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The Role of Systemic Inflammatory Indices in Predicting Postpartum Hemorrhage Following Cesarean Delivery

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ABSTRACT

Objective: To determine whether preoperative inflammation-related indices calculated from routine hematological parameters are associated with objectively measured postpartum hemorrhage (PPH) following cesarean section.

Methods: A retrospective cohort design was applied at a tertiary referral center and included 226 women who delivered by cesarean section between July 2025 and January 2026. Postpartum blood loss was calculated using a standardized formula-based approach. According to the quantified blood loss, patients were classified as <1000 mL or ≥1000 mL. We conducted a comparative analysis of preoperative inflammatory markers—specifically neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, monocyte-to-lymphocyte ratio, systemic immune-inflammation index (SII), systemic inflammatory response index, and pan-immune-inflammation value—across the study cohorts. Furthermore, the prognostic utility of these indices was assessed using receiver operating characteristic (ROC) curves, and multivariable logistic regression modeling was subsequently used to identify independent associations.

Results: Baseline maternal and obstetric characteristics did not differ significantly between the study groups. Among the investigated indices, only SII values were significantly higher in women with postpartum blood loss ≥1000 mL. SII demonstrated a weak but statistically significant positive correlation with total blood loss. ROC analysis yielded an area under the curve (AUC) of 0.625 for SII. After adjustment for relevant covariates, an SII threshold of ≥1175 remained independently associated with an increased risk of clinically significant PPH.

Conclusion: Although the discriminative ability of SII as a standalone test was modest (AUC=0.625), elevated preoperative SII levels were independently associated with increased postpartum blood loss after cesarean delivery. Therefore, SII may serve as an adjunctive biomarker that complements established clinical risk factors in preoperative risk stratification.

Keywords: Postpartum hemorrhage, term birth, inflammation, biomarkers, cesarean section, risk assessment

INTRODUCTION

Postpartum hemorrhage (PPH) continues to represent a major contributor to maternal morbidity and mortality worldwide, being responsible for nearly 27% of maternal deaths (1). The World Health Organization characterizes PPH as blood loss of ≥500 mL following delivery, while losses reaching or exceeding 1000 mL are classified as severe PPH (1). Against the backdrop of increasing cesarean delivery rates worldwide, perioperative bleeding has emerged as a major management challenge in contemporary obstetric practice. Despite well-established etiological factors such as uterine atony, genital tract trauma, and coagulopathy, a

considerable number of PPH cases have been observed in women without identifiable antenatal risk factors (2,3).

Labor is an inflammatory process characterized by myometrial infiltration of leukocytes, macrophages, and neutrophils that secrete proinflammatory cytokines (4). The bidirectional interplay between systemic inflammation and the coagulation system directly shapes hemostatic balance through the modulation of platelets, the endothelium, and the coagulation cascade by inflammatory mediators (4,5). Within this biological framework, it is postulated that the magnitude of the systemic inflammatory response may correspond to bleeding tendency during delivery;

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therefore, inflammatory markers could play a potential role in the assessment of postpartum bleeding risk (6,7).

In recent years, studies in the literature have demonstrated that inflammatory biomarkers based on routine hematological indices, including the neutrophil-to-lymphocyte ratio (NLR), possess prognostic relevance across a broad spectrum of obstetric pathologies (8,9). Furthermore, more contemporary composite indices—including the systemic immune-inflammation index (SII), the systemic inflammatory response index (SIRI), and the pan-immune-inflammation value (PIV)—reflect the inflammatory profile in a more integrated manner by allowing for the simultaneous evaluation of different immune cell series (10-12). Although these indices have been extensively investigated for their prognostic relevance in major obstetric conditions such as preeclampsia, data regarding their predictive performance for PPH risk among patients undergoing cesarean delivery remain limited. As a result, further studies are essential to validate the practical application of systemic inflammatory biomarkers, specifically SII, SIRI, and PIV.

