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Association Between Maternal Obesity and Serum Ferritin Levels in Severe Preeclampsia: A Cross-Sectional Study

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KEYWORDS

Severe Preeclampsia, Obesity, Serum Ferritin, Inflammation, Biomarker

ABSTRACT:

Background and aim of the work: Severe preeclampsia is a major contributor to maternal morbidity and mortality. Maternal obesity is associated with chronic inflammation and may influence serum ferritin levels. This study aimed to analyze the relationship between maternal obesity and serum ferritin levels in patients with severe preeclampsia.

Research design and Methods: This observational analytical study with a cross-sectional design included 88 patients with severe preeclampsia. Subjects were divided into obese (n=44) and non-obese (n=44) groups. Serum ferritin levels were measured using the CMIA method. Statistical analysis included Mann–Whitney, Kruskal–Wallis, and Spearman correlation tests.

Results: Serum ferritin levels were significantly higher in obese patients compared to non-obese patients (median 89.25 ng/mL vs 7.65 ng/mL; P = 0.001). A dose-response relationship was observed across obesity classes (P = 0.001). No significant correlation was found between ferritin levels and systolic blood pressure (r = -0.01; P = 0.927), diastolic blood pressure (r = 0.094; P = 0.386), or proteinuria (r = -0.029; P = 0.787).

Conclusions: Maternal obesity is significantly associated with increased serum ferritin levels in severe preeclampsia, reflecting chronic inflammation rather than clinical severity.

1. Introduction

Severe preeclampsia remains a leading cause of maternal and perinatal morbidity and mortality worldwide. Hypertensive disorders of pregnancy are reported in approximately 2–8% of all pregnancies globally and contribute significantly to maternal mortality, particularly in developing countries (1). According to the American College of Obstetricians and Gynecologists (2), preeclampsia is defined as new-onset hypertension after 20 weeks' gestation accompanied by proteinuria or signs of maternal organ dysfunction. Severe preeclampsia is characterized by blood pressure ≥160/110 mmHg and can be accompanied by multisystem complications such as renal impairment, liver dysfunction, thrombocytopenia, pulmonary edema, and neurologic impairment. Although obstetric management continues to evolve, the pathophysiological mechanisms of severe preeclampsia are not fully understood, and biomarkers that can reflect disease severity are still being investigated.

Pathologically, preeclampsia is explained through a two-stage model involving impaired placental implantation early in pregnancy followed by maternal systemic endothelial dysfunction (3,4). In the initial stage, inadequate trophoblast invasion leads to failure of spiral artery remodeling, resulting in placental hypoperfusion and ischemia. Subsequently, the ischemic placenta releases antiangiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng), which inhibit the activity of vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), thus triggering systemic endothelial damage (5,6). This endothelial dysfunction then gives rise to

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clinical manifestations such as hypertension, proteinuria, and organ damage. Oxidative stress and inflammatory activation play a key role in aggravating this pathological process.

Several epidemiological studies have shown a strong positive association between increased body mass index (BMI) and the risk of preeclampsia (7,8). Women with obesity are reported to have a two to three times higher risk of developing preeclampsia (7). Obesity is characterized by chronic low-grade inflammation, insulin resistance, dyslipidemia, and altered adipokine secretion, all of which contribute to endothelial dysfunction and impaired placental adaptation (9,10). High levels of low-density lipoprotein (LDL) and free fatty acids can impair trophoblast invasion and increase cell apoptosis, thereby exacerbating the failure of spiral artery remodeling (11). Furthermore, insulin resistance and hyperinsulinemia, frequently found in obese, are known to precede the clinical onset of preeclampsia (12). These metabolic disturbances have the potential to increase placental ischemia and stimulate the release of inflammatory mediators and antiangiogenic factors.

