2 Sample collection and biochemical measurements

After a night without food or drink, 5 mL of blood was drawn from the veins, put into simple tubes, and spun at 3000 rpm for 10 minutes to get a clot. Aliquots of serum were divided and kept at -20°C until analysis.

We followed the manufacturer's directions to measure the serum adiponectin and resistin concentrations using an ELISA. The following enzymes were routinely tested on

automated analyzers: urea, creatinine, alanine aminotransferase (ALT), and aspartate aminotransferase (AST).

2.3 Statistical analysis

Visual examination of distributions and Shapiro-Wilk tests were used to determine normality. Mean ± SD is used to display continuous variables when they are regularly distributed, and median (IQR) is used when they are not.

For normally distributed variables, one-way ANOVA was employed for group comparisons, whereas non-parametric variables were tested using Kruskal-Wallis. Post-hoc pairwise comparisons used Tukey's HSD or Dunn's test as appropriate, with Bonferroni adjustment when needed (alpha = 0.0167). The Cohen's d was used to express the effect size.

Using Spearman correlation coefficients, we assessed the associations between analytes and clinical factors. Logistic regression models were fitted to evaluate predictors of preeclampsia severity, with odds ratios (OR) and 95% confidence intervals (CI) for disease presence and severity.

To measure the efficacy of the categorization process, ROC curves were employed. By comparing AUC values, we were able to use Youden's index to determine the best cutoffs. A two-tailed p-value < 0.05 was considered significant.

2.4. Ethical approval

The ethical approval was approved by the Institution Review Board of Collage of Medicine/ Al-Nahrain University/ Iraq (IRB/55, Number 20250594)

3. Results

3.1 Demographic and Clinical Characteristics

The data in Table 1 describe the participants' attributes. Although the age distributions of the groups were very similar, the measurements of blood pressure rose steadily from the control group to those with mild and severe illness. Severe disease was associated with elevated renal and hepatic indices when compared to controls ($p < 0.01$).

Table 1. Demographic and Clinical Characteristics of Study Groups

Parameter	Control (n=30)	Mild PE (n=29)	Severe PE (n=29)	p-value
Age (years)	26.0 (24.0-28.8)	34.0 (28.0-39.0)	33.0 (28.0-37.0)	<0.001*
GA (weeks)	33.51±3.98 (27.85-36.42.)	28.67±5.78 (25.71-35.14)	32.53±6.34 (30.00-36.42)	0.217
SBP (mmHg)	100 (100-110)	140 (140-150)	160 (160-170)	<0.001*
DBP (mmHg)	70 (70-70)	90 (90-100)	100 (90-110)	<0.001*
Blood urea (mg/dl)	14.0 (13.0-17.8)	16.0 (13.0-20.0)	20.0 (15.0-31.0)	0.007*
S. creatinine (mg/dl)	0.4 (0.3-0.5)	0.4 (0.3-0.5)	0.5 (0.4-0.6)	0.002*
Uric acid (mg/dl)	5.0 (4.6-5.1)	4.8 (3.9-5.9)	5.9 (5.4-6.6)	<0.001*

*Data are median (IQR). PE = preeclampsia; GA = gestational age; BMI = body mass index; SBP/DBP = systolic/diastolic blood pressure. *Significant by Kruskal-Wallis test.).*

3.2 Serum Adipokine Levels

Table 2 and picture 1 show that there was a graded increase in serum Adiponectin and resistin across all three groups. There was a distinct demarcation between severe illness and controls, and resistin rose sharply from controls to moderate to severe preeclampsia. Although there was less of a difference in magnitude and more variability, adiponectin also rose.

In controls, the resistin level was 8.55 ± 1.43 mg/L, in moderate preeclampsia it was 10.98 ± 1.06 mg/L, and in severe preeclampsia it was 14.16 ± 1.04 mg/L. Just like in controls, moderate preeclampsia had adiponectin levels of 10.93 ± 2.43 mg/L, and severe preeclampsia had levels of 16.83 ± 4.19 mg/L.

