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Bidirectional Prognostic Value of Platelet -to-Lymphocyte Ratio in Critically Ill Patients After Major Abdominal Surgery

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Abstract

Background: Patients admitted to the intensive care unit (ICU) after major abdominal surgery are at high risk for gastrointestinal complications and unplanned hospital readmission. However, reliable and easily applicable biomarkers for early risk stratification are limited. This study evaluated the prognostic value of serial platelet-to-lymphocyte ratio (PLR) measurements in critically ill postoperative patients. **Method**: This retrospective observational study included adults admitted to the ICU after major abdominal surgery at a tertiary referral center between July 2020 and March 2025. The PLR was calculated from routine complete blood counts obtained at ICU admission and 24, 48, and 72 hours postoperatively. Multivariate logistic regression analysis was performed to identify the independent predictors of major gastrointestinal complications and hospital readmission rate. **Results**: Among 962 critically ill postoperative patients, nine (0.9%) developed major gastrointestinal complications. These patients had significantly higher disease severity scores and elevated PLRs at 72 hours postoperatively. In multivariate analysis, the PLR at 72 hours was an independent predictor of complications (odds ratio [OR] = 1.006; 95% confidence interval [CI]: 1.002–1.009). Conversely, a lower PLR at the same time point was independently associated with an increased risk of hospital readmission (OR = 0.993; 95% CI: 0.987–0.998). **Conclusions**: The postoperative PLR at 72 hours showed bidirectional prognostic value in critically ill patients after major abdominal surgery. Elevated PLRs were associated with major gastrointestinal complications, whereas lower PLRs were associated with hospital readmission. Thus, the PLR may serve as a practical biomarker for postoperative risk stratification and discharge decision-making in the ICU.

Keywords: platelet-to-lymphocyte ratio; critical illness; Intensive care unit; postoperative complications

1. Introduction

Patients admitted to the intensive care unit (ICU) after major abdominal surgery have often undergone extensive procedures involving the resection of intra-abdominal organs, vascular structures, and technically complex anastomoses. Thus, they are at high risk of postoperative major gastrointestinal complications (MGCs), such as severe ileus and gastrointestinal (GI) bleeding. Among patients who exhibit hemodynamic instability immediately after surgery, these complications are strongly associated with prolonged recovery periods, delays in the initiation of subsequent treatments, and poorer long-term outcomes [1–4].

Therefore, the early identification of patients at high risk for postoperative complications is crucial for timely intervention and improved outcomes [3,4]. In particular, the availability of reliable early predictors of postoperative complications in critically ill patients after major surgery is considered a key element in optimizing risk management and guiding clinical decision-making. Such

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predictors may also support discharge decisions and potentially reduce unplanned hospital readmission, the inefficient use of healthcare resources, and socioeconomic burden on patients.

Among the parameters derived from the complete blood count (CBC), the platelet-to-lymphocyte ratio (PLR) has been proposed as a potential predictor of acute abdominal conditions and postoperative complications following major abdominal surgery [5]. The PLR can be easily and repeatedly measured in most hospital laboratories, and it has recently gained increasing attention as a useful prognostic indicator in a wide range of diseases [6–10]. Accumulating evidence suggests that platelets and lymphocytes play important regulatory roles in immune responses, coagulation pathways, and systemic inflammation [11]. Elevated PLRs have been associated with greater disease severity, enhanced interaction between inflammation and coagulation, and increased platelet activation, potentially mediated by endothelial dysfunction [12]. Increases in PLR levels on postoperative days 1 and 3 have been significantly associated with delayed recovery after laparoscopic colectomy surgery [13]. However, the clinical utility of PLR for predicting MGCs and supporting discharge decisions remains unclear. Therefore, we aimed to evaluate the prognostic value of serial PLR measurements for predicting postoperative MGCs and unplanned hospital readmission in critically ill patients after major abdominal surgery.

2. Materials and Methods

2.1. Study Design and Patient Enrollment

This study was conducted in the ICU of a tertiary referral hospital between July 2020 and March 2025. Clinical data were collected prospectively and analyzed retrospectively. Adult patients aged 18 years or older who were admitted to the ICU for postoperative care after major abdominal surgery were eligible for inclusion. Patients were excluded from the analysis if they met any of the following criteria: (1) age under 18 years, (2) pregnant, (3) death within 48 hours after surgery, (4) vascular procedures performed solely under local anesthesia, or (5) ICU admission for medical treatment without undergoing surgery.

