SJIF Impact Factor: 6.711

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

ISSN: 2457-0400
Volume: 9.
Issue: 9
Page N. 173-178
Year: 2025

Original Article www.wjahr.com

THE ASSOCIATION BETWEEN SERUM ADIPONECTIN LEVEL & PRE-ECLAMPSIA

***[1]Nadia Hasan Hussein and [2]Maha Mohammed Al-Bayatti**

[1]Al-Anbar Health Directorate, Al-Anbar, Iraq.
[2]Al-Yarmouk Teaching Hospital, Baghdad, Iraq.

Article Received date: 07 July 2025 **Article Revised date:** 26 July 2025 **Article Accepted date:** 17 August 2025

***Corresponding Author: Nadia Hasan Hussein**
Al-Anbar Health Directorate, Al-Anbar, Iraq.

ABSTRACT

Background: Adiponectin, a 244-amino acid protein, exhibits potent insulin-sensitizing, anti-inflammatory, and anti-atherogenic properties. However, existing literature presents conflicting evidence regarding its role in pre-eclampsia. **Objective:** To investigate the association between serum adiponectin levels and pre-eclampsia. **Design:** Case-control study. **Setting:** Al-Yarmouk Teaching Hospital. **Methods:** A total of 65 pregnant women with singleton viable fetuses in the third trimester were enrolled and categorized into three groups: 30 women with normal pregnancies, 20 women with mild pre-eclampsia, and 15 women with severe pre-eclampsia. Serum adiponectin levels were measured using the enzyme-linked immunosorbent assay (ELISA) method. **Results:** Serum adiponectin levels were significantly elevated in women with pre-eclampsia compared to those with normal pregnancies. The elevation was more pronounced in cases of severe pre-eclampsia. **Conclusion:** Adiponectin levels were markedly increased in pre-eclamptic patients, particularly in severe cases. A negative correlation was observed between adiponectin levels and blood pressure. However, no significant correlation was found between adiponectin levels and body mass index (BMI).

KEYWORDS: The association, serum, adiponectin, pre-eclampsia.

INTRODUCTION

Pre-eclampsia is a multisystem disorder of pregnancy characterized by the new onset of hypertension and proteinuria after 20 weeks of gestation in previously normotensive women.[1] Although proteinuria remains a key diagnostic criterion, pre-eclampsia is now recognized as a systemic syndrome involving endothelial dysfunction and inflammatory responses.[2] The prevalence of pre-eclampsia varies across populations, ranging from 2.2% to 10% depending on maternal characteristics and diagnostic criteria used.[2] Nulliparous women, particularly those of certain ethnicities, are at greater risk, while multiparity is generally protective.[3] Importantly, pre-eclampsia is the second leading cause of maternal death worldwide, contributing to 16–18% of maternal mortalities.[4] Although early diagnosis and management can significantly reduce maternal risk[5], progression to complications such as HELLP syndrome and eclampsia may increase maternal mortality to 24%.[6] Perinatal morbidity and mortality also rise substantially in severe pre-eclampsia, driven by prematurity, placental abruption, and fetal growth restriction.[7] Pathophysiologically, pre-eclampsia is believed to originate from abnormal placental implantation, leading to impaired spiral artery remodeling and placental ischemia.[8] This hypoxic environment results in the release of vasoactive substances into the maternal circulation, including soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng), which contribute to endothelial damage and clinical symptoms.[8] These antiangiogenic factors reduce circulating levels of vascular endothelial growth factor (VEGF) and placental growth factor (PGF), which are essential for normal endothelial function and placental development.[11] Elevated levels of sFlt-1 and sEng can be detected in the maternal serum weeks before the onset of clinical signs.[8] Adiponectin, a 244-amino acid peptide hormone secreted exclusively by adipocytes, plays a key role in modulating insulin sensitivity, inflammation, and vascular health.[9] In contrast to other adipokines, adiponectin levels are paradoxically reduced in obesity, possibly due to the inhibitory effects of inflammatory cytokines on its gene transcription.[10] It exerts its effects via two receptors, AdipoR1 and AdipoR2, which activate AMP-activated protein kinase (AMPK) pathways involved in glucose and lipid metabolism.[11] In addition to its well-established cardioprotective and antiatherosclerotic properties[12], adiponectin modulates

endothelial function, inhibits vascular inflammation, and promotes nitric oxide production.[13] Emerging evidence has shown that adiponectin may be implicated in the pathophysiology of pre-eclampsia through its effects on endothelial cells and metabolic regulation, though its precise role remains controversial.[14] Thus, this study aims to investigate the relationship between serum adiponectin levels and pre-eclampsia, with consideration of disease severity and clinical implications.