Table 2. Serum adiponectin and resistin levels by group

Marker	Control (n=30)	Mild PE (n=29)	Severe PE (n=29)	p-value
Resistin (mg/L)	8.55 ± 1.43	10.98 ± 1.06	14.16 ± 1.04	<0.001*
Adiponectin (mg/L)	5.05 ± 1.19	10.93 ± 2.43	16.83 ± 4.19	<0.001*

*Values are mean +/- SD. PE = preeclampsia. *Significant (ANOVA for resistin; Kruskal-Wallis for adiponectin).*

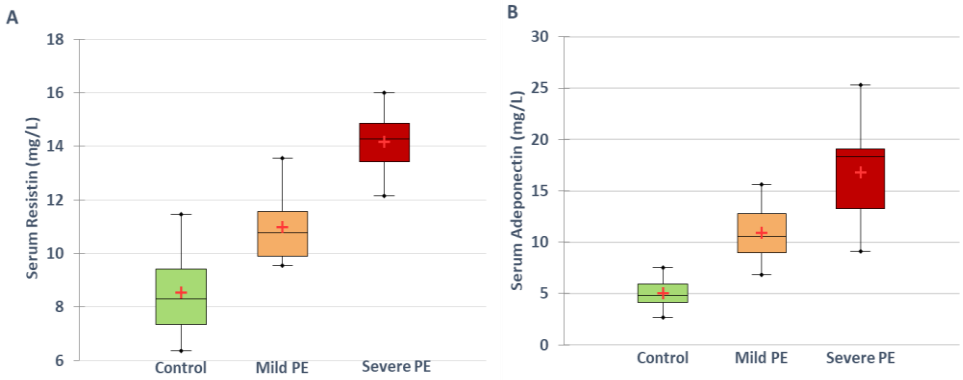

Figure 1. Serum resistin and Adiponectin concentrations across control, mild preeclampsia, and severe preeclampsia groups.

3.3 Correlation Analysis

Table 3 shows the results of the correlation study between the adipokines and the clinical factors in preeclampsia, which depend on the severity of the condition. In the combined preeclampsia group, resistin correlated positively with diastolic blood pressure (r=0.448, p=0.0004) and also with systolic blood pressure most strongly (r=0.816, p<0.0001), uric acid (r=0.49, p=0.0001), and gestational age (r=0.477, p=0.0002), while adiponectin correlated with systolic blood pressure (r=0.533, p<0.0001), uric acid (r=0.497, p=0.0001), creatinine (r=0.387, p=0.0027), gestational age (r=0.377, p=0.0035), and urea (r=0.288, p=0.0283) but not diastolic pressure (r=0.192, p=0.1498); importantly, adiponectin and resistin were positively interrelated in the combined cohort (r=0.577, p<0.0001), implying coupled adipokine disturbance. Adiponectin maintained its correlations with uric acid (r=0.612, p=0.0004), creatinine (r=0.441, p=0.0165), and gestational age (r=0.434, p=0.0187) in severe preeclampsia, although resistin only showed a correlation with uric acid (r=0.509, p=0.0048). Resistin had a weak link with gestational age (r=0.393, p=0.0351) and no other significant associations were found in mild preeclampsia; similarly, adiponectin did not show any substantial correlation with resistin (r=0.018, p=0.9271), in contrast to the combined group.