The primary objective of this study is to evaluate the association between preoperatively measured complete blood count-based inflammatory indices and the objectively quantified amount of postpartum blood loss in women undergoing cesarean delivery.

METHODS

Study Design

The present investigation adopted a retrospective observational cohort design. No additional diagnostic or therapeutic procedures were implemented; the study was conducted entirely using data recorded during routine clinical practice. All study procedures were conducted in accordance with the ethical standards established by the Declaration of Helsinki. Ethical approval was granted by the Clinical Research Ethics Committee of the University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital (decision no: KAEK/28.01.2026.19, date: 03.02.2026).

Setting

This research was conducted at a single tertiary-care center in the University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital, Department of Obstetrics and Gynecology. The research cohort comprised patients who underwent cesarean deliveries at our center between July 2025 and January 2026. Obstetric management and follow-up for all cases were performed in accordance with our institution's established clinical standards.

Participants

The study population was selected from patients who delivered by cesarean section at our clinic during the study period. Inclusion criteria were defined as maternal age between 18-40 years, delivery occurring at ≥ 37 gestational weeks, and the presence of a singleton pregnancy. Cases of multiple pregnancies, obstetric complications (preeclampsia, gestational hypertension), chronic maternal diseases (e.g., pregestational diabetes), chorioamnionitis

with premature rupture of membranes, requirement for blood transfusion or management of massive hemorrhage, placental pathologies (previa, abruption), and missing clinical data were excluded from the analysis.

Variables

The primary outcome variable was the amount of postpartum bleeding. Independent variables included preoperative hematological inflammatory markers [NLR, platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), SII, SIRI, and PIV], total leukocyte count, coagulation parameters [prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR)], and demographic and obstetric factors [maternal age, body mass index (BMI), gravidity, parity, gestational week at delivery, and clinical characteristics related to birth].

Data Sources/Measurement

Relevant clinical data were retrieved retrospectively via the institution's digital registry and corresponding patient files. Preoperative laboratory parameters were extracted from complete blood counts and coagulation tests performed as part of the routine antepartum evaluation. The amount of postpartum blood loss was quantitatively calculated in milliliters using the Stafford formula, incorporating maternal height and weight as well as preoperative and 6-hour postoperative hematocrit values (13). The NLR, PLR, and MLR were calculated by determining the ratios of the respective cell parameters (neutrophils, lymphocytes, monocytes, and platelets). In addition, composite indices reflecting systemic inflammatory and immune status—SII, SIRI, and PIV—were calculated using the following formulas, respectively: $\text{neutrophils} \times \text{platelets} / \text{lymphocytes}$, $\text{neutrophils} \times \text{monocytes} / \text{lymphocytes}$, and $\text{neutrophils} \times \text{platelets} \times \text{monocytes} / \text{lymphocytes}$. Patients were stratified for analysis based on calculated postpartum blood loss into Group 1 (< 1000 mL) and Group 2 (≥ 1000 mL).

Bias

To minimize potential selection bias in the study design, all consecutive patients who presented to the clinic within the predefined time frame and met the eligibility criteria were included. To reduce information bias, the amount of blood loss was quantified using an objective formula based on laboratory and anthropometric parameters rather than visual estimation. Moreover, data reliability was enhanced by excluding patients who received blood transfusions or developed massive hemorrhage requiring surgical intervention, because these conditions could alter hematocrit concentrations and introduce error into the mathematical calculation. All laboratory measurements and clinical data were acquired at the same center using identical analytical methods and standard protocols for both groups. Finally, to limit confounding bias, conditions that could influence baseline levels of inflammatory biomarkers—such as infection, preeclampsia, and chronic systemic diseases—were excluded.

Study Size

To determine the necessary cohort size before the study onset, we performed a statistical power estimation using the G*Power package (v3.1, developed at Heinrich Heine University, Düsseldorf) (14). Based on data reported by Akay et al. (7), the effect size (Cohen's *d*) was set at 0.386, with a type I error rate (α) of 0.05 and a statistical power ($1-\beta$) of 0.80, yielding a required minimum sample size of 214 participants. During the retrospective screening process, 226 patients who met the study criteria (Group 1: 185; Group 2: 41) were included in the analysis, resulting in a sample size exceeding the predetermined requirement.