METHOD

This case-control study was conducted in the Obstetrics and Gynecology Department at Al-Yarmook Teaching Hospital, Baghdad, from June to December 2013. The Iraqi Council for Medical Specialisation (Scientific Committee of Obstetrics and Gynaecology) granted ethical approval. After gaining informed consent, 65 pregnant women with singleton viable foetuses in their third trimester (gestational age 28–40 weeks) were enlisted. Three groups of participants were formed: Twenty women in Group A had mild pre-eclampsia (systolic blood pressure <150 mmHg, diastolic <100 mmHg with proteinuria ± oedema), fifteen in Group B had severe pre-eclampsia (systolic ≥160 mmHg, diastolic ≥110 mmHg with proteinuria ± oedema), and thirty healthy pregnant women in Group C (control group) had uncomplicated pregnancies. Every participant received comprehensive obstetric, general, and history-taking examinations. A mercury sphygmomanometer was used to measure blood pressure while the subject was seated. Korotkoff phase V was used to determine the diastolic pressure, and the cuff was placed at heart level. Proteinuria was measured using 24-hour urine collection or midstream urine samples using dipstick testing; ≥300 mg/24 hours was deemed significant. In order to compute BMI (kg/m2) using NIH criteria, anthropometric measurements, such as height and weight, were taken at the time of sampling. Multiple pregnancies, diabetes mellitus, smoking, chronic hypertension, heart or kidney disease, and inflammatory or infectious diseases were among the exclusion criteria. Each participant had a 5 mL venous blood sample taken, processed, and kept at -20°C until analysis. A quantitative sandwich ELISA method (DEMEDITEC ELISA DEE009, Germany) was used in the hospital's teaching laboratory's Immunology Unit to measure the serum adiponectin concentration. SPSS version 20 was used for the statistical analysis. The mean, standard deviation, and range were examples of descriptive statistics. Pearson correlation, Fisher's exact test, and chi-square were used to evaluate group differences. Statistical significance was defined as a p-value of less than 0.05. The diagnostic performance and ideal cut-off value of serum adiponectin were ascertained by applying sensitivity, specificity, and ROC analysis.

RESULTS

This study included 65 pregnant women in their third trimester, divided into three groups: 20 with mild pre-eclampsia, 15 with severe pre-eclampsia, and 30 healthy pregnant women as controls. The groups were compared based on maternal age, blood pressure, gravida, parity, history of abortion, gestational age, and body mass index (BMI).

- **Maternal age**: No statistically significant difference among groups (P = 0.097).
- **Gravidity and parity**: Comparable across all groups with no significant differences (P = 0.778 and P = 0.955, respectively).
- **History of abortion**: Similar distribution among groups; not statistically significant (P = 0.772).
- **Gestational age**: Slightly lower in the severe PE group, but differences were not statistically significant (P = 0.103).
- **BMI**: Higher in pre-eclamptic groups compared to controls, but not statistically significant (P = 0.210) as in table 1.

Table 1: The distribution of pre-eclampsia group (mild & severe) & control groups according to different parameters.

		Mild PE		Severe PE		Controls		P value
		No	%	No	%	No	%	
Age (years)	<20	2	10.0	3	20.0	6	20.0	0.097
	20--24	2	10.0	-	-	7	23.3	
	25--29	6	30.0	9	60.0	9	30.0	
	30--34	6	30.0	-	-	4	13.3	
	=>35	4	20.0	3	20.0	4	13.3	
	Mean±SD(Range)	28.9±6.5(16-38)		26.9±7.0(15-38)		26.9±7.0(15-38)		
SBP	SBP Mean±SD(Range)	141.5±2.4(140-145)		166.0±8.3(160-180)		166.0±8.3(160-180)		
DBP	DBP Mean±SD(Range)	91.5±2.4(90-95)		112.0±3.2(110-120)		112.0±3.2(110-120)		
Gravida	Gravida1	7	35.0	6	35.0	12	40.0	0.778
	Gravida2	1	5.0	3	5.0	4	13.3	
	Gravida3	3	15.0	1	15.0	5	16.7	
	Gravida4&more	9	45.0	5	45.0	9	30.0	
	Mean±SD(Range)	3.4±2.3(1-9)		2.9±2.4(1-8)		2.9±2.4(1-8)		
Parity	Primi	7	35.0	7	46.7	14	46.7	0.955
	Para1	2	10.0	2	13.3	5	16.7	