Table 3. Correlations between adipokines and clinical variables in PE patients

		Sever PE		Mild PE		All PE	
Parameter		Adiponectin (mg/L)	Resistin (mg/L)	Adiponectin (mg/L)	Resistin (mg/L)	Adiponectin (mg/L)	Resistin (mg/L)
DBP (mmHg)	r	0.215	0.324	-0.234	0.244	0.192	**0.448**
	p	0.2625	0.0862	0.2215	0.2018	0.1498	**0.0004**
SBP (mmH)	r	0.11	0.277	-0.308	0.212	**0.533**	**0.816**
	p	0.5715	0.1463	0.1036	0.2684	**<0.0001***	**<0.0001***
GA (Wks)	r	**0.434**	0.356	0.091	**0.393**	**0.377**	**0.477**
	p	**0.0187***	0.0584	0.639	**0.0351***	**0.0035***	**0.0002***
B. Urea (mg/dl)	r	0.179	-0.067	0.044	-0.087	**0.288**	0.193
	p	0.353	0.7279	0.8195	0.6528	**0.0283***	0.1458
S. Cr (mg/dl)	r	0.441	0.037	0.219	-0.29	**0.387**	0.191
	p	0.0165*	0.8481	0.2548	0.1266	**0.0027***	0.1504
Uric Acid (mg/dl)	r	**0.612**	**0.509**	0.182	0.123	**0.497**	**0.49**
	p	**0.0004***	**0.0048***	0.3439	0.5246	**0.0001***	**0.0001***
Adiponectin (mg/dl)	r		0.176		0.018		**0.577**
	p		0.3604		0.9271		**<0.0001**

Spearman correlation coefficients (r) with p-values

3.4 Diagnostic Performance

Classification performance for differentiating illness groups was evaluated using ROC analysis (Table 4) and (Figure 2). Several comparisons showed that resistin had higher AUC values than adiponectin; this was especially true when it came to detecting severe preeclampsia. While adiponectin could still be useful on a mixed panel, resistin offered cleaner separation in a practical sense.

Table 4. ROC performance of adiponectin and resistin for disease classification

Comparison	Marker	AUC (95% CI)	Cutoff	Sens %	Spec %
PE vs Control	Adiponectin	0.997 (0.987-1.000)	≥7.80	94.8	100.0
PE vs Control	Resistin	0.951 (0.908-0.994)	≥9.55	100.0	76.7
Severe vs Mild PE	Resistin	0.980 (0.942-1.000)	≥12.55	96.6	89.7
Severe vs Mild PE	Adiponectin	0.866 (0.771-0.962)	≥16.20	69.0	100.0

AUC = area under curve; CI = confidence interval. Cutoffs by Youden's index.