Statistical Analysis

Statistical computations were performed using IBM SPSS Statistics (v26.0; IBM Corp., Armonk, NY, USA). To verify whether continuous variables conformed to a Gaussian distribution, the Kolmogorov-Smirnov test was applied. For descriptive reporting, data exhibiting a Gaussian distribution were cited as mean $\pm$ standard deviation, while non-parametric data were summarized as medians (minimum-maximum). Between-group comparisons were conducted using Student's *t*-test or the Mann-Whitney *U* test, as appropriate, based on the data distribution. Categorical data were summarized as frequencies (*n*) and percentages (%), with intergroup differences evaluated via the chi-square test. The relationship between inflammatory indices and PPH was examined using Spearman's rank correlation analysis or Fisher's exact test, as appropriate. The discriminative performance of SII for predicting PPH (≥ 1000 mL) was assessed by receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) was reported with a 95% confidence interval (CI). The optimal cut-off value was determined using the Youden index. A multivariable binary logistic regression analysis was executed to determine independent risk factors for PPH (≥ 1000 mL). Parity, gestational age at delivery, and SII (≥ 1175) were included in the model based on their clinical relevance and potential association with PPH. The adequacy of the model was evaluated via the Hosmer-Lemeshow goodness-of-fit test. For all statistical comparisons, a two-sided $p < 0.05$ was considered statistically significant.

RESULTS

The final analytical cohort encompassed 226 eligible women who underwent cesarean delivery at our institution. Stratification of the study population revealed that 185 participants (81.8%) belonged to Group 1, whereas 41 (18.1%) were assigned to Group 2. As no missing data were identified, all statistical analyses were performed on this final study cohort.

The baseline demographic and obstetric characteristics of the study groups are presented comparatively in Table 1. No statistically significant differences were detected between Group 1 and Group 2 regarding maternal age, BMI, gravidity, parity, and the gestational week at delivery (all $p > 0.05$). Similarly, the groups demonstrated comparable distributions concerning birth weight, rates of primary cesarean delivery, presence of labor induction, and the proportion of cesarean deliveries performed during the active phase of labor (all $p > 0.05$).

In the evaluation of preoperative laboratory parameters and inflammatory indices, no statistically significant differences were observed between the groups with respect to complete blood count components (hemoglobin, hematocrit, leukocyte, platelet, neutrophil, lymphocyte, and monocyte counts) or coagulation tests (PT, aPTT, and INR) (all $p > 0.05$). Likewise, no statistically significant variations were detected between the cohorts regarding the inflammatory markers NLR, PLR, MLR, SIRI, and PIV (all $p > 0.05$). In contrast, SII levels were significantly higher in Group 2 compared with Group 1 (median 1229.40 vs. 969.33, $p = 0.012$) (Table 2).

The relationship between inflammatory indices and the amount of postpartum blood loss was examined using Spearman correlation analysis. A weak but statistically significant positive correlation was identified between the amount of postpartum blood loss and SII levels among the inflammatory indices ($r = 0.211$, $p < 0.001$). ROC curve analysis was subsequently conducted to evaluate the discriminative capacity of SII for clinically significant PPH (≥ 1000 mL) (Figure 1). The analysis yielded an AUC value of 0.625 for SII (95% CI: 0.519-0.731; $p = 0.012$). The optimal cut-off value determined by the Youden index was 1175, yielding a sensitivity of 58.5% and a specificity of 77.8% (Table 3).

