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Evaluation of inflammatory markers in pregnancies with hyperemesis gravidarum

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Ethics Committee Approval
The study was approved by the Amasya University Ethics Committee (Date: October 19, 2023; Approval No: 111).
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

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No conflict of interest was declared by the authors.

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Abstract

Background/Aim: Hyperemesis gravidarum (HG) is a severe clinical condition characterized by intractable nausea and vomiting during pregnancy, yet its exact etiology remains elusive. This study aimed to investigate the association between HG and the inflammatory markers procalcitonin (PCT), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) to better understand the role of systemic inflammation in the disorder.

Methods: This prospective controlled study included 82 pregnant women diagnosed with HG and 82 healthy pregnant controls. Demographic data, serum PCT levels, NLR, PLR, and obstetric and neonatal outcomes were compared between the two groups. Additionally, patients in the HG group were analyzed according to the severity of ketonuria.

Results: No significant differences were observed in demographic characteristics between the groups. The HG group demonstrated significantly higher PCT and PLR levels than the control group ($P<0.001$ and $P=0.013$, respectively). Regarding obstetric and neonatal outcomes, both infant birth weight and first-minute Apgar scores were significantly lower in the HG group ($P=0.013$ and $P=0.043$, respectively). Within the HG group, patients exhibiting +3 urinary ketones had significantly higher PCT and PLR values compared to those with lower ketone levels ($P<0.001$ and $P=0.041$, respectively). No significant differences were found in other obstetric and neonatal parameters.

Conclusion: The elevation of PCT and PLR in pregnant women with HG suggests a potential involvement of systemic inflammation in the pathogenesis of the disorder. This inflammatory response may be associated with disease severity and contribute to adverse neonatal outcomes, such as reduced birth weight and lower first-minute Apgar scores.

Keywords: hyperemesis gravidarum, procalcitonin, neutrophil-lymphocyte ratio, platelet-lymphocyte ratio

Introduction

Nausea, with or without vomiting, is a frequently encountered symptom during pregnancy. Although mild cases are generally considered physiological adaptations to gestation, severe and persistent forms may lead to substantial maternal weight loss, ketonuria, fluid and electrolyte imbalances, and acid–base disturbances, a condition referred to as hyperemesis gravidarum (HG) [1-3]. The incidence of severe HG ranges between 0.3% and 3%, with critical cases often necessitating hospitalization. Women affected by severe HG are at increased risk of serious complications, including central pontine myelinolysis and Wernicke's encephalopathy, both of which can be life-threatening [4, 5]. Furthermore, severe HG has been associated with adverse fetal outcomes such as intrauterine growth restriction (IUGR), low birth weight (LBW), preterm birth, and low Apgar scores [6, 7]. Consequently, early recognition and appropriate management are essential to protect both maternal and fetal health.

Despite extensive research, the pathophysiology of HG has not yet been fully elucidated. Various mechanisms—including hormonal alterations, *Helicobacter pylori* infection, gastrointestinal dysmotility, placental dysfunction, and psychosocial factors—have been proposed; however, the precise etiology remains controversial. Traditionally, HG has not been classified as an inflammatory disorder. Nevertheless, emerging evidence suggests a possible association with inflammatory processes, supported by reports of elevated inflammatory biomarkers in affected women [8-11].

Procalcitonin (PCT), a peptide precursor of calcitonin secreted by the parafollicular cells of the thyroid gland, is recognized as a proinflammatory mediator. It contributes to inflammatory responses by up-regulating CD16 and CD14 surface proteins on neutrophils and lymphocytes [12, 13]. Although PCT is primarily used to assess the severity of bacterial infections, evidence indicates that PCT levels may also increase in noninfectious chronic inflammatory conditions [14, 15]. Elevated PCT concentrations have been observed in several pregnancy-related complications, including preeclampsia, eclampsia, gestational diabetes mellitus (GDM), and preterm premature rupture of membranes (PPROM) [16, 17].

During systemic inflammation, characteristic changes occur in circulating leukocytes, typically involving an increase in neutrophil counts and a decrease in lymphocyte counts. This makes the neutrophil-to-lymphocyte ratio (NLR) a practical marker of inflammatory activity, where higher values indicate more pronounced inflammation [18]. Similarly, platelets play a pivotal role in immune and inflammatory responses, and the platelet-to-lymphocyte ratio (PLR) has emerged as another inflammation-related index influenced by cytokine-mediated mechanisms [19]. Both NLR and PLR have been investigated in various chronic inflammatory conditions, gynecologic malignancies, and reproductive disorders [20-22]. In obstetric practice, elevated NLR levels have also been reported in conditions such as GDM, preeclampsia, and intrahepatic cholestasis of pregnancy [19, 23].