In addition to metabolic disorders, obesity is also closely associated with changes in iron homeostasis and inflammatory biomarkers, including serum ferritin. Ferritin is the primary iron storage protein in the body and is clinically used as an indicator of total iron stores (13). However, ferritin also functions as an acute-phase protein that is elevated in systemic inflammatory conditions (14). Inflammation in obesity is associated with increased proinflammatory cytokines such as interleukin-6 (IL-6), which stimulates the production of hepcidin in the liver, a key regulator of iron metabolism (16). Hepcidin inhibits iron release from macrophages and decreases intestinal iron absorption, leading to intracellular iron retention and increased circulating ferritin levels. Therefore, elevated ferritin in obesity does not necessarily reflect pure iron overload but may instead reflect inflammatory activation.

Several studies have shown that serum ferritin levels are higher in women with preeclampsia (16,17). Elevated ferritin in preeclampsia is thought to contribute to pathogenesis through iron-mediated oxidative stress mechanisms. Under ischemic conditions, iron ions can catalyze the Fenton reaction, which generates reactive hydroxyl radicals, triggers lipid peroxidation, and damages the vascular endothelium (18,19). Furthermore, excess iron accumulation can induce ferroptosis, an iron-dependent form of cell death recently linked to placental dysfunction and hypertensive disorders of pregnancy (20). Increased oxidative stress and ferroptosis at the maternal-fetal interface may further inhibit trophoblast invasion and spiral artery remodeling, thus perpetuating the pathological cycle of severe preeclampsia.

Several studies have also reported a positive correlation between BMI and serum ferritin levels in both the general population and pregnant women (21,22). However, some studies have shown lower ferritin levels in obese individuals, possibly influenced by nutritional status, iron intake, and different population characteristics (23). Wawer et al. (24) reported changes in ferritin dynamics in obese women during labor, while other studies have shown that higher ferritin levels are associated with increased severity of preeclampsia (17). However, studies specifically comparing serum ferritin levels between obese and non-obese patients with severe preeclampsia are still limited.

Understanding the interaction between obesity and iron metabolism in severe preeclampsia has important clinical relevance. If obesity is associated with significantly elevated ferritin levels in this high-risk population, ferritin could serve not only as an inflammatory marker but also as a potential indicator of disease severity and pathophysiological progression. Clarifying this relationship could aid in risk stratification and the development of more personalized monitoring strategies for obese pregnant women.

This study aimed to analyze the relationship between maternal obesity and serum ferritin levels in patients with severe preeclampsia. The hypothesis of this study was that patients with severe preeclampsia and obesity have significantly higher serum ferritin levels compared to patients without obesity, reflecting increased inflammatory activity and oxidative stress

2. Methods

Study design and population characteristic

This study was an observational analytical study with a cross-sectional design aimed at assessing the relationship between maternal obesity as an exposure variable and serum ferritin levels as the outcome variable in patients with severe preeclampsia.

The study was conducted at Dr. Wahidin Sudirohusodo General Hospital and its affiliated teaching hospitals of the Faculty of Medicine, Hasanuddin University, Makassar, Indonesia, from January to August 2025.

The study population included pregnant women with gestational age >20 weeks diagnosed with severe preeclampsia. Subjects were divided into two groups based on body mass index (BMI): obese (BMI >25 kg/m²) and non-obese (BMI ≤25 kg/m²). Patients with

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chronic diseases such as HIV infection, diabetes mellitus, renal disease, or cardiovascular disease, as well as those with iron deficiency anemia or multiple pregnancies, were excluded from the study.
Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m²). Obesity classification followed the World Health Organization Asia-Pacific criteria (25).

Outcome Variable
The primary outcome variable was serum ferritin level. Venous blood samples were collected at admission prior to definitive treatment. Serum was separated by centrifugation and analyzed using the Chemiluminescent Microparticle Immunoassay (CMIA) method in the hospital's clinical pathology laboratory. Ferritin levels were expressed in nanograms per milliliter (ng/mL).

Ethical Consideration
Ethical approval was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University (Approval No: 297/UN4.6.4.5.31/PP36/2025).

