Jinekoloji - Obstetrik ve Neonatoloji Tıp Dergisi
The Journal of Gynecology-Obstetrics and Neonatology

ÖZGÜN ARAŞTIRMA / ORIGINAL ARTICLE

DOI: 10.38136/jgon.1643530

The Relationship Between Omentin-1 Levels and Disease Severity in Pregnant Women Diagnosed with Preeclampsia

Preeklampsi Tanısı Alan Gebe Kadınlarda Omentin-1 Düzeyleri ile Hastalık Şiddeti Arasındaki İlişki

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ABSTRACT

Aim: The aim of this study is to compare serum Omentin-1 levels in pregnant women diagnosed with preeclampsia with those in normal pregnancies, and to investigate the role of this molecule in the pathogenesis of the condition.

Materials and Methods: This study included voluntary pregnant women who applied to the Gynecology and Obstetrics Clinic of a tertiary hospital, between June 2018 and September 2018. The study group consisted of 90 pregnant women diagnosed with preeclampsia, while the control group included 90 pregnant women with no complications during follow-up. The groups were compared in terms of Omentin-1 levels.

Results: A total of 180 pregnant women, aged between 18 and 40, were evaluated. The mean age was 28.35 ± 6.13 years (29.13 ± 6.25 in the patient group and 27.58 ± 6.02 in the control group). The mean Omentin-1 level was 63.10 Ng/mL in the preeclampsia group and 64.85 Ng/mL in the control group. There were no significant differences in Omentin-1 levels between the groups (p>0.005).

Conclusion: When comparing the serum Omentin-1 levels of the 90 pregnant women diagnosed with preeclampsia to those of the 90 normal pregnant women, no statistically significant differences were observed between the groups (p>0.005).

Keywords: Hypertension, omentin-1, preeclampsia

ÖZ

Amaç: Bu çalışmanın amacı preeklampsi tanısı almış gebe kadınlarda serum Omentin-1 düzeylerini normal gebeliklerdekilerle karşılaştırmak ve bu molekülün hastalığın patogenezindeki rolünü araştırmaktır.

Gereç ve Yöntemler: Bu çalışmaya Haziran 2018 ile Eylül 2018 tarihleri arasında üçüncü basamak bir hastanenin Kadın Hastalıkları ve Doğum Kliniğine başvuran gönüllü gebeler dahil edildi. Çalışma grubunu preeklampsi tanısı almış 90 gebe kadın, kontrol grubunu ise takip sırasında herhangi bir komplikasyon gelişmeyen 90 gebe kadın oluşturdu. Gruplar Omentin-1 düzeyleri açısından karşılaştırıldı.

Bulgular: 18-40 yaş aralığında toplam 180 gebe kadın değerlendirildi. Ortalama yaş 28,35 ± 6,13 yıldı (hasta grubunda 29,13 ± 6,25 ve kontrol grubunda 27,58 ± 6,02). Preeklampsi grubunda ortalama Omentin-1 düzeyi 63,10 Ng/mL ve kontrol grubunda 64,85 Ng/mL idi. Gruplar arasında Omentin-1 düzeylerinde anlamlı bir fark yoktu (p>0,005).

Sonuç: Preeklampsi tanısı konulan 90 gebe kadının serum Omentin-1 düzeyleri 90 normal gebe kadının düzeyleriyle karşılaştırıldığında, gruplar arasında istatistiksel olarak anlamlı bir fark gözlenmedi (p>0,005).

Anahtar Kelimeler: Hipertansiyon, omentin-1, preeklampsi

Cite as: Özdemir Bilginer H, Zorlu U, Durmaz G, Çift T. The Relationship Between Omentin-1 Levels and Disease Severity in Pregnant Women Diagnosed with Preeclampsia. Jinekoloji-Obstetrik ve Neonatoloji Tıp Dergisi 2026;23(1):34–40.