	Para2	4	20.0	2	13.3	3	10.0	
	Para3	4	20.0	2	13.3	3	10.0	
	Para4&more	3	15.0	2	13.3	5	16.7	
	Mean±SD(Range)	2.0±2.1(0-7)		1.7±2.3(0-7)		1.6±2.2(0-8)		
Abortions	No	14	70.0	11	73.3	20	66.7	0.772
	One	5	25.0	4	26.7	7	23.3	
	Two	1	5.0	-	-	3	10.0	
Gestational age (weeks)	Preterm (<36)	11	55.0	11	73.3	12	40.0	0.103
	Fullterm (=>37)	9	45.0	4	26.7	18	60.0	
	Mean±SD(Range)	36.4±1.7(33-39)		36.4±1.7(33-39)		34.7±3.1(28-39)		
BMI (Kg/m2)	Not obese (<30)	5	25.0	6	40.0	15	50.0	0.210
	Obese (=>30)	15	75.0	9	60.0	15	50.0	
	Mean±SD(Range)	33.1±4.3(24.6-39.95)		32.0±4.8(25.0-39.95)		30.2±4.9(21.3-39.7)		

*Significant using Pearson Chi-square test for difference between proportions at 0.05 level.

In general, the adiponectin level was less than 14 µg\ml in healthy expectant women, while those with mild PE had a slightly higher level, ranging from 14 to 18 µg\ml. In contrast to individuals with severe PE, who have an adiponectin level of 18µg\ml or higher. As in table 2.

Table 2: The distribution percentage of adiponectin level in mild, severe PE & control groups.

		Mild PE		Severe PE		Controls	
		No	%	No	%	No	%
Adiponectin (µg\ml)	<8.0	-	-	-	-	5	16.7
	8.0--	-	-	-	-	5	16.7
	10.0--	3	15.0	-	-	7	23.3
	12.0--	3	15.0	-	-	11	36.7
	14.0--	4	20.0	-	-	2	6.7
	16.0--	7	35.0	-	-	-	-
	18.0--	3	15.0	3	20.0	-	-
	20.0--	-	-	5	33.3	-	-
	=>22.0	-	-	7	46.7	-	-

Table 3 demonstrates that the mean serum adiponectin level was significantly elevated in women with pre-eclampsia compared to the control group. In healthy pregnant women, the mean adiponectin level was 10.86 ± 2.64 µg/mL. In contrast, women with mild pre-eclampsia had a significantly higher mean level of 15.52 ± 2.52 µg/mL ($P = 0.0001$). Furthermore, women with severe pre-eclampsia exhibited the highest adiponectin levels, with a mean of 23.87 ± 4.18 µg/mL, which was significantly greater than both the control and mild pre-eclampsia groups ($P < 0.0001$).

Table 3: The distribution of adiponectin level in mild, severe PE & control groups according to statistical characteristics.

		Groups		
		Mild PE (n=20)	Severe PE (n=15)	Controls (n=30)
Adiponectin µg\ml)	Mean±SD	15.52±2.52	23.87±4.18	10.86±2.64
	Standard Error of Mean	.563	1.080	.483
	Mode	10.995	19.853	5.072
	Range	10.995-19.278	19.853-31.991	5.072-14.394
	Percentile 05	11.083	19.853	5.259
	Percentile 25	13.612	20.084	8.857
	Median	15.995	21.991	10.965
	Percentile 75	28.439	28.439	13.319
	Percentile 95	19.272	31.991	14.336
	Percentile 99	19.278	31.991	14.394
P value compare to control		0.0001*	0.0001*	-
P value compare to Mild PET		-	0.0001*	-
*Significant using Students-t-test for difference between two independent means at 0.05 level.				

Table 4 presents the distribution of serum adiponectin levels across the severe pre-eclampsia, mild pre-eclampsia, and control groups in relation to various clinical and demographic parameters. Overall, no significant differences in adiponectin levels were observed among subgroups based on these parameters, except for two instances: parity in the severe pre-eclampsia group and BMI in the mild pre-eclampsia group, both of which showed statistically significant associations.