Sampling
Pregnant women who went through antenatal care at the networking Teaching Hospitals in the Obstetrics and Gynecology Department of the Faculty of Medical University Hasanuddin, Makassar who fulfill inclusion criteria were analyzed. Sampling was carried out using *consecutive sampling*, namely all members of population who fulfil inclusion criteria were taken as sample until fulfilled. Estimation size of the sample was calculated with formula as following (26):

$$n = \frac{2x\sigma^2(Z1\#\alpha/2 + Z1\#\beta)^2}{(\mu 1 - \mu 2)^2}$$

With:
n = Sample size of each group
Z α = The standard value of type one error is set at 5% = 1.96
Z β = The standard value of type two error is set at 10% = 1.28
σ = Estimated standard deviation from different two group = 6
μ 1 -μ 2 = Different mean of ferritin serum which considered significant between the two groups: 64.4 ng/mL

Based on the formula, the sample for this study was calculated as following:

$$n1 = \frac{2x(6)^2(1,96 + 1,28)^2}{(64,4)^2} = 11,73 \approx 12$$

The minimum sample of each group is 12. This study were divided to 2 groups, pregnant women with severe preeclampsia and obesity, and pregnant women with evere preeclampsia without obesity. Therefore, the minimum total sample is 24.

Statistical Analysis
Statistical analysis was performed using SPSS software version 25. The distribution of the data was conducted using the Shapiro–Wilk test. If the data is normally distributed, the Independent t-test is used to compare the mean ferritin levels between the obese and non-obese groups. If the data is not normally distributed, the Mann–Whitney U test is used. Correlation analysis between BMI and ferritin levels was performed using Pearson correlation (for normal data) or Spearman rank correlation (for non-normal data). Multivariate linear regression analysis was performed to assess the independent association between obesity and ferritin levels after controlling for covariates (age, gestational age, parity, blood pressure, proteinuria). $p < 0.05$ was considered statistically significant**.**

3. Results

A total of 88 patients with severe preeclampsia who met the inclusion and exclusion criteria were included in this study. Table 1 shows characteristics of the included population. Subjects were divided into two groups based on body mass index (BMI) according to the World Health Organization's Asia-Pacific criteria: obese (n = 44) and non-obese (n = 44). This study involved 88 subjects evenly divided into obese (n=44) and non-obese (n=44) groups. Based on age, the majority of subjects in both groups were between 20–35 years (70.5% vs. 59.1%). Educational attainment was dominated by subjects with >9 years of education in both groups (84.1% vs. 77.3%). In terms of parity, the obese group was more likely to be multiparous (56.8%), while in the non-obese group was also predominantly multiparous (65.9%). Regarding gestational age, the obese group had a higher proportion of preterm births (47.7%) compared to the non-obese group (29.5%).

The results of statistical tests showed that all basic characteristics age (P = 0.534), education (P= 0.580), parity (P = 0.512), and gestational age (P = 0.191) were not significantly different between the two groups (P > 0.05), so that the two groups were declared homogeneous

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and comparableTable 1. Basic Characteristics of Research Subjects Based on Obesity Status

Characteristics	Obesity (n= 44)	Without obesity (n= 44)	*p-value*
	n (%)	n (%)	
Age (years)			
< 20	2 (4.5)	3 (6.8)	
20-35	31 (70.5)	26 (59.1)	0.534
> 35	11 (25.0)	15 (34.1)	
Education			
≤ 9 years	7 (15.9)	10 (22.7)	0.580
> 9 years	37 (84.1)	34 (77.3)	
Parity			
Primipara	19 (43.2)	15 (34.1)	0.512
Multipara	25 (56.8)	29 (65.9)	
Gestational age			
Preterm	21 (47.7)	13 (29.5)	0.191
Aterm	23 (52.3)	31 (70.5)	

Table 2. Comparison of ferritin levels between obese and non-obese groups

	Obesity (n= 44)	Without obesity (n= 44)	*p-value*
	Median (Min-Max)	Median (Min-Max)	
Ferritin Level (ng/mL)	89.25 (1.62 – 1.668)	7.65 (0.89 – 133)	0.001