Table 1. Comparison of demographic and obstetric characteristics of the study groups

	Group 1 (n=185)	Group 2 (n=41)	p
Maternal age ^b (years)	28 (18-40)	30 (19-40)	0.869
BMI ^b (kg/m ²)	30.4 (20.2-49.3)	30.4 (23.7-44.3)	0.890
Gravidity ^b	2 (1-8)	3 (1-8)	0.502
Parity ^b	1 (0-6)	1 (0-6)	0.108
GA at delivery (weeks) ^b	38.7 (37.0-41.0)	38.9 (37.0-41.0)	0.204
Primary cesarean section ^c	102 (55.1%)	25 (61.0%)	0.495
Birth weight ^a (g)	3106.86 $\pm$ 511.75	3133.90 $\pm$ 584.60	0.766
Labor induction ^c	61 (33.0%)	15 (36.6%)	0.658
Active phase of labor ^c	17 (9.2%)	3 (7.3%)	1.000

^a: Normal distribution, mean $\pm$ standard deviation, ^b: Non-normal distribution, median (minimum-maximum), ^c: Categorical data, number (percentage %), BMI: Body mass index, GA: Gestational age

Table 2. Comparison of preoperative laboratory parameters and systemic inflammatory indices between the study groups

	Group 1 (n=185)	Group 2 (n=41)	p
Hb ^b (g/dL)	11.6 (8.7-15.0)	12.0 (8.9-13.7)	0.348
HCT ^b (%)	35.1 (27.8-44.3)	36.6 (30.7-39.3)	0.164
WBC ^b (10 ⁹ /L)	10.31 (5.23-18.56)	10.26 (6.03-18.50)	0.741
PLT ^b (10 ⁹ /L)	250 (117-597)	241 (82-597)	0.827
Neutrophil ^b (10 ⁹ /L)	7.70 (2.23-18.68)	7.95 (3.06-19.78)	0.330
Lymphocyte ^b (10 ⁹ /L)	2.13 (0.65-7.90)	2.14 (0.90-20.20)	0.858
Monocyte ^b (10 ⁹ /L)	0.74 (0.26-1.66)	0.70 (0.40-6.00)	0.168
PT ^b (s)	8.4 (7.5-10.5)	8.4 (7.6-9.6)	0.818
aPTT ^a (s)	27.41±2.95	27.20±3.87	0.692
INR ^b	0.9 (0.8-1.1)	0.9 (0.8-1.1)	0.797
NLR ^b	3.60 (1.33-13.09)	3.44 (0.47-17.43)	0.882
PLR ^b	119.39 (28.99-567.14)	108.89 (7.03-412.31)	0.593
MLR ^b	0.34 (0.10-1.76)	0.29 (0.13-1.09)	0.374
SII ^b	969.33 (316.27-3050.51)	1229.40 (66.36-1790.77)	0.012
SIRI ^b	2.56 (0.58-12.09)	2.48 (0.81-13.83)	0.674
PIV ^b	691.90 (158.14-3542.48)	791.42 (137.08-1934.03)	0.663

Bold values indicate statistical significance ($p < 0.05$), ^a: Normal distribution, mean $\pm$ standard deviation, ^b: Non-normal distribution, median (minimum-maximum), Hb: Hemoglobin, HCT: Hematocrit, WBC: White blood cell count, PLT: Platelet count, PT: Prothrombin time, aPTT: Activated partial thromboplastin time, INR: International normalized ratio, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, MLR: Monocyte-to-lymphocyte ratio, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index, PIV: Pan-immune-inflammation value