Patients were primarily classified according to the occurrence of MGCs during hospitalization. Demographic characteristics, hemodynamic parameters, and clinical outcomes were compared between the two groups. Hospital readmission after discharge was analyzed as a secondary outcome. All patients underwent routine laboratory testing, including CBCs, liver function tests, coagulation profiles, and serum electrolyte measurements, both preoperatively and daily throughout the postoperative period. In patients undergoing elective surgery, prophylactic antibiotics were administered 30 minutes prior to the anesthesia induction and continued for up to 3 days postoperatively. In cases of emergency surgery, such as those due to panperitonitis, antibiotics were initiated immediately upon diagnosis, and the duration of antibiotic administration was determined according to each patient's postoperative course. Abdominal drains were placed when clinically indicated in accordance with institutional protocols. The timing of drain removal was determined on a case-by-case basis according to the characteristics, color, and volume of the drainage fluid, and the patient's overall clinical condition.

Hospital discharge decisions were based on a comprehensive clinical assessment. Patients were considered eligible for discharge when vital signs and laboratory parameters were stable, they could perform daily activities independently, and their overall condition was deemed acceptable by the attending physician. Additionally, patients were required to tolerate more than half of the prescribed diet and show no evidence of postoperative complications, such as infection, bleeding, or anastomotic leakage. No issues related to surgical drains or wound sites could be present. Patient discharge was permitted when their pain was adequately controlled, defined as a visual analog scale score of 3 or lower, with no additional discomfort reported. Finally, discharge was approved only when the final imaging studies showed no clinically significant abnormalities.

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This study was approved by the Institutional Review Board of The Catholic University of Korea, Seoul St. Mary's Hospital (IRB No. KC26RISI0097) and adhered to the Declaration of Helsinki and its later amendments.

2.2. Measurement of Complete Blood Count Parameters

Blood sampling was performed at four predefined time points: immediately upon ICU admission after surgery (T0), and 24 hours (T1), 48 hours (T2), and 72 hours (T3) postoperatively. All enrolled patients had a central venous catheter in place, which was used for blood collection to minimize patient discomfort and ensure sampling consistency. Blood samples were obtained by well-trained medical personnel in accordance with standardized institutional protocols to maintain sample integrity and minimize the risk of contamination. A venous blood sample collected in tubes containing an anticoagulant, typically ethylenediaminetetraacetic acid, was used for CBCs [14]. After collection, the samples were gently mixed to prevent clot formation and analyzed using an automated hematology analyzer [15]. These analyzers use either electrical impedance or optical/laser-based flow cytometry to count and classify blood cells.

The key hematologic parameters measured included the total WBC, red blood cell count, hemoglobin concentration, hematocrit, platelet count, and red cell indices, such as mean corpuscular volume, mean corpuscular hemoglobin, and red cell distribution width [16]. WBC differentials, including neutrophils, lymphocytes, monocytes, eosinophils, and basophils, were measured using automated analyzers and confirmed by microscopic examination when clinically indicated [17]. Routine quality control procedures were applied to ensure the accuracy and reproducibility of the laboratory results. The PLR, neutrophil-to-lymphocyte ratio (NLR), and monocyte-to-lymphocyte ratio (MLR) were calculated using absolute cell counts as follows:

PLR (platelet-to-lymphocyte ratio) = platelet count/lymphocyte count

NLR (neutrophil-to-lymphocyte ratio) = neutrophil count/lymphocyte count

MLR (monocyte-to-lymphocyte ratio) = monocyte count/lymphocyte count

2.3. Data collection and Study Outcomes

Electronic medical records were used to prospectively collect clinical data and were retrospectively analyzed. The acquired information included demographic characteristics, underlying diseases, and disease severity assessed using Acute Physiology and Chronic Health Evaluation II (APACHE II) scores at ICU admission. Organ dysfunction was evaluated daily using Sequential Organ Failure Assessment (SOFA) scores, starting from the time of ICU admission.

Hemodynamic and inflammatory parameters such as C-reactive protein or lactate levels were assessed using daily laboratory measurements, including CBCs and other inflammatory markers. Postoperative morbidity was monitored throughout the initial hospitalization and recorded for clinical outcome evaluation. Complications were categorized according to the Clavien–Dindo classification [18], and only complications of grade III or higher were included in the analysis. Grade III complications were defined as those requiring therapeutic intervention through endoscopic, radiological, or surgical procedures. Grade IV complications were defined as life-threatening events requiring ICU management due to single- or multi-organ dysfunction, while grade V represented postoperative mortality.