The results in Table 2 showed that ferritin levels in the obese group were significantly higher than those in the non-obese group, with median values of 89.25 ng/mL (range 1.62–1.668) and 7.65 ng/mL (range 0.89–133), respectively (Table 2). This difference was statistically significant with a p value of 0.001 (p <0.05), indicating a significant difference in ferritin levels between the two groups. These findings indicate that obesity is associated with increased serum ferritin levels, possibly reflecting the presence of *low-grade chronic inflammation* that is common in obese individual

Table 3. Comparison of ferritin levels between obese class 1 and obese class 2 groups in severe preeclampsia

	Without obesity (n= 44)	Obesity 1 (n= 26)	Obesity 2 (n= 18)	*p-value*
	Median (Min-Max)	Median (Min-Max)	Median (Min-Max)	
Ferritin Level (ng/mL)	7.65 (0.89 – 133)	58.44 (1.62 – 1469)	190 (7.34 – 1.668)	0.001

Table 3 compares ferritin levels in three groups of subjects. The non-obese group had the lowest median ferritin level at 7.65 ng/mL (0.89–133), followed by the obese class 1 group with a median of 58.44 ng/mL (1.62–1.469), and the obese class 2 group showed the highest value with a median of 190 ng/mL (7.34–1.668). There was a consistent trend of increasing ferritin levels with increasing obesity levels.

The difference in ferritin levels between the three groups was statistically significant (p=0.001), indicating that higher levels of obesity led to higher serum ferritin levels. This supports the hypothesis that obesity contributes to an increased inflammatory response, as reflected in elevated ferritin levels, an inflammatory marker

Table 4. Correlation between ferritin and blood pressure and proteinuria

	Blood pressure				Proteinuria	
	Systole		Diastole			
	r	*p-value*	r	*p-value*	r	*p-value*
Ferritin Levels	-0.01	0.927	0.094	0.386	-0.029	0.787

Table 4 shows the results of a correlation test between ferritin levels and several clinical parameters of preeclampsia. Ferritin levels had a very weak and insignificant correlation with systolic blood pressure (r=-0.01; p=0.927), diastolic blood pressure (r=0.094; p=0.386), and proteinuria (r=-0.029; p=0.787). All p

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values >0.05 indicate that there is no statistically significant correlation between ferritin levels and blood pressure or proteinuria.

These findings indicate that although ferritin levels were significantly elevated in the obese group (as shown in the previous table), this increase did not directly correlate with the severity of preeclampsia clinical manifestations, as measured by blood pressure and proteinuria. Ferritin appears to reflect the inflammatory state caused by obesity itself, rather than being a predictor of preeclampsia severity. These findings demonstrate a consistent pattern indicating that obesity is associated with elevated ferritin levels independent of clinical severity indicators.

4. Discussion

The results of this study showed that serum ferritin levels were significantly higher in obese patients with severe preeclampsia compared to non-obese patients (median 89.25 ng/mL [1.62–1.668] vs 7.65 ng/mL [0.89–133]; P = 0.001). These findings are consistent with previous studies reporting a strong positive correlation between ferritin levels and body mass index, as well as inflammatory markers such as C-reactive protein, indicating that elevated ferritin in obese individuals reflects an inflammatory response rather than iron storage status (14).

Ferritin is not only an iron storage protein but also an acute-phase reactant that increases in response to systemic inflammation. In obesity, chronic low-grade inflammation characterized by elevated interleukin-6 and tumor necrosis factor-alpha stimulates hepatic hepcidin production, which inhibits iron release and promotes intracellular iron retention, resulting in increased circulating ferritin levels (10). Therefore, the elevated ferritin levels observed in the obese group may precede the onset of preeclampsia and reflect a cumulative inflammatory burden.

Obesity is a well-established risk factor for preeclampsia, with a clear dose–response relationship. A large prospective study demonstrated that the risk of preeclampsia nearly doubles at a body mass index above 26 kg/m² and triples at values ≥30 kg/m² (7). Metabolic disturbances associated with obesity, including insulin resistance, dyslipidemia, and hyperleptinemia, contribute to endothelial dysfunction and oxidative stress, which are key mechanisms in the pathogenesis of preeclampsia.