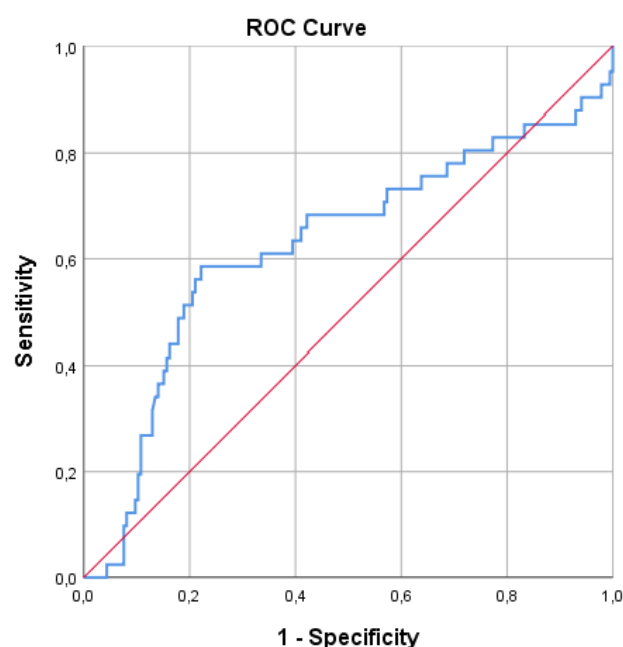


Figure 1. Receiver operating characteristic (ROC) curve illustrating the discriminative performance of the systemic immune-inflammation index for predicting postpartum hemorrhage ≥ 1000 mL after cesarean section. The area under the curve was 0.625 (95% confidence interval: 0.519-0.731; $p = 0.012$)

We executed a multivariable binary logistic regression analysis to determine the independent predictors of PPH (≥ 1000 mL) (Table 4). The analysis demonstrated that neither parity nor gestational age at delivery was significantly associated with PPH (≥ 1000 mL) ($p = 0.153$ and $p = 0.186$, respectively). However, an SII level above the defined cut-off (≥ 1175) was independently associated with a statistically significant increase in the risk of PPH (≥ 1000 mL) (adjusted odds ratio: 4.887; 95% CI: 2.378-10.043; $p < 0.001$). The model showed good fit, as confirmed by the Hosmer-Lemeshow goodness-of-fit test ($p = 0.087$).

DISCUSSION

Principal Findings

This study examined the relationship between preoperative complete blood count-based inflammatory indices and objectively quantified postpartum blood loss among women undergoing cesarean delivery. The principal observation was that while routine hemogram parameters and other derived inflammatory indices did not differ between the groups, preoperative SII levels were significantly elevated in patients who developed PPH. Furthermore, our analysis unveiled a significant positive linear relationship linking preoperative SII concentrations directly to the volume of PPH (≥ 1000 mL). In ROC curve analysis, SII yielded an AUC of 0.625, while multivariable logistic regression revealed an independent association between elevated SII levels and PPH (≥ 1000 mL).

Table 3. ROC analysis of the SII for predicting postpartum hemorrhage ≥ 1000 mL

	Cut-off	AUC	95% CI	Sensitivity (%)	Specificity (%)	p
SII	1175	0.625	0.519-0.731	58.5	77.8	0.012

ROC: Receiver operating characteristic, SII: Systemic immune-inflammation index, AUC: Area under the curve, CI: Confidence interval

Table 4. Multivariate logistic regression analysis of independent predictors for postpartum hemorrhage ≥ 1000 mL

Variable	B	Adjusted OR [Exp(B)]	95% CI for OR	p
Parity	0.145	1.156	0.947-1.411	0.153
GA at delivery (weeks)	0.209	1.232	0.904-1.679	0.186
High SII (≥ 1175)	1.587	4.887	2.378-10.043	<0.001

Note: The multivariate model was adjusted for parity and gestational age. The cut-off value for SII was determined using the Youden index derived from the receiver operating characteristic curve analysis. The model showed good fit (Hosmer-Lemeshow goodness-of-fit test, $p=0.087$). Bold value indicate statistical significance ($p<0.05$)

B: Regression coefficient, OR: Odds ratio, CI: Confidence interval, GA: Gestational age, SII: Systemic immune-inflammation index

Interpretation

The primary observation of this study is that elevated preoperative SII values are associated with the development of clinically significant hemorrhage following cesarean section. A review of the literature examining the role of inflammatory indices in predicting PPH risk indicates that no clear consensus exists regarding the prognostic value of SII. In a cohort involving placental pathologies, SIRI and PLR were reported to have the strongest predictive value for intraoperative blood loss, whereas NLR and SII showed significant increases in cases of massive hemorrhage (15). Likewise, a recent study reported that elevated NLR, PLR, and SII levels were important prognostic markers for predicting composite adverse outcomes in severe PPH requiring fibrinogen replacement therapy (6). In contrast, Akay et al. (7) observed significantly lower SII levels in patients who developed PPH compared with the control group. This heterogeneity in the literature may stem from several factors, including variations in PPH definitions, sample selection (particularly inclusion of cases requiring massive transfusion or surgical intervention), and timing of measurements. In addition, concomitant clinical conditions that modify the inflammatory response may contribute.