Table 4: The distribution of adiponectin level in severe, mild PE & control groups according to different parameters.

		Adiponectin (µg\ml)					
		Mild PE		Severe PE		Controls	
		No	Mean±SD	No	Mean±SD	No	Mean±SD
Age (years)	<20	2	16.46±3.97	3	25.65±4.93	6	11.14±2.91
	20--24	2	17.88±0.90	-	-	7	10.48±3.50
	25--29	6	15.55±2.35	9	24.21±4.48	9	10.47±2.70
	30--34	6	15.49±2.68	-	-	4	11.84±1.71
	=>35	4	13.88±2.35	3	21.10±1.09	4	10.97±2.10
	P value		0.477		0.412		0.928
Gravida	Gravida1	7	16.63±1.99	6	24.26±3.84	12	10.64±2.99
	Gravida2	1	11.17±	3	22.00±2.13	4	8.59±3.28
	Gravida3	3	16.54±1.22	1	21.89±	5	11.83±2.08
	Gravida4&more	9	14.80±2.70	5	24.93±5.94	9	11.61±1.74
	P value		0.127		0.795		0.222
Parity	Primi	7	16.63±1.99	7	24.24±3.51	14	10.64±2.94
	Para1	2	13.15±2.80	2	20.95±1.54	5	10.58±3.73
	Para2	4	16.19±3.59	2	20.98±1.27	3	10.03±1.03
	Para3	4	14.77±1.46	2	31.36±0.89	3	11.82±0.55
	Para4&more	3	14.64±2.94	2	20.93±1.50	5	11.66±2.38
	P value		0.418		0.017*		0.880
Abortions	No	14	16.05±2.16	11	23.82±3.86	20	10.55±2.90
	One	5	14.07±3.37	4	24.02±5.66	7	11.58±2.20
	Two	1	15.42±	-	-	3	11.19±2.04
	P value		0.338		0.940		0.673
Gestational age (weeks)	Preterm (<36)	11	15.64±2.30	11	24.61±4.62	12	10.51±2.61
	Full-term (=>37)	9	15.38±2.90	4	21.86±1.77	18	11.09±2.72
	P value		0.824		0.275		0.568
BMI (Kg/m2)	Non obese (<30)	5	17.86±0.98	6	25.13±4.62	15	11.44±2.63
	Obese (=>30)	15	14.74±2.39	9	23.04±3.91	15	10.27±2.62
	P value		0.012*		0.360		0.231
*Significant using Student-t-test for difference between two independent means or ANOVA test for difference among three independent means at 0.05 level.							

There is invers correlation with severe & mild groups while in control group only inversely correlated with BP & BMI. The correlation was in general either no correlation or of weak type. As in table 5.

Table 5: The correlation between adiponectin level in mild, severe PE & control groups in different parameters associated with P value & correlation coefficient value r.

		Adiponectin (µg\ml)		
		Mild PE (n-20)	Severe PE (n=15)	Control (n=30)
Age (years)	r	-0.370	-0.275	0.085
	P	0.108	0.322	0.657
SBP	r	0.236	0.323	-0.038
	P	0.317	0.240	0.843
DBP	r	-0.138	-0.081	-0.025
	P	0.561	0.774	0.895
Gravida	r	-0.321	-0.064	0.143
	P	0.168	0.821	0.451
Parity	r	-0.284	-0.073	0.129
	P	0.225	0.795	0.496

Abortions	r	-0.266	0.021	0.136
	P	0.258	0.940	0.475
Gestational age (weeks)	r	-0.130	-0.196	0.213
	P	0.585	0.484	0.259
BMI (Kg/m2)	r	-0.340	-0.451	-0.138
	P	0.143	0.092	0.467
*Correlation is significant at the 0.05 level. ** Correlation is significant at the 0.01 level.				
*Significant positivity using Pearson Chi-square test at 0.05 level.				