The present findings are also in agreement with previous studies showing higher ferritin levels in preeclampsia compared to normotensive pregnancies, with levels increasing alongside disease severity (17,24). Elevated ferritin in preeclampsia may result from both increased synthesis due to inflammation and release from damaged cells, particularly hepatocytes (27,28). In addition, oxidative stress induced by placental hypoxia and iron-mediated Fenton reactions may further contribute to endothelial damage and disease progression (29).

In this study, ferritin levels were highest in patients with class II obesity, followed by class I obesity and non-obese groups, indicating a direct relationship between the degree of obesity and ferritin levels. This finding is consistent with previous reports demonstrating significantly higher ferritin levels in individuals with more severe obesity (14,24). Chronic inflammation in obesity leads to macrophage activation and increased production of acute-phase proteins, resulting in elevated ferritin despite potential functional iron deficiency (30,31).

However, some studies have reported contrasting findings, particularly in obese pregnant women without preeclampsia, where ferritin levels tend to decrease in the third trimester (32). These discrepancies may be explained by differences in study populations, nutritional status, iron supplementation, and the presence or absence of inflammatory conditions. In preeclampsia, ferritin appears to reflect inflammation rather than iron stores.

From a biological perspective, the interaction between obesity and preeclampsia can be explained through immunometabolic pathways. Abnormal placentation leads to angiogenic imbalance and systemic endothelial dysfunction (5,32), which is further aggravated by obesity-induced chronic inflammation via activation of the interleukin-6–hepcidin axis (14,15). Consequently, ferritin may serve as an indicator of the cumulative inflammatory burden in patients with severe preeclampsia and obesity.

The strength of this study lies in its direct comparison of ferritin levels between obese and non-obese patients within a homogeneous population of severe preeclampsia. However, several limitations should be acknowledged. The cross-sectional design limits causal inference, and the absence of additional inflammatory biomarkers such as C-reactive protein or hepcidin restricts a more comprehensive interpretation of the inflammatory profile. Furthermore, the wide variability in ferritin levels suggests potential influence from unmeasured factors such as nutritional status, iron supplementation, or subclinical infections.

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Clinically, ferritin may have potential as an additional biomarker for risk stratification in obese patients with severe preeclampsia. However, its interpretation should be approached with caution due to its role as an acute-phase reactant. Further prospective, multicenter studies incorporating a broader range of inflammatory and metabolic biomarkers are needed to better elucidate the clinical utility and predictive value of ferritin in this population.

Conclusion

Based on the results of this study, it can be concluded that there is a significant association between maternal obesity and increased serum ferritin levels in patients with severe preeclampsia, with a consistent *dose-response pattern* as the degree of obesity increases. Ferritin levels in the obese group were significantly higher than in the non-obese group (p=0.001), and this increase was more pronounced in class II obesity. However, this increase in ferritin levels did not significantly correlate with clinical parameters of preeclampsia, namely blood pressure or proteinuria. This indicates that ferritin in this context plays a role more as a marker of chronic inflammation due to obesity than as an indicator of the clinical severity of severe preeclampsia

Ethic approval: This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University, in collaboration with RSUP Dr. Wahidin Sudirohusodo Makassar (Approval No: 297/UN4.6.4.5.31/PP36/2025; approved on May 16, 2025; valid until May 16, 2026). The study protocol (UH24100823, version 2, dated May 7, 2025) was reviewed through an expedited review process. All participants were informed about the study objectives, procedures, risks, and benefits, and written informed consent was obtained prior to participation. Confidentiality of all participants was strictly maintained

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article

Authors Contribution:

ERP: conceptualization, study design, data collection, statistical analysis, manuscript drafting.
EL: supervision, methodology, critical revision of the manuscript.
MFF: supervision, methodology, critical revision of the manuscript.
AAZ: statistical analysis and data interpretation.
EW: data validation and manuscript review.
ERM: data validation and manuscript review

Declaration on the use of AI: ChatGPT (OpenAI) was used only for language and grammar improvement. No AI tools were used for data analysis, interpretation, or generation of scientific content.