The observation that SII emerged as the only significant biomarker, despite the lack of association between other inflammatory indices and PPH, may be explained by differences in the underlying cellular mechanisms upon which these indices are based. Ratios derived from three cell lineages—namely NLR, PLR, and MLR—may have failed to yield significant results due to their limited biological representativeness during the peripartum period, characterized by substantial physiological variability. SIRI, which does not incorporate platelets, and PIV, which combines multiple cellular parameters into a single formula, may inadequately capture the obstetric bleeding phenotype, thereby diluting the biological signal. Conversely, SII excludes cell lineages such as monocytes, which are typically associated with delayed responses or chronic processes, and directly reflects both inflammatory activity and platelet-mediated hemostatic capacity (10). Consequently, the

distinct performance of SII compared with other indices appears to be related to its specific structure, which effectively integrates acute inflammation and hemostatic processes, suggesting that SII is a sensitive marker for assessing PPH risk.

The establishment of hemostasis in the postpartum period depends not only on mechanical uterine contractions but also on local thrombotic mechanisms involving tissue factor activation and systemic coagulation components (16,17). It has been suggested that increased inflammatory cell infiltration and cytokine release during the peripartum period may adversely affect myometrial contractility, thereby predisposing to the development of uterine atony (18,19). In other words, despite the absence of an identifiable microbial pathogen during this period, the myometrial tissue is dominated by an acute myometritis pattern characterized by massive cellular infiltration rich in neutrophils, macrophages, and mast cells (20). Additionally, histopathological examinations of cases with refractory PPH of unknown etiology following cesarean delivery have revealed that inflammatory and anaphylactoid reactions involve not only the uterine corpus but also the isthmus (21). Mandel et al. (22) further highlighted the regulatory role of platelets within the inflammation-coagulation axis, noting that platelet function may be compromised independently of platelet count under conditions of increased inflammatory activity. Taken together, data from the literature explain the pathophysiological basis by which SII, reflecting both inflammation and hemostasis, emerges as a more sensitive biomarker for predicting PPH risk than other inflammatory indices.

Clinically, a key finding of this study is that elevated SII levels are independently and significantly associated with postpartum blood loss. However, the modest but statistically significant discriminative power indicated by ROC analysis supports positioning SII not as a standalone diagnostic test but rather as an adjunctive parameter for preoperative risk stratification. As emphasized in numerous studies in the literature, the low cost and widespread availability of these hematological indices represent a major advantage, given their potential to provide complementary

value in clinical assessment (23,24). For clinical interpretation, SII values ≥ 1175 may help identify patients who could benefit from increased perioperative vigilance and preventive strategies for PPH. In light of the current findings, incorporating SII alongside established clinical risk factors may allow PPH risk to be addressed through a more holistic strategy and may enhance the predictive strength of preoperative evaluation.

Study Limitations

A notable strength of the present study is the quantitative assessment of postpartum blood loss, as opposed to reliance on subjective estimation. This approach reduces measurement bias, thereby enhancing the internal validity of the study and the reliability of the obtained results. Another notable strength is the exclusion of clinical conditions that could substantially influence the inflammatory profile, thereby limiting the impact of potential confounding factors. This strategy ensured a homogeneous cohort, allowing for a more robust and comprehensive assessment of the association between inflammatory indices and PPH.