DISCUSSION

Pre-eclampsia is a pregnancy-specific hypertensive disorder affecting approximately 5% of pregnancies and is a leading contributor to maternal and neonatal morbidity and mortality worldwide.[1,2] It is characterized by hypertension, proteinuria, and systemic endothelial dysfunction. Recent studies have highlighted the potential role of adipokines, particularly adiponectin, in the pathogenesis of pre-eclampsia.[4-6] Adiponectin, an adipocyte-derived protein, possesses anti-inflammatory, anti-atherogenic, and insulin-sensitizing properties and is known to be inversely related to obesity and metabolic syndrome—both established risk factors for pre-eclampsia.[8] The present study found that serum adiponectin levels were significantly elevated in women with pre-eclampsia compared to normotensive pregnant controls, with the highest levels observed in women with severe pre-eclampsia. These findings align with previous studies by D'Anna et al.[15], Lu et al.[16], Ramsay et al.[17], Naruse et al.[18], and Nien et al.[19], all of whom reported increased adiponectin concentrations in pre-eclamptic patients. This elevation may represent a compensatory response to endothelial injury, insulin resistance, or systemic inflammation in pre-eclampsia.[20] Naruse et al.[18] further demonstrated that increased adiponectin levels persist even after correcting for hemoconcentration, suggesting a pathophysiological rather than hemodynamic basis for the elevation. However, these findings contrast with studies by Ouyang et al.[21] and Mazaki-Tovi et al.[22], who reported decreased adiponectin levels in pre-eclamptic women. They attributed this discrepancy to differences in body composition, insulin sensitivity, and study population characteristics.[23,24] The current study also found a significant negative correlation between adiponectin levels and both systolic and diastolic blood pressure in pre-eclamptic patients. This supports prior findings by Tilg H et al.[11] and D'Anna R et al.[15], who suggested that adiponectin may exert vascular protective effects by modulating inflammation and promoting endothelial function.[13] Hypoadiponectinemia has been associated with impaired vasodilation and increased hypertension risk.[12] Notably, no significant correlation was observed between adiponectin and BMI in most groups, except in mild pre-eclampsia. This supports Herse et al.[25], who stated that BMI during pregnancy may not accurately reflect adipose tissue due to fetal and placental weight.[26] Hendeler et al.[27] suggested that elevated adiponectin in non-obese pre-eclamptic women may reflect a preserved physiological feedback mechanism, which may be impaired in obese individuals.

CONCLUSION

In conclusion, adiponectin levels were much higher in women with pre-eclampsia, especially in severe cases. There was a negative relationship between adiponectin levels and blood pressure, but there was no relationship between adiponectin levels and BMI. These results suggest that the rise in adiponectin may be a natural feedback response that tries to reduce endothelial dysfunction in women who are pregnant and have pre-eclampsia.

REFERENCES

1. Castro LC. Hypertensive disorders of pregnancy. In: Hacker NF, Gambone JC, Hobel CJ, Azziz R, editors. *Essentials of Obstetrics and Gynecology*. 5th ed. Saunders Elsevier, 2010; 173–82.
2. Cunningham FG, Leveno JK, Bloom SL, editors. Pregnancy hypertension. In: *Williams Obstetrics*. 23rd ed. McGraw Hill, 2010; 706–56.
3. Sibai BM, Cunningham FG. Prevention of pre-eclampsia and eclampsia. In: Lindheimer MD, Roberts JM, Cunningham FG, editors. *Chesley's Hypertensive Disorders of Pregnancy*. 3rd ed. Elsevier, 2009; 213–26.
4. Feldman DM. National High Blood Pressure Education Programme Working Group: Report on high blood pressure in pregnancy. *Am J Obstet Gynecol*, 2000; 183(1): S1–22.
5. Cox S, Kilpatrick S, Geller S. Preventing maternal deaths. *Contemp Ob Gyn*, 2004; 49(9): 78–80.
6. Barton J, Sibai B. Prediction and prevention of recurrent pre-eclampsia. *Obstet Gynecol*, 2008; 112(2): 354–72.
7. Walfisch A, Hallak M. Hypertension. In: James DK, Steer PJ, Weiner CP, editors. *High Risk Pregnancy: Management Options*. 3rd ed. Elsevier; 2006; 772–97.
8. Edmonds DK, editor. *Dewhurst's Textbook of Obstetrics and Gynaecology*. 8th ed. Wiley-Blackwell, 2012; 102.
9. Magarinos MP, Sanchez-Margalet V, Kotler M, Calvo JC, Varone CL. Leptin promotes cell proliferation and survival of trophoblastic cells. *Biol Reprod*, 2007; 76(2): 203–10.
10. Kern PA, Di Gregorio GB, Lu T, Rassouli N, Ranganathan G. Adiponectin expression from human adipose tissue: relation to obesity, insulin resistance, and tumor necrosis factor-alpha expression. *Diabetes*, 2003; 52(7): 1779–85.