In light of these considerations, the present study aimed to investigate the association between inflammatory markers—namely PCT, NLR, and PLR—and hyperemesis gravidarum. Elucidating these relationships may enhance the understanding of the potential inflammatory mechanisms involved in HG and contribute to improved strategies for preventing HG-related complications.

Materials and methods

Ethical approval for this study was obtained from the Amasya University Ethics Committee (Date: October 19, 2023; Approval No: 111). The study was conducted in accordance with the principles of the Declaration of Helsinki.

This prospective controlled study was conducted between November 1, 2023, and January 10, 2024, including 82 pregnant women diagnosed with HG and 82 healthy pregnant controls. The following variables were analyzed: demographic characteristics (age, weight, height, body mass index [BMI], educational status, parity, previous surgical history, presence of chronic disease, smoking and alcohol use, week of diagnosis, and steroid use), serum PCT levels, neutrophil, lymphocyte, and platelet counts, NLR, PLR, and obstetric and neonatal outcomes.

A diagnosis of HG was established in women presenting with severe vomiting before the 20th week of gestation, resulting in >5% weight loss and necessitating hospitalization. Exclusion criteria included hypertension, diabetes mellitus, thrombophilia, imminent abortion, autoimmune disorders, renal, cardiac, or hepatic disease, preeclampsia, GDM, HELLP syndrome, or other inflammatory conditions. Additional exclusions included fetal anomalies and maternal conditions causing nausea, such as urinary tract infection or migraine. Only participants with viable intrauterine pregnancies confirmed by fetal cardiac activity on ultrasonography were included. Informed consent was obtained from all participants prior to enrollment.

Ultrasonographic examinations were performed by a single clinician (A.T.T.) using a Mindray DC-7 Ultrasound System. Fetal assessment included gestational age, growth, cardiac activity, amniotic fluid volume, and examination of the gestational sac. Blood samples were obtained under standardized conditions for hemogram analysis (neutrophil, lymphocyte, and platelet counts), liver and renal function tests, and serum PCT concentration. Urinalysis was performed to determine ketone levels. Hematologic parameters were measured using a laser optics analyzer (XN-1000, Siemens, Japan). Serum PCT levels were quantified via electrochemiluminescence immunoassay (Cobas e 411, Roche, Japan), and urinalysis was performed using a digital flow-cell system (FUS-200, Dirui, China).

Obstetric and neonatal outcomes (birth weight, gestational week at delivery, mode of delivery, NICU admission, 1st- and 5th-minute Apgar scores) were recorded. HG patients were classified into three subgroups based on urinary ketone levels (+1, +2, +3) for subgroup analysis.

Sample size

The required sample size was calculated using G*Power 3.1 software based on a previous study [10]. With an effect size of w=0.951 and a two-tailed hypothesis at a 95% confidence level, a minimum of 80 participants per group was determined to provide adequate statistical power.

Statistical analysis

Data were analyzed using IBM SPSS Statistics Version 23.0. The Kolmogorov–Smirnov test assessed data normality. Categorical variables were analyzed using the Chi-square or Fisher's exact test. For continuous variables, independent samples t-test or Mann–Whitney U test was applied. For multiple group comparisons, one-way ANOVA or Kruskal–Wallis test was used. A *P*-value <0.05 was considered statistically significant.

Results

No significant differences were observed between the HG and control groups regarding demographic characteristics, smoking or alcohol consumption, gestational week at diagnosis, or steroid administration for fetal lung maturation (Table 1). Serum PCT and PLR levels were significantly higher in the HG group compared with the control group (0.050 (0.013) vs 0.043 (0.013), *P*<0.001 and 128.80 (35.56) vs 116.81 (45.56), *P*=0.013, respectively), while no statistically significant difference was observed in NLR (Table 2).