Nevertheless, certain limitations of the present investigation warrant consideration. First, the retrospective study design restricts the ability to draw definitive causal inferences. Second, the single-institution design may limit the extent to which these results can be extrapolated to broader, more diverse patient populations. Another limitation is the reliance on measurements obtained at a single preoperative time point, which precludes the evaluation of dynamic fluctuations in inflammatory indices throughout pregnancy. The modest number of patients with clinically significant PPH may have constrained the statistical robustness of subgroup analyses. We did not measure all biochemical or cytokine markers that modulate the inflammatory response. Therefore, we cannot definitively exclude the presence of residual confounding factors. Therefore, future large-scale, multicenter, prospective studies are warranted to more comprehensively evaluate the clinical utility of inflammatory indices in predicting the risk of PPH.

CONCLUSION

This study demonstrates that elevated SII levels constitute an independent and significant risk factor for the development of PPH (≥ 1000 mL) in women undergoing cesarean delivery. However, given its modest discriminative ability ($AUC=0.625$), SII should be interpreted as an adjunctive biomarker rather than a standalone predictor. That SII emerged as the sole marker retaining statistical significance, in contrast to other inflammatory indices, underscores its potential clinical relevance. In women with elevated SII levels, optimizing perioperative preparation and implementing proactive preventive measures may substantially reduce maternal morbidity.

Ethics

Ethics Committee Approval: Ethical approval was granted by the Clinical Research Ethics Committee of the University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital (decision no: KAEK/28.01.2026.19, date: 03.02.2026).

Informed Consent: Due to the retrospective nature of the study, the requirement for informed consent was waived. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Footnotes

Author Contributions: Surgical and Medical Practices - Y.K., E.D.; Concept - Y.K., E.D.; Design - Y.K., E.D.; Data Collection and/or Processing - Y.K., İ.Y., E.A., S.C.; Analysis and/or Interpretation - Y.K., E.A., S.C.; Literature Search - Y.K., E.D., İ.Y., E.A., S.C.; Writing - Y.K., İ.Y.

Conflict of Interest: The authors have no conflicts of interest to declare.