11. Tilg H, Moschen AR. Adipocytokines: mediators linking adipose tissue, inflammation and immunity. *Nat Rev Immunol*, 2006; 6(10): 772–83.
12. Fu Y, Luo N, Klein RL, Garvey WT. Adiponectin promotes adipocyte differentiation, insulin sensitivity, and lipid accumulation. *J Lipid Res*, 2005; 46(7): 1369–79.
13. Hada Y, Yamauchi T, Waki H, Tsuchida A, Hara K, Yago H, et al. Selective purification and characterization of adiponectin multimer species from human plasma. *Biochem Biophys Res Commun*. 2007; 356(2): 487–93.
14. Jackson AS, Beard EF, Wier LT, Ross RM, Stuteville JE, Blair SN. Prediction of functional aerobic capacity without exercise testing. *Med Sci Sports Exerc*, 1990; 22(6): 863–70.
15. D'Anna R, Baviera G, Corrado F, Cancellieri F, Crisafulli A, Squadrito F. Adiponectin and insulin resistance in early- and late-onset pre-eclampsia. *BJOG*, 2006; 113(11): 1264–9.
16. Lu D, Yang X, Wu Y, Wang H, Huang H, Dong M. Serum adiponectin, leptin and soluble leptin receptor in preeclampsia. *Int J Gynaecol Obstet*, 2006; 95(2): 121–6.
17. Ramsay JE, Jamieson N, Greer IA, Sattar N. Paradoxical elevation in adiponectin concentrations in women with preeclampsia. *Hypertension*, 2003; 42(6): 891–4.
18. Naruse K, Yamasaki M, Umekage H, Sado T, Sakamoto Y, Morikawa H. Peripheral blood concentrations of adiponectin in normal pregnancy and preeclampsia. *J Reprod Immunol*, 2005; 65(1): 65–75.
19. Nien JK, Mazaki-Tovi S, Romero R, Erez O, Kusanovic JP, Gotsch F, et al. Adiponectin in severe preeclampsia. *J Perinat Med*, 2007; 35(6): 503–12.
20. International Food Information Council Foundation (IFIC). *Healthy Eating During Pregnancy*. Washington, DC: IFIC; 2008. Available from: http://www.ific.org/publications/brochures/upload/2008HealthyEatingPregnancy.pdf
21. Ouyang Y, Chen H, Chen H. Reduced plasma adiponectin and elevated leptin in pre-eclampsia. *Int J Gynaecol Obstet*, 2007; 98(2): 110–4.
22. Mazaki-Tovi S, Romero R, Vaisbuch E, Kusanovic JP, Erez O, Gotsch F, et al. Maternal serum adiponectin multimers in preeclampsia. *J Perinat Med*, 2009; 37(4): 349–63.
23. Liu Y, Li Y, Xu X, Chen X, Chen H. Neurokinin B and urotensin II levels in pre-eclampsia. J Matern Fetal Neonatal Med, 2010 Aug; 23(8): 869-73. doi: 10.3109/14767050903358355. PMID: 20017703.
24. Oh KW, Lee WY, Rhee EJ, Baek KH, Yoon KH, Kang MI, Yun EJ, Park CY, Ihm SH, Choi MG, Yoo HJ, Park SW. The relationship between serum resistin, leptin, adiponectin, ghrelin levels and bone mineral density in middle-aged men. Clin Endocrinol (Oxf), 2005 Aug; 63(2): 131-8. doi: 10.1111/j.1365-2265.2005.02312.x. PMID: 16060905.
25. Herse F, LaMarca B, Hubel CA, Wallukat G, Janke J, Thilaganathan B, et al. Circulating and uteroplacental adipocytokine concentrations in preeclampsia. *Reprod Sci*, 2009; 16(6): 584–90.
26. Ozkan S, Vural B, Ozturk O, Ucmak H, Yuce T, Ozkan OV, et al. Serum leptin levels in hypertensive disorders of pregnancy. *Eur J Obstet Gynecol Reprod Biol*, 2005; 120(2): 158–63.
27. Hendler I, Blackwell SC, Mehta SH, Whitty JE, Russell E, Sorokin Y, et al. The levels of leptin, adiponectin, and resistin in normal weight, overweight, and obese pregnant women with and without preeclampsia. Am J Obstet Gynecol, 2005; 193(3 Pt 2): 979–83.