Table 1: Comparison of the groups in terms of demographic characteristics, smoking, alcohol use, diagnosis week and steroid administration for lung development

		Study group n=82	Control group n=82	*P*-value
		Mean (SD)	Mean (SD)	
Age (year)		27.84 (4.92) (18 - 42)	27.82 (6.47) (18 - 43)	0.696
Weight (kg)		64.65 (14.22) (45 - 130)	65.27 (13.35) (41 - 109)	0.612
Height (cm)		161.07 (5.40) (146 - 172)	160.91 (6.12) (148 - 180)	0.861
BMI (kg/m^2)		24.93 (5.45) (16.26 - 48.93)	25.24 (5.20) (16.80 - 42.58)	0.539
Diagnosis week		9.88 (2.68)	10.24 (2.52)	0.228
		n (%)	n (%)	
Parity	Nulliparity	45 (45.9%)	38 (46.3%)	0.274
	Multiparity	37 (45.1%)	44 (53.7%)	
Education	Primary school	13 (15.9%)	10 (12.1%)	0.836
	Middle school	20 (24.4%)	24 (29.3%)	
	High school	26 (31.7%)	24 (29.3%)	
	University	23 (28.0%)	24 (29.3%)	
Previous surgery		17 (20.7%)	10 (12.2%)	0.140
Chronic disease		9 (11.0%)	5 (6.1%)	0.264
Smoking		2 (2.4%)	2 (2.4%)	1.00
Alcohol use		0 (0.0%)	0 (0.0%)	-
Steroid administration		5 (6.1%)	5 (6.1%)	1.00

P-values were calculated with the independent t test (height), Mann-Whitney U test (age, weight, BMI and diagnosis week) and chi-squared test.

Table 2: Comparison of laboratory results of groups

		Study group n=82 Mean (SD)	Control group n=82 Mean (SD)	*P*-value
Procalcitonin (ng/mL)		0.050 (0.013)	0.043 (0.013)	<0.001
Neutrophil (×10^9 /L)		6.02 (1.89)	6.28 (2.00)	0.429
Lymphocyte (×10^9 /L)		2.06 (0.63)	2.54 (2.66)	0.141
Platelet (×10^9 /L)		249.64 (54.35)	238.23 (51.34)	0.212
NLR		3.08 (1.16)	3.04 (1.42)	0.488
PLR		128.80 (35.56)	116.81 (45.56)	0.013
		n (%)	**n (%)**	
Ketones in urine	0	0 (0.0%)	82 (100.0%)	<0.001
	+1	41 (50.0%)	0 (0.0%)	
	+2	24 (29.3%)	0 (0.0%)	
	+3	17 (20.7%)	0 (0.0%)	

P-values were calculated with the Mann Whitney U test and chi-squared test (Ketones in urine).

Within the HG group, the severity of ketonuria was recorded as follows: 50.0% of patients had +1 ketones, 29.3% had +2, and 20.7% had +3. Birth weight and first-minute Apgar scores were significantly lower in pregnancies complicated by HG compared with healthy controls (*P*=0.013 and *P*=0.043, respectively) (Tables 3 and 4).

Table 3: Comparison of obstetric and neonatal results of the groups

		Study group n=82 Mean (SD)	Control group n=82 Mean (SD)	*P*-value
Infant weight (g)		3073.35 (509.45)	3252.68 (447.23)	0.013
Birth week (week)		38.38 (1.76)	38.79 (1.67)	0.112
1st minute Apgar scores		8.57 (0.68)	8.78 (0.52)	0.043
5th minute Apgar scores		9.76 (1.15)	9.80 (0.39)	0.544
		n (%)	n (%)	
Delivery type	Vaginal birth	50 (61.0%)	44 (53.7%)	0.344
	Cesarean	32 (39.0%)	38 (46.3%)	
Indications of cesarean	Vaginal birth	50 (61.0%)	44 (53.7%)	0.798
	Previous cesarean	16 (19.5%)	18 (22.0%)	
	Fetal distress	9 (11.0%)	12 (14.6%)	
	Cephalopelvic disproportion	5 (6.1%)	6 (7.3%)	
	Prolonged action	2 (2.4%)	1 (1.2%)	
	Ablation placenta	0 (0.0%)	1 (1.2%)	
Preterm birth		12 (14.6%)	7 (8.5%)	0.222
Intrauterine growth retardation		9 (11.0%)	4 (4.9%)	0.148
Low-birth-weight infants		9 (11.0%)	5 (6.1%)	0.264
Post-maturity		6 (7.3%)	6 (7.3%)	1.00
Neonatal intensive care needs		10 (12.2%)	4 (4.9%)	0.094

P-values were calculated with the Mann Whitney U test (infant weight, birth week, 1st minute Apgar scores and 5th minute Apgar scores) and chi-squared test. IUGR: Intrauterine growth retardation, RDS: Respiratory distress syndrome

Table 4: Comparison of the groups formed according to ketone levels in patients with hyperemesis gravidarum in terms of demographic characteristics, smoking, alcohol use, diagnosis week and steroid administration for lung development.