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REFERENCES

1. WHO Guidelines Approved by the Guidelines Review Committee. WHO recommendations on the assessment of postpartum blood loss and use of a treatment bundle for postpartum haemorrhage. Geneva: World Health Organization; 2023.
2. McIntock C. Prevention and treatment of postpartum hemorrhage: focus on hematological aspects of management. Hematology Am Soc Hematol Educ Program. 2020; 2020: 542-6.
3. Dağdeviren E, Tercan C, Yeniocak AS, Sarı N, Keskin ŞB, Yaman İ. Platelet indices as predictors of hemoglobin decline following cesarean section: a retrospective cohort analysis. Z Geburtshilfe Neonatol. 2026; 230: 122-8.
4. Jiang H, Shi H, Chen L, Yang J, Yuan P, Wang W, et al. Is there a relationship between plasma, cytokine concentrations, and the subsequent risk of postpartum hemorrhage? Am J Obstet Gynecol. 2022; 226: 835.e1-17.
5. Tercan C, Yeniocak AS, Dağdeviren E, Can S, İnce A, Azman MK, et al. Evaluating platelet indices, inflammatory markers, and the systemic immune-inflammation index as predictors of erythrocyte suspension transfusion in total abdominal hysterectomy: a matched cohort study. Kocaeli Medical Journal. 2025; 14: 91-8.
6. Tokalioglu EO, Tanacan A, Agaoglu MO, Özkavak O, Atalay A, Uzuner P, et al. The role of systemic inflammatory indices in predicting composite adverse outcomes of peripartum hemorrhage treated with fibrinogen concentrate. Indian J Hematol Blood Transfus. 2025; 41: 934-41.
7. Akay E, Şirin Donbalıoğlu G, Yeniocak AS, Dağdeviren E, Tercan C, Polat İ. Prognostic evaluation of systemic immune inflammatory index and hematological parameters in postpartum hemorrhage: a retrospective analysis. Ginekoloj. 2025; 96: 287-93.
8. Gabbai D, Gilboa I, Lavie A, Yogev Y, Attali E. Neutrophil-to-lymphocyte ratio at admission helps to predict the need for blood transfusion after vaginal delivery. Int J Gynaecol Obstet. 2026; 173: 1489-95.
9. Özkan Y, Genç S, Kaya Y, Kaya DY, Mihmanlı V. Evaluation of neutrophil/lymphocyte and thrombocyte/lymphocyte ratios in cases of preterm premature rupture of membranes. Bagcilar Med Bull. 2024; 9: 9-14.
10. Hu B, Yang XR, Xu Y, Sun YF, Sun C, Guo W, et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. Clin Cancer Res. 2014; 20: 6212-22.
11. Qi Q, Zhuang L, Shen Y, Geng Y, Yu S, Chen H, et al. A novel systemic inflammation response index (SIRI) for predicting the survival of patients with pancreatic cancer after chemotherapy. Cancer. 2016; 122: 2158-67.
12. Lin F, Zhang LP, Xie SY, Huang HY, Chen XY, Jiang TC, et al. Pan-immune-inflammation value: a new prognostic index in operative breast cancer. Front Oncol. 2022; 12: 830138.
13. Stafford I, Dildy GA, Clark SL, Belfort MA. Visually estimated and calculated blood loss in vaginal and cesarean delivery. Am J Obstet Gynecol. 2008; 199: 519.e1-7.
14. Faul F, Erdfelder E, Buchner A, Lang AG. Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. Behav Res Methods. 2009; 41: 1149-60.
15. Balkaş G, Çelen Ş. Role of inflammatory and coagulation biomarkers in distinguishing placenta accreta from placenta previa and associated hemorrhage. J Clin Med. 2025; 14: 3884.
16. Warren BB, Moyer GC, Manco-Johnson MJ. Hemostasis in the pregnant woman, the placenta, the fetus, and the newborn infant. Semin Thromb Hemost. 2023; 49: 319-29.

17. Edwards HM. Aetiology and treatment of severe postpartum haemorrhage. *Dan Med J*. 2018; 65: B5444.
18. Shynlova O, Lee YH, Srikhajon K, Lye SJ. Physiologic uterine inflammation and labor onset: integration of endocrine and mechanical signals. *Reprod Sci*. 2013; 20: 154-67.
19. Zhang P, Jia Y, Song H, Fan Y, Lv Y, Geng H, et al. Novel biomarkers for prediction of atonic postpartum hemorrhage among 'low-risk' women in labor. *Front Immunol*. 2024; 15: 1416990.
20. Oda T, Tamura N, Ide R, Kawai K, Narumi M, Matsumoto M, et al. Postpartum acute myometritis suppresses expression of contraction-associated proteins in the gravid uterus. *J Reprod Immunol*. 2024; 165: 104299.
21. Jain D, Oda T, Kohmura-Kobayashi Y, Furuta-Isomura N, Yaguchi C, Uchida T, et al. Acute inflammation in the uterine isthmus coincides with postpartum acute myometritis in the uterine body involving refractory postpartum hemorrhage of unknown etiology after cesarean delivery. *J Reprod Immunol*. 2020; 139: 103116.
22. Mandel J, Casari M, Stepanyan M, Martyanov A, Deppermann C. Beyond hemostasis: platelet innate immune interactions and thromboinflammation. *Int J Mol Sci*. 2022; 23: 3868.
23. Cicek S, Coskun EI, Abdurahmanova N, Yilmaz E. Inflammation-driven pathogenesis of hyperemesis gravidarum: assessing the role of pan-immune-inflammation value and hematologic parameters. *Arch Gynecol Obstet*. 2025; 312: 2001-10.
24. Yang S, Shi H, Ren S, Chen Y, Jin X, Han L, et al. Association of inflammatory markers with pregnancy loss: Global Burden (1990-2021) and NHANES survey (2005-2016). *BMC Pregnancy Childbirth*. 2025; 25: 996.