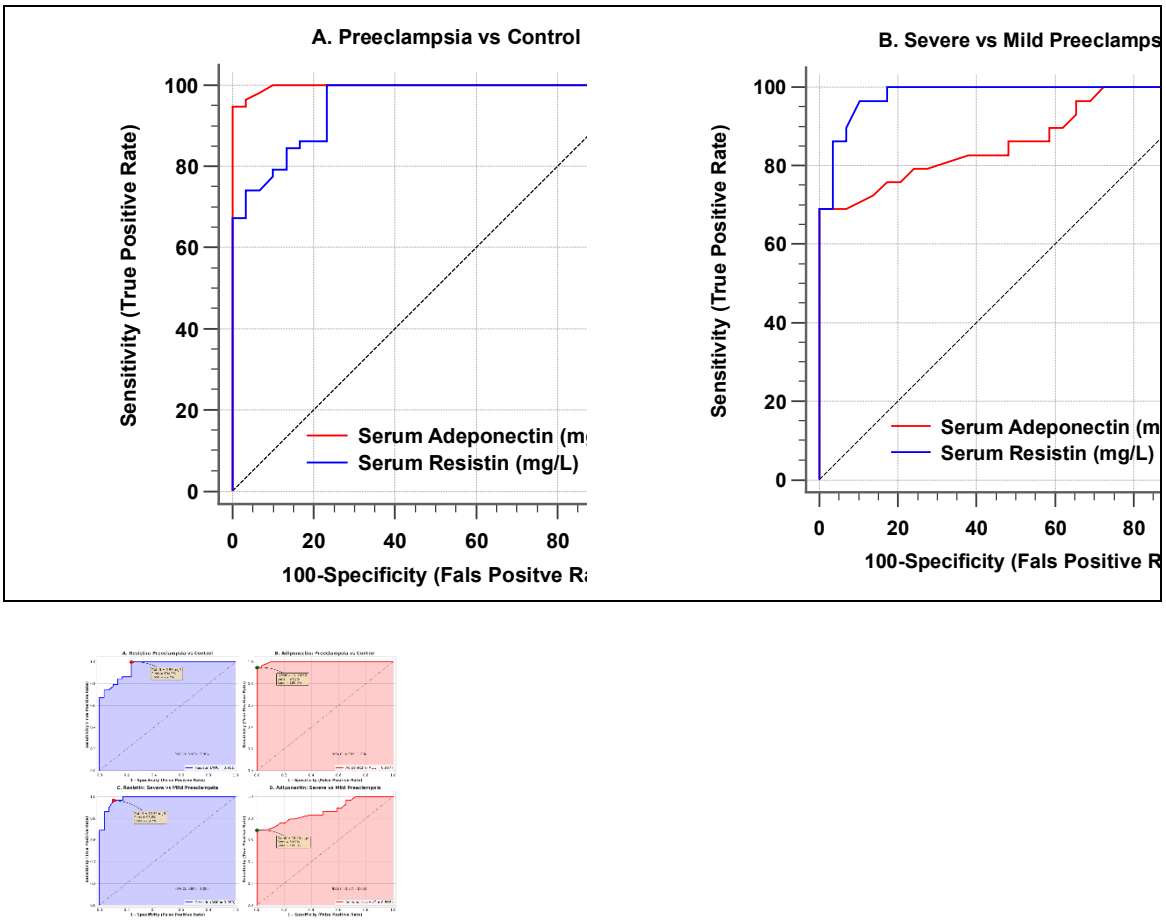

Figure 2. ROC curves showing diagnostic performance of adiponectin and resistin for classification of preeclampsia categories.

3.5 Logistic Regression Analysis

Both adipokines were found to be significant independent predictors of preeclampsia,

according to binary logistic regression analysis (Table 5). The risks of preeclampsia were 4.93 times higher for every 1 mg/L increase in resistin (95% CI: 2.31-10.54; $p < 0.001$). Once the variables were taken into account, resistin continued to be a strong predictor of disease severity, whereas adiponectin's independent contribution was reduced. This in no way rules out the possibility of adiponectin, but it does raise the possibility that its signal is either reduced or shared with other variables.

Table 5. Logistic regression models for prediction of preeclampsia severity

Model	Variable	OR	95% CI	p-value
PE vs Control	Resistin	4.93	2.31-10.54	<0.001*
PE vs Control	Adiponectin	28.40	1.25-642.70	0.035*
Severe vs Mild PE	Resistin	16.64	2.86-97.01	0.002*
Severe vs Mild PE	Adiponectin	1.59	1.27-2.00	<0.001*
Severity (Combined)	Resistin	10.54	2.21-50.36	0.003*
Severity (Combined)	Adiponectin	1.39	0.94-2.05	0.095

OR = odds ratio; CI = confidence interval.

4. Discussion

The present study showed in table 1, the maternal age for control, mild, and severe preeclampsia groups were 33.5, 34.0 and 33.0 respectively, and it has no significant difference between preeclampsia groups and control group (P=0.231). This result is agreed with the conclusion obtained from [25].

This study showed, the levels of urea for control, mild, and severe preeclampsia groups were 14.0, 16.0, and 20 (mg/dl) respectively, with significance differences, but still with the reference value, and for creatinine were 0.4, 0.4 and 0.6 (mg/dl) respectively, with no significance differences. This result is agreed with the conclusion obtained from John and Abraham [26], that creatinine considered as marker for kidney function, but it's not suitable for detect the early stage of kidney dysfunction which associated with PE, and disagreement with the study presented by Aleksandra et al, which may be contributed to the differences of gestational age used [27].

The results of uric acids that shown in table 1 for control, mild, and severe preeclampsia groups were 5.0, 5.3, and 5.9 (mg/dl) respectively, with significantly elevated in PE groups comparing with the control group (P<0.01) These values showed that high levels of uric acid were associated with increased risk of renal dysfunction which reported by [27].

The present study showed in table 2 and figure 1, demonstrates a significant and progressive increase in serum resistin levels across control, mild, and severe preeclampsia groups, with adiponectin showing a less discriminative elevation. Instead of metabolic alterations, inflammatory activation and endothelial dysfunction are related with preeclampsia severity [1,4].