Geliş/Received: 21.02.2025 • **Kabul/Accepted:** 18.04.2025

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Çevrimiçi Erişim/Available online at: https://dergipark.org.tr/tr/pub/jgon

INTRODUCTION

Preeclampsia is a pregnancy complication that has significant negative impacts on both the fetus and the mother, resulting in high rates of illness and death. It affects about 4.6% of all pregnancies globally (1, 2). This condition is defined by the sudden development of high blood pressure (>140/90 mm Hg) and the presence of excess protein in the urine (>0.3 g) after the 20th week of pregnancy (3).

Despite an incomplete understanding of the exact cause of preeclampsia during pregnancy, there are various clinical factors that contribute to its development. These risk factors encompass primiparity, a previous occurrence of preeclampsia during pregnancy, chronic hypertension, chronic kidney disease, a history of thrombophilia, multiple pregnancies, in vitro fertilization, a family history of preeclampsia, diabetes, obesity, systemic lupus erythematosus, and advanced maternal age (over 40 years old) (4).

Given that the placenta is the sole recognized remedy for preeclampsia, it indicates that the placenta has a crucial function in the development of preeclampsia. The earliest phases in the development of preeclampsia are characterized by inadequate invasion of placental trophoblasts, defective trophoblast invasion, and insufficient remodeling of maternal spiral arteries, leading to placental ischemia and hypoxia (5). However, the occurrence of placental ischemia by itself is not enough to fully account for the clinical symptoms observed in cases of preeclampsia. Additional molecular pathways that contribute to the development of preeclampsia include changes in the balance of angiogenic factors, widespread inflammation throughout the body, and disruption of the renin-angiotensin system. The correlations between these mechanisms remain incompletely comprehended (6).

Omentin is a newly identified adipocytokine that was first described in the intestinal Paneth cells. It is secreted from visceral adipose tissue. It has two homologous isoforms: Omentin-1 and Omentin-2. Omentin-1 is main the predominant form in the bloodstream (7).

Metabolic disorders such as elevated blood pressure, increased waist circumference, dyslipidemia, and glucose intolerance have been associated with low levels of circulating Omentin-1 (8). Omentin has been found to possess preventive characteristics against metabolic syndrome, mostly attributed to its anti-inflammatory, anti-atherogenic, anti-cardiovascular, and anti-diabetic activities (9). Moreover, studies have demonstrated that Omentin promotes the widening of blood vessels, a process known as vasodilation, and that there is an inverse correlation between Omentin levels and systolic blood pressure (10). The secretion of circulating Omentin-1 has been shown to be lower in obese patients. Furthermore, the negative correlation between Omentin levels and circulating leptin levels, alongside the lower serum concentration of Omentin in obese patients, suggests that leptin regulates Omentin levels (9).

In individuals with chronic artery disease, Omentin has been found to be negatively correlated with inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin (IL)-6. This negative correlation suggests that Omentin exerts an inhibitory effect on inflammation (11).

Given that inflammation is known to play a role in the development of preeclampsia, it is plausible that Omentin could also be influential in its pathogenesis. In this study, we aimed to compare the serum Omentin-1 concentrations between normal pregnant women and those with preeclampsia, and to investigate whether the severity of preeclampsia is associated with Omentin-1 concentrations.

MATERIALS AND METHOD

The study comprised 180 pregnant women who intentionally applied the Obstetrics and Gynecology Clinic maternity unit at the University of Health Sciences Bursa High Specialization Training and Research Hospital from July to September 2018. The study group consisted of 90 patients who were diagnosed with preeclampsia, a condition characterized by high blood pressure during pregnancy. The control group, on the other hand, included 90 pregnant women who did not have high blood pressure during their pregnancy monitoring and were considered normotensive. The sample size was determined based on a review of previous studies investigating omentin levels in preeclamptic patients. Studies that reported statistically significant differences were considered, and their sample sizes were taken into account. Accordingly, to ensure adequate statistical power, the study included 90 patients diagnosed with preeclampsia and a control group of 90 healthy pregnant women.

The study started on June 20, 2018, after receiving approval from the Clinical Research Ethics Committee of the University of Health Sciences Bursa High Specialization Training and Research Hospital, with approval number 2011-KAEK-25 2018/06-35. Participants were notified about the study and given an informed consent form to review and sign.