		+1 ketone in urine n=41 Mean (SD)	+2 ketone in urine n=24 Mean (SD)	+3 ketone in urine n=17 Mean (SD)	*P*-value
Age (year)		28.12 (4.69)	26.83 (5.16)	28.59 (5.20)	0.471
Weight (kg)		66.29 (16.04)	61.83 (11.96)	64.65 (12.49)	0.481
Height (cm)		161.71 (5.19)	160.17 (5.85)	160.82 (5.34)	0.533
BMI (kg/m^2)		25.36 (6.06)	24.15 (4.83)	24.99 (4.90)	0.695
Diagnosis week		10.03 (3.14)	9.52 (2.13)	10.01 (2.22)	0.747
		n (%)	n (%)	n (%)	
Parity	Nulliparity	23 (56.1%)	15 (62.5%)	7 (41.2%)	0.391
	Multiparity	18 (43.9%)	9 (37.5%)	10 (58.8%)	
Education	Primary school	4 (9.8%)	4 (16.7%)	5 (29.5%)	0.368
	Middle school	10 (24.4%)	6 (25.0%)	4 (23.5%)	
	High school	17 (41.4%)	5 (20.8%)	4 (23.5%)	
	University	10 (24.4%)	9 (37.5%)	4 (23.5%)	
Previous surgery		11 (26.8%)	3 (12.5%)	3 (17.6%)	0.365
Chronic disease		6 (14.6%)	1 (4.2%)	2 (11.8%)	0.425
Smoking		1 (2.4%)	1 (4.2%)	0 (0.0%)	0.696
Alcohol use		0 (0.0%)	0 (0.0%)	0 (0.0%)	-
Steroid administration		4 (9.8%)	0 (0.0%)	1 (5.9%)	0.284

P-values were calculated with the Kruskal Wallis and Chi-square test.

Subgroup analysis based on ketone severity revealed that serum PCT levels were highest in the +3 ketone subgroup, while no significant difference was noted between the +1 and +2 subgroups (*P*<0.001). Similarly, PLR values were significantly elevated in the +3 ketone subgroup (*P*=0.041). However, no statistically significant differences were observed among the ketone subgroups regarding other obstetric or neonatal outcomes (Tables 5 and 6).

Table 5: Comparison of laboratory results of groups formed according to ketone levels in patients with hyperemesis gravidarum

	+1 ketone in urine n=41 Mean (SD)	+2 ketone in urine n=24 Mean (SD)	+3 ketone in urine n=17 Mean (SD)	*P*-value
Procalcitonin (ng/mL)	0.045 (0.01)a	0.050 (0.00)a	0.063 (0.00)b	<0.001
Neutrophil (×10^9 /L)	5.99 (2.41)	6.22 (1.69)	5.82 (1.54)	0.794
Lymphocyte (×10^9 /L)	2.21 (0.72)a	1.97 (0.54)ab	1.79 (0.37)b	0.048
Platelet (×10^9 /L)	255.60 (63.60)	231.33 (37.52)	261.11 (45.67)	0.137
NLR	2.87 (1.32)	3.28 (1.06)	3.31 (0.83)	0.274
PLR	125.63 (44.12)a	120.87 (22.28)a	147.64 (18.04)b	0.041

P-values were calculated with the Kruskal Wallis test.

Table 6: Comparison of obstetric and neonatal outcomes of groups formed according to ketone levels in patients with hyperemesis gravidarum