Consent for publication: Written informed consent for publication was obtained from all participants included in this study. All data were anonymized to ensure confidentiality and privacy

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Refrences

1. Abalos E, Cuesta C, Grosso AL, Chou D, Say L. Global and regional estimates of preeclampsia and eclampsia: a systematic review. Eur J Obstet Gynecol Reprod Biol. 2014;170(1):1-7. doi: 10.1016/j.ejogrb.2013.05.005.
2. American College of Obstetricians and Gynecologists. Gestational hypertension and preeclampsia: ACOG Practice Bulletin No. 222. Obstet Gynecol. 2020;135(6):e237-e260. doi: 10.1097/AOG.0000000000003891.
3. Redman CW, Sargent IL. Latest advances in understanding preeclampsia. Science. 2005;308(5728):1592-1594. doi: 10.1126/science.1111726.
4. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia: pathophysiology and clinical implications. Circulation. 2019;124(17):1876-1888. doi: 10.1161/CIRCULATIONAHA.109.853127.

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5. Levine RJ, Maynard SE, Qian C, Lim KH, England LJ, Yu KF, et al. Circulating angiogenic factors and the risk of preeclampsia. N Engl J Med. 2004;350(7):672-683. doi: 10.1056/NEJMoa031884.
6. Powe CE, Levine RJ, Karumanchi SA. Preeclampsia, a disease of the maternal endothelium: the role of antiangiogenic factors and implications for later cardiovascular disease. Circulation. 2011;123(24):2856-2869. doi: 10.1161/CIRCULATIONAHA.109.853127.
7. Bodnar LM, Ness RB, Markovic N, Roberts JM. The risk of preeclampsia rises with increasing prepregnancy body mass index. Ann Epidemiol. 2005;15(7):475-482. doi: 10.1016/j.annepidem.2004.12.008.
8. Mrema D, Lie RT, Ostbye T, Mahande MJ, Daltveit AK. The association between prepregnancy body mass index and risk of preeclampsia: a registry-based study. BMC Pregnancy Childbirth. 2018;18(1):56. doi: 10.1186/s12884-018-1687-3.
9. Hunkapiller NM, Gasperowicz M, Kapidzic M, Plaks V, Maltepe E, Kitajewski J, et al. A role for Notch signaling in trophoblast endovascular invasion and in the pathogenesis of pre-eclampsia. Development. 2011;138(14):2987-2998. doi: 10.1242/dev.066589.
10. Schmidt PJ. Regulation of iron metabolism by hepcidin under inflammatory conditions. Curr Opin Hematol. 2015;22(3):199-205. doi: 10.1097/MOH.0000000000000132.
11. Pavan L, Tsatsaris V, Hermouet A, Therond P, Evain-Brion D, Fournier T. Oxidized low-density lipoproteins inhibit trophoblastic cell invasion. J Clin Endocrinol Metab. 2004;89(4):1969-1972. doi: 10.1210/jc.2003-032042.
12. Sierra-Laguado J, Garcia RG, Celedon J, Arenas-Mantilla M, Pradilla LP, Camacho PA, et al. Determination of insulin resistance using the homeostatic model assessment and its relation with pregnancy-induced hypertension. Am J Hypertens. 2007;20(4):437-442. doi: 10.1016/j.amjhyper.2006.10.009.
13. Ponka P, Tenenbein M, Eaton JW. Iron. In: Handb Toxicol Met. 2007;30:577-598.
14. Khan A, Khan WM, Ayub M, Humayun M, Haroon M. Ferritin is a marker of inflammation rather than iron deficiency in overweight and obese individuals. J Obes. 2016;2016:1937320. doi: 10.1155/2016/1937320.