The standards outlined in the 2013 European Society of Hypertension (ESH) - uropean Society of Cardiology (ESC) standards for the management of arterial hypertension, jointly issued by the ESH and the European Society of Cardiology (ESC), were adhered to in order to ensure precise blood pressure monitoring. The clinic utilized the usual measurement technique to assess the blood pressure of each participant. The blood pressure was measured during a 5-minute

period of rest while sitting, with the evaluation of the brachial artery blood pressure at the level of the heart. Measurements were obtained by a proficient healthcare team member utilizing a suitably proportioned blood pressure monitor.

The study applied the diagnostic criteria for preeclampsia as defined in the 2013 recommendations released by the American College of Obstetricians and Gynecologist.

The study excluded patients who were under the age of 18 and had additional medical disorders such as type 1 and type 2 diabetes, gestational diabetes, hypothyroidism, hyperthyroidism, multiple pregnancies, or pregnancies below 20 weeks. A control group consisting of healthy pregnant women who had the same gestational weeks and background information as the patient group was formed. Obstetric evaluations and fetal growth assessments were performed using the medical histories and ultrasound measures of all pregnant mothers.

Maternal blood samples were obtained from all preeclamptic patients who visited our clinic after reaching 20 weeks of pregnancy, precisely at the moment of diagnosis. Blood samples were collected from healthy pregnant women who were at a comparable stage of pregnancy to the patient group, before to the start of any treatment. The blood samples were subjected to centrifugation at a speed of 2000 revolutions per minute for a duration of 20 minutes in order to obtain serum. The obtained serum were subsequently stored at a temperature of -80°C.

Statistical analyses were performed using the SPSS 22.0 (Statistical Programme for Social Sciences) software. Categorical variables were presented as frequencies and percentages. For numerical data, normally distributed variables were expressed as mean and standard deviation, while non-normally distributed variables were presented with median, range, mean, and standard deviation. The Kolmogorov-Smirnov test was used to assess the normality of the data distribution. For data comparison, the chi-square test was used for categorical variables. In the comparison of numerical data, One-Way ANOVA was applied for normally distributed variables, whereas the Kruskal-Wallis test was used for non-normally distributed variables. The relationship between numerical variables was evaluated using Spearman correlation analysis. All statistical analyses were performed within a 95% confidence interval, with a significance level of $p<0.05$.

RESULTS

A total of 180 pregnant women participated in the study, of whom 90 (50.0%) were normotensive while 55 (30.6%) had preeclampsia and 35 (19.4%) had preeclampsia with severe features. Demographic characteristics and blood pressure frequencies and percentages were shown in Table 1.

There were significant differences in terms of gestational age, HCT, BUN, creatinine, and proteinuria values according to the presence of preeclampsia in the pregnant women included in the study. Gestational age showed statistical differences in all groups (Table 2).

When evaluating the groups in terms of HCT value, low HCT value in mild preeclamptic pregnant women compared to normotensive and severe preeclamptic pregnant women was statistically significant (p: 0.016). In severe preeclamptic pregnant women, the BUN value was found to be higher compared to normotensive and

Table 1. Comparison of the study participants' blood pressure measures and demographic data based on their preeclampsia status

	Normotensive	Preeclampsia	Preeclampsia with severe	P
Age (year) *	27.58±6.02	29.00±6.05	29.34±6.63	0.232
Height (cm)*	161.34±5.30	161.00±5.63	159.23±5.80	0.152
Weight (kg)*	78.06±14.04	84.78±16.95	82.20±13.37	**0.028** ^{P1}
BMI (kg/m²)*	29.96±5.18	32.64±5.80	32.50±5.50	**0.006** ^{P1}
Systolic blood pressure (mm/Hg)$^{\delta}$	110 (90-140)	150 (140-160)	160 (150-180)	**<0.001** ^{p1,p2,p3}
Diastolic blood pressure (mmHg)$^{\delta}$	70 (50-90)	90.00 (70-110)	110 (80-130)	**<0.001** ^{p1,p2,p3}