Table 3 shows higher resistin levels in preeclampsia, consistent with previous research and meta-analyses [10,11]. Resistin increases pro-inflammatory cytokine production and decreases nitric oxide bioavailability, worsening endothelial dysfunction and increasing vascular resistance and hypertension [6,12]. This study's strong link between resistin and systolic blood pressure supports Banjac et al. (2021)'s claim that resistin measures endothelial damage [10].

The strong correlation between resistin and uric acid suggests a relationship to renal dysfunction and oxidative stress, both hallmarks of preeclampsia [5,28]. These findings suggest that resistin may be a biomarker and a modulator of disease development through inflammation-driven vascular damage [11].

This study found a positive link between adiponectin and kidney markers, suggesting metabolic stress rather than pathological involvement. Studies of pregnancy problems' adipokine profiles found similar results [9].

However, preeclampsia adiponectin literature is conflicting. Metabolic phenotype may be involved in decreasing levels, especially in obese or insulin-resistant populations [14,15]. Additionally, maternal BMI and gestational age are significant confounders of adiponectin levels [16].

As shown in table 4 and figure 2, resistin's high sensitivity and specificity in ROC analysis separate severe from moderate preeclampsia, supporting previous results [10,11].

Logistic regression analysis showed resistin as an independent predictor of illness severity, suggesting its clinical use in risk stratification. In contrast, adiponectin had higher levels but decreased discriminatory power. In numerous investigations, this counterintuitive rise may be a compensatory response to systemic inflammation and endothelial dysfunction [8,13].

Adiponectin has anti-inflammatory and vasodilatory properties, therefore its rise may be an adaptive response to vascular injury [17].

The increase and association of resistin and adiponectin in preeclampsia suggest a coordinated dysregulation of adipokine signaling due to metabolic and inflammatory pathways [8]. However, stronger connections for resistin suggest a greater role in disease pathogenesis.

This study suggests that resistin may be a valuable biomarker for preeclampsia severity. A single biomarker is not enough for clinical decision-making. A multimarker strategy including angiogenic factors such sFlt-1 and PlGF and clinical parameters may improve diagnostic accuracy [4,19,21].

Logistic regression analysis (Table 5) shows that resistin strongly predicts preeclampsia presence and severity, with a high odds ratio (OR = 4.93, $p < 0.001$), showing a significant risk increase with higher levels. This supports its role as a clinically meaningful biomarker for inflammation and endothelial dysfunction, which are key to disease development [10,11]. Resistin's relevance after correction suggests that it may directly contribute to disease mechanisms [6,12].

Recent study and meta-analyses show greater resistin levels in preeclampsia and a robust connection with sickness severity [10,11]. Resistin's high severity model odds ratio suggests vascular injury progression and intensity.

In contrast, adiponectin predicts poorly. Although significant in some comparisons, its influence reduces after covariate correction, suggesting it is a compensatory or secondary biomarker rather than harmful [8,13]. Its anti-inflammatory qualities and flexibility to BMI and metabolic status suggest this [14-16].

Resistin's persistence in multivariate models supports its independent diagnostic significance, while adiponectin's decline suggests overlap with other factors. Resistin may be useful for risk categorization and severity assessment, although it would perform best in a multimarker panel with angiogenic markers [4,19].

Despite these findings, several restrictions exist. The case-control approach and small sample size may hinder causal inference and generalizability [29]. Confounding factors like as BMI, parity, and gestational age can impact adipokine levels [16]. One-time-point data also hamper biomarker temporal analysis [22].

To evaluate adipokines' predictive value during pregnancy, longitudinal research with larger populations are needed. Adding adipokines to biomarkers may improve early diagnosis and risk classification [20,23].

5. Conclusion

In determining the severity of preeclampsia, serum resistin rises with each grade and outperforms adiponectin as a diagnostic tool. Adiponectin changes are less discriminating. Resistin may be a useful clinical biomarker, although prospective longitudinal sampling is recommended.

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