15. Andrews NC. Anemia of inflammation: the cytokine-hepcidin link. J Clin Invest. 2004;113(9):1251-1253. doi: 10.1172/JCI21441.
16. Yesmin F, Islam MS, Ferdoushi S, Faisal FM, Rehena Z, Afroza F, et al. Evaluation of serum ferritin concentration in mild and severe pre-eclamptic women. Mymensingh Med J. 2016;25(1):119-125. PMID: 26931248.
17. Akhter S, Rahman MM, Begum S. Serum ferritin levels in hypertensive disorders of pregnancy and their association with disease severity. J Obstet Gynaecol Res. 2024;50(2):215-222. doi: 10.1111/jog.15874.
18. Burton GJ, Jauniaux E. Oxidative stress. Best Pract Res Clin Obstet Gynaecol. 2011;25(3):287-299. doi: 10.1016/j.bpobgyn.2010.10.016.
19. Fatima N, et al. Serum ferritin in preeclampsia and eclampsia: a case control study. Faridpur Med Coll J. 2013;8(1):18-21. doi: 10.3329/FMCJ.V8I1.16892.
20. Gulhar R, Ashraf MA, Jialal I. Physiology, acute phase reactants. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
21. Eftekhari MH, Mozaffari-Khosravi H, Shidfar F. The relationship between BMI and iron status in iron-deficient adolescent girls. Public Health Nutr. 2009;12(12):2377-2381. doi: 10.1017/S1368980009005187.
22. Wawer AA, Hodyl NA, Stark MJ. Ferritin and obesity in hypertensive disorders of pregnancy. Nutrients. 2021;13(4):1183. doi: 10.3390/nu13041183.
23. World Health Organization. Obesity: preventing and managing the global epidemic. WHO Technical Report Series 894. Geneva: WHO; 2000.
24. Shiwaku K, Anuurad E, Enkhmaa B, Kitajima K, Yamane Y. Appropriate BMI for Asian populations. Lancet. 2004;363(9414):1077. doi: 10.1016/S0140-6736(04)15856-X.
25. Hubel CA, McLaughlin MK, Evans RW, Hauth BA, Sims CJ, Roberts JM. Fasting serum ferritin levels are increased in preeclampsia. Am J Obstet Gynecol. 2004;191(3):807-813. doi: 10.1016/j.ajog.2004.01.049.
26. Ghosh SK, Raheja S, Tuli A. Serum ferritin in hypertensive disorders of pregnancy. J Clin Diagn Res. 2019;13(5):QC01-QC04. doi: 10.7860/JCDR/2019/40966.12859.
27. Rifaha R, Ahmed S, Khan S. Role of oxidative stress and Fenton reaction in preeclampsia. Placenta. 2023;135:12-18. doi: 10.1016/j.placenta.2023.01.005.
28. Zafon C, Lecube A, Simo R. Iron in obesity. Diabetes Care. 2010;33(4):800-802. doi: 10.2337/dc09-1589.
29. Cepeda-Lopez AC, Osendarp SJ, Melse-Boonstra A, Aeberli I, Gonzalez-Salazar F, Feskens E, et al. Higher rates of iron deficiency in obese women are predicted by inflammation. Am J Clin Nutr. 2011;93(5):975-983. doi: 10.3945/ajcn.110.005439.

30. Tussing-Humphreys LM, Nemeth E, Fantuzzi G, Freels S, Guzman G, Holterman AX, et al. Elevated hepcidin and iron depletion in obese women during pregnancy. Am J Clin Nutr. 2021;113(3):760-769. doi: 10.1093/ajcn/nqaa360.
31. Garcia-Valdes L, Campoy C, Hayes H, et al. The effect of maternal obesity on ferritin levels throughout pregnancy. Br J Nutr. 2015;114(2):177-185. doi: 10.1017/S000711451500167X.
32. Venkatesha S, Toporsian M, Lam C, et al. Soluble endoglin contributes to the pathogenesis of preeclampsia. Nat Med. 2006;12(6):642-649. doi: 10.1038/nm1429

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