*The mean ($\bar{X}$) ± standard deviation (SD) was used to analyze data with a normally distributed distribution. $^{\delta}$When analyzing data that did not exhibit a normal distribution, the median (range) was used. ^{P1}:Comparing pregnant women with mild preeclampsia to normotensive women, ^{p2}: Comparing pregnant women with severe preeclampsia to normotensive women, ^{P3}: Comparing pregnant women with mild preeclampsia to severe women
BMI: Body Mass Index

Table 2. An analysis compared the blood results of mild and severe preeclamptic pregnant women with normotensive women

	Normotensive		Preeclampsia		Preeclampsia with severe		P
Gestational age$^{\delta}$	38	(29-41)	37.00	(28-41)	35	(28-39)	**<0.001**^{p1,p2,p3}
WBC (x10^3/mm^3)$^{\delta}$	9.8	(5.6-22.6)	10.6	(7.0-20.4)	10.6	(6.9-25.6)	0.295
HB$^{\delta}$	11.5	(6.5-14.8)	11.6	(8.5-14.6)	11.9	(9.3-16.4)	0.080
PLT*	231.09	±67.98	232.75	±66.58	237.11	±70.33	0.906
RDW$^{\delta}$	14.9	(12.1-115.7)	15.3	(12.5-25.0)	15.1	(13.1-24.8)	0.615
PDW$^{\delta}$	17.3	(15.2-19.5)	17.4	(16.1-92.1)	17.4	(16.2-19.0)	0.979
HCT^{v}	35.25	(21.4-240.0)	34.0	(17.6-42.0)	35.4	(28.7-48.2)	**0.016**^{p1,p3}
MCV^{v}	85.2	(10.7-102.0)	83.9	(17.2-99.8)	83.6	(70.0-92.0)	0.318
BUN$^{\delta}$	7.0	(3.2-12.1)	8.8	(3.74-1028.0)	11.6	(6.0-71.8)	**<0.001**^{p2,p3}
Creatinine(mg/dL)$^{\delta}$	0.63	(0.46-58.00)	0.67	(0.43-73.00)	0.74	(0.54-74.00)	**<0.001** ^{p2}
AST (U/L)$^{\delta}$	20.0	(11.0-35.0)	21.0	(10.0-71.0)	22.0	(13.0-179.0)	0.153
ALT (U/L)$^{\delta}$	10.0	(4.0-29.0)	11.0	(3.0-46.00)	12.0	(4.0-258.0)	0.080
Proteinuria$^{\delta}$	0	(0.0-1.0)	1.0	(0.0-3.0)	3.0	(0.0-3.0)	**<0.001**^{p1,p2}
Omentin-1$^{\delta}$	63.1	(15.5-366.2)	66.3	(20.3-274.5)	63.2	(21.9-310.4)	0.732

*The mean (X) ± standard deviation (SD) was used to analyze data with a normally distributed distribution. $^{\delta}$When analyzing data that did not exhibit a normal distribution, the median (range) was used. ^{P1}: Comparing pregnant women with mild preeclampsia to normotensive women, ^{P2}: Comparing pregnant women with severe preeclampsia to normotensive women, ^{P3}: Comparing pregnant women with mild preeclampsia to severe women

mild preeclamptic pregnant women. This elevation was statistically significant (p: <0.001). When evaluating the groups in terms of creatinine value and presence of proteinuria, in severe preeclamptic pregnant women, the creatinine value and presence and amount of proteinuria were statistically significantly higher compared to normotensive pregnant women (p: <0.001). When evaluating the groups in terms of Omentin-1, no statistically significant difference was found among the groups (Table 2).

When groups were evaluated separately, there was no statistically significant relationship between Omentin-1 and gestational age, WBC, HB, PLT, RDW, PDW, HCT, BUN, Creatinine, AST, ALT, and proteinuria (Table 3).