	+1 ketone in urine n=41	+2 ketone in urine n=24	+3 ketone in urine n=17	*P*-value
Infant weight (g)	3176.59 (575.87)	2955.42 (390.42)	2990.88 (458.58)	0.182
Birth week (week)	38.29 (1.92)	38.55 (1.61)	38.34 (1.63)	0.856
1st minute Apgar scores	8.61 (0.66)	8.63 (0.64)	8.41 (0.79)	0.556
5th minute Apgar scores	9.76 (0.48)	9.88 (0.33)	9.59 (0.50)	0.145
	n (%)	**n (%)**	**n (%)**	***P*-value**
Delivery type				
Vaginal birth	29 (70.7%)	12 (50.0%)	9 (52.9%)	0.191
Cesarean	12 (29.3%)	12 (50.0%)	8 (47.1%	
Indications of cesarean				
Vaginal birth	30 (73.2%)	12 (50.0%)	8 (47.1%)	0.344
Previous cesarean	5 (12.2%)	6 (25.0%)	5 (29.4%)	
Fetal distress	5 (12.2%)	3 (12.5%)	1 (5.9%)	
Cephalopelvic disproportion	1 (2.4%)	2 (8.3%)	2 (11.8%)	
Prolonged action	0 (0.0%)	1 (4.2%)	1 (5.9%)	
Preterm birth	7 (17.1%)	3 (12.5%)	2 (11.8%)	0.821
Intrauterine growth retardation	4 (9.8%)	3 (12.5%)	2 (11.8%)	0.937
Low-birth-weight infants	5 (12.2%)	2 (8.3%)	2 (11.8%)	0.885
Post-maturity	2 (4.9%)	2 (8.3%)	2 (11.8%)	0.640
Neonatal intensive care needs	5 (12.2%)	1 (4.2%)	4 (23.5%)	0.175

P-values were calculated with the Kruskal Wallis and Chi-square test. a-b: There is no difference between groups with the same letters ($p>0.05$). Different letters indicate significantly different groups ($P<0.05$).

Discussion

The main findings of the present study indicate that serum PCT and PLR levels were significantly higher in patients with HG compared with healthy pregnant controls. Conversely, no significant difference was observed in the NLR. Subgroup analysis revealed that patients with +3 ketonuria exhibited significantly higher PCT and PLR values than those with lower degrees of ketonuria. Furthermore, infant birth weight and first-minute Apgar scores were significantly lower in the HG cohort, which aligns with previous reports demonstrating an association between HG and adverse perinatal outcomes [6, 7].

Several studies have reported elevated NLR and PLR values in women with HG, supporting their potential role as diagnostic or prognostic markers [9, 20, 25, 26]. Nevertheless, evidence regarding the association between ketonuria severity and clinical severity remains inconclusive. While some studies have demonstrated a significant correlation between ketonuria levels and clinical severity [9, 25], others have failed to identify such an association [20, 26]. Data investigating the specific relationship between PCT and HG are scarce, and the clinical significance of PCT in this context is not yet firmly established.

In recent years, growing attention has been directed toward inflammatory processes in the pathogenesis of HG [25, 27, 28]. Studies have demonstrated elevated proinflammatory cytokines, such as TNF-α and IL-6, and alterations in hematological indices in HG patients [9-11]. Furthermore, higher circulating levels of vaspin and elevated serum sirtuin-1 concentrations have been observed in these patients, further supporting the inflammatory hypothesis [10, 29].

PCT is a well-established biomarker of systemic inflammation, released by various cell types during severe inflammatory responses [30]. It has been used to identify preeclampsia severity and PPROM [17, 31]. While data on PCT in HG are limited, the hematological pattern of increased neutrophil counts and reduced lymphocyte counts—the basis for NLR and PLR—is frequently seen in systemic inflammation [32, 33]. These markers have been investigated extensively in oncology, cardiology, and reproductive medicine [21, 22, 34-38].

In accordance with previous reports, the present study demonstrated significantly higher PLR values in pregnant women with HG, although no significant difference was observed in the NLR. The association between elevated PLR and PCT levels with increasing severity of ketonuria suggests a potential link between inflammatory activity and clinical severity.

Consistent with prior documentation, our findings revealed significantly lower neonatal birth weights and 1-minute Apgar scores in the HG group [6, 7]. However, the lack of significant differences in outcomes between ketone subgroups may be due to the relatively small sample size. This limitation suggests that a larger cohort might provide greater statistical power for evaluating the relationship between inflammatory markers and specific perinatal outcomes. A major strength of this study is the simultaneous assessment of PCT, NLR, and PLR in conjunction with neonatal parameters, offering a comprehensive view of inflammatory mechanisms in HG.

Conclusion

In conclusion, the elevated serum PCT and PLR levels observed in women with HG support the hypothesis that inflammatory mechanisms contribute to the etiopathogenesis of the disorder. The association between increased inflammatory markers and ketonuria severity may provide clinically useful insights for predicting disease severity. While HG is linked to adverse neonatal outcomes such as lower birth weight and reduced 1-minute Apgar scores, these outcomes do not appear to be directly correlated with ketonuria levels.

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