A low-grade positive relationship was found between Omentin-1 and AST in normotensive pregnant women. In mild preeclamptic pregnant women, there is a low-grade negative relationship between Omentin-1 and creatinine. In severe preeclamptic pregnant women, there is a moderate positive relationship between Omentin-1 and creatinine, and a moderate negative relationship between Omentin-1 and ALT (Table 3).

DISCUSSION

Preeclampsia, a pregnancy complication, is defined by the sudden start of high blood pressure and the presence of protein in the urine. It is a significant contributor to both mother and fetal death and morbidity (1). Placenta removal is the sole recognized treatment for preeclampsia, indicating that the placenta has a substantial involvement in the development of preeclampsia. The first stage in the development of preeclampsia is believed to be the insufficient invasion of placental trophoblasts, poor remodeling of maternal spiral arteries, and hypoxia caused by placental ischemia.

Preeclampsia is linked to substantial cardiovascular illness and death. It is crucial to detect pregnancies that are at risk of preeclampsia early on, in order to initiate preventive treatment promptly. Biomarkers have been widely accepted for early disease diagnosis and therapy monitoring across several disease processes. Adipokines are cell signaling proteins that are synthesized by adipocytes in adipose tissue and have an impact on the metabolism of glucose and lipids. The substances encompass TNF-α, interleukin-6, leptin, resistin, visfatin, and chemerin (12-16). They have significant impacts on the progression of obesity, metabolic syndrome, diabetes, and cardiovascular disease esearch on preeclampsia has demonstrated a correlation between the occurrence of preeclampsia and the levels of IL-6, (12) chemerin, (13) adiponectin, (14) visfatin (15) and leptin (16) in the bloodstream.

The disparity between angiogenic factors and antiangiogenic factors in the development of preeclampsia has garnered significant interest over the last 10 years (17). The human placenta produces a range of angiogenic agents. The primary factor of utmost significance is vascular endothelial growth factor (VEGF). Omentin-1

Table 3. Analysis of the Spearman correlation between Omentin-1 and other study group variables

		OMENTİN-1			
		Normotensive	**Preeclampsia (total)**	**Mild Preeclampsia**	**Preeclampsia with severe**
Age (year)	r	0.119	-0.017	-0.077	0.042
	p	0.262	0.875	0.579	0.811
BMI (kg/m²)	r	-0.204	0.174	0.228	0.109
	p	0.054	0.101	0.094	0.534
Sistolic blood pressure (mmHg)	r	-0.014	-0.082	-0.033	-0.232
	p	0.892	0.443	0.809	0.181
Diastolic blood pressure (mmHg)	r	-0.067	-0.130	-0.215	-0.108
	p	0.527	0.223	0.115	0.535
Gravida	r	0.037	0.057	0.156	-0.103
	p	0.727	0.591	0.255	0.557
Parity	r	0.125	-0.012	0.092	-0.172
	p	0.241	0.911	0.502	0.324
Abortion	r	-0.018	0.039	0.071	0.004
	p	0.867	0.716	0.604	0.984
WBC	r	-0.045	-0.201	-0.184	-0.300
	p	0.675	0.057	0.179	0.080
HGB	r	0.005	-0.075	-0.167	0.096
	p	0.963	0.482	0.223	0.584
PLT	r	0.085	0.089	0.066	0.155
	p	0.424	0.406	0.630	0.374
RDW	r	-0.111	-0.069	-0.084	-0.104
	p	0.298	0.517	0.540	0.550
PDW	r	0.031	-0.195	-0.142	-0.200
	p	0.770	0.066	0.300	0.250
HCT	r	-0.068	-0.146	-0.212	-0.005
	p	0.522	0.170	0.120	0.975
MCV	r	0.154	-0.046	0.047	-0.169
	p	0.147	0.669	0.734	0.332
BUN	r	-0.042	0.001	-0.096	0.160
	p	0.696	0.991	0.484	0.360
CR	r	-0.091	0.014	**-0.270***	**0.348***
	p	0.393	0.895	**0.046**	**0.040**
AST	r	**0.215***	-0.044	0.027	-0.160
	p	**0.041**	0.679	0.843	0.358
ALT	r	0.055	-0.176	0.004	**-0.416***
	p	0.606	0.096	0.976	**0.013**
Proteinuria	r	-0.077	0.083	-0.043	0.276
	p	0.468	0.439	0.757	0.108

WBC: White Blood Count, HGM:Hemoglobine, PLT:Platelet, RDW: Red cell distribution width, PDW: Platelet distribution width, HCT: Hematocrit, MCV: mean corpuscular volume, BUN: Blood urea nitrogen,CR: Creatinine, AST: Aspartat Aminotransferase ALT: Alanin Aminotransferase, r: Correlation Coefficient.

has the potential to greatly reduce the migration of endothelial cells and the formation of new blood vessels in human microvascular endothelial cells, which is stimulated by VEGF (18). This discovery indicates that Omentin-1 may function as a regulator that inhibits the growth of new blood vessels and may play a role in the development of preeclampsia by exerting antiangiogenic actions. Omentin has demonstrated its role as an anti-inflammatory agent and has been found to suppress TNF-induced vascular inflammation in human endothelial cells, as observed in a particular investigation (19). Another study found that Omentin blocked the expression of vascular cell adhesion molecule-1 caused by TNF-α. This was achieved by inhibiting the activation of p38 and JNK, at least partially through the suppression of superoxide formation (20). Furthermore, studies have demonstrated a negative correlation between serum Omentin-1 concentrations and the levels of inflammatory molecules such as TNF-α, IL-6, and CRP (21, 22). Collectively, these findings

indicate that Omentin may have a role in the anti-inflammatory process. Considering the potential involvement of inflammation in preeclampsia, it is possible that Omentin-1 plays a role in the development of preeclampsia by inhibiting the inflammatory pathway.

Research indicates that Omentin-1 is produced as a result of inflammation and is involved in vasodilation through multiple mechanisms. This suggests that Omentin-1 may contribute to the development of preeclampsia. A study found that decreased levels of circulating Omentin-1 were linked to the development of specific problems, including type 2 diabetes, diabetic vascular disease, cardiomyopathy, and retinopathy. The observed effect is believed to be associated with the inhibition of the NF-KB pathway by Omentin-1, as well as the activation of the Akt and AMPK pathways (23).

A recent study found that individuals with low levels of Omentin-1 in their bloodstream have a higher risk of experiencing ischemic stroke. Concurrently, increased levels of Omentin-1 have been discovered to provide protection against cerebral ischemia (24).

Liu and colleagues conducted a study which revealed a substantial decrease in serum Omentin-1 concentrations in patients diagnosed with preeclampsia. Furthermore, they provided evidence that the quantity of Omentin-1 is inversely correlated with the intensity of preeclampsia (25).

While our investigation revealed elevated levels of Omentin-1 in preeclamptic pregnant women compared to normotensive pregnant women, the difference was not statistically significant. In addition, we observed markedly elevated levels of blood urea nitrogen (BUN), creatinine, alanine aminotransferase (ALT), and proteinuria in pregnant women with preeclampsia as compared to pregnant women without hypertension.

This study aimed to illustrate the probable involvement of Omentin-1 in the development of preeclampsia. The levels of serum Omentin-1 were examined in a prospective manner in both the patient and control groups. The study's short duration and the low number of patients included constrained the study's statistical power. In addition, the study failed to sufficiently distinguish between distinct subgroups of preeclampsia, and the comparisons of blood Omentin-1 concentrations in cases of early or late onset preeclampsia were inadequate. Further investigation is required to clarify the involvement of Omentin-1 in hypertensive disorders during pregnancy. This molecule, believed to contribute to the development of conditions like obesity, coronary artery disease, and diabetes, may also play a role in gestational hypertensive disorders. Additional research on Omentin-1 and hypertensive disorders of pregnancy may help identify the abnormal structure of blood vessels in the placenta and the resulting lack of blood supply as crucial factors in the development of these conditions. This could lead to more sophisticated investigations and treatments for predicting and preventing gestational hypertensive disorders.

Competing interests: There are no conflicts of interest among the authors.

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