2.3.1. Major Gastrointestinal Complications

Postoperative MGCs were defined as clinically significant adverse events involving the GI tract that occurred after surgery and required therapeutic intervention, including surgical, endoscopic, or radiologic intervention. These included conditions such as postoperative ileus and postoperative GI bleeding. Postoperative ileus was defined as delayed bowel motility leading to abdominal distension,

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nausea, vomiting, and the inability to tolerate oral intake. Conditions were classified as an MGC only when persistent bowel dilatation was confirmed by radiologic imaging (X-ray or computed tomography (CT)) and conservative management failed. The failure of conservative management was defined as the progressive worsening of abdominal pain despite fasting and nasogastric decompression with a Levin tube. Patients requiring endoscopic, interventional radiologic, or surgical procedures were included in this definition. Postoperative GI bleeding, manifested by hematemesis, melena, or hematochezia, was classified as an MGC only when diagnostic evaluations confirmed hemorrhage. It was defined as bleeding corresponding to at least Advanced Trauma Life Support class II, indicating clinically significant blood loss with hemodynamic alterations such as tachycardia, narrowed pulse pressure, and reduced urine output [19]. Cases requiring active therapeutic intervention, including blood transfusion, endoscopic hemostasis, interventional radiology, or surgical procedures, were included in this definition.

2.3.2. Infectious Complications

Postoperative infectious complications were defined as infections occurring during the postoperative period that required antimicrobial therapy or interventional procedures, supported by clinical signs, radiological findings, or positive microbiological culture results [20].

2.3.3. Intra-Abdominal Fluid Collection

Intra-abdominal fluid collection was defined as sterile or infected fluid accumulation within the abdominal cavity. A non-abscess fluid collection was defined as an accumulation measuring ≥ 3 cm in any dimension (length, width, or depth) detected on CT or ultrasonography without clinical or microbiological evidence of infection. In contrast, an intra-abdominal abscess was defined as an imaging-confirmed fluid collection accompanied by positive microbiological cultures obtained from percutaneous drainage or surgical intervention [21].

2.3.4. Major Gastrointestinal Complications

Pleural effusion was diagnosed based on the presence of interlobar fluid accumulation or blunting of the costophrenic angle observed on chest radiography and determined by comparing preoperative and postoperative images [22]. Chest X-rays were obtained with the patient in a semi-upright position whenever feasible. Additionally, routine bedside ultrasonography was performed in accordance with institutional protocols to evaluate pleural effusion as part of volume status assessment.

The treatment course and duration of mechanical ventilation and continuous renal replacement therapy (CRRT) were recorded for applicable patients. The length of ICU stays, postoperative length of hospital stays, and total hospital stays were also documented.

The primary outcome was the association between PLRs and the occurrence of MGCs in critically ill patients. The secondary outcome was the comparison of hospital readmission according to postoperative PLRs.

2.4. Statistical Analysis

All statistical analyses were conducted using the SPSS statistical package software (version 28.0 for Windows; SPSS, Inc, Chicago, IL, USA). The patients were divided into two groups based on MGC occurrence. Demographic characteristics, hemodynamic parameters, and clinical outcomes were compared between the two groups. Continuous variables are presented as mean ± standard deviation, and overall differences were evaluated using the Student's t-test or analysis of variance. The normal distribution of variables was assessed using the Kolmogorov–Smirnov test. Variables that were not normally distributed were analyzed using the Mann–Whitney U test. Categorical variables were analyzed using Fisher's exact test or the chi-squared ($\chi 2$) test. The relationship between PLRs and various parameters was assessed using partial correlation analysis, with

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adjustments for age, sex, and body mass index (BMI) to account for their potential confounding effects. These variables were included as covariates in the partial correlation analysis, as these demographic and anthropometric factors are known to influence platelet and lymphocyte counts as well as systemic inflammatory status, and thus may act as potential confounders in the relationship between PLRs and clinical outcomes. Differences were considered statistically significant for p-values of < 0.05. Multivariate logistic regression analysis was performed to identify predisposing factors for MGCs and hospital readmission using variables found to be significant in the univariate analysis.

3. Results

During the study period, a total of 2,183 patients were admitted to the ICU. Of these, 962 patients were included in the analysis (Figure 1). The mean age of the study population was 68.1 years, and 537 (55.8%) were male. The most frequently performed surgery was hepatobiliary surgery, accounting for 392 cases (40.8%). Among the 962 patients, nine (0.9%) developed MGCs during their ICU stay and 953 (99.1%) did not (Table 1). The participants were divided into the MGC group (n = 9, 0.9%) and the no-MGC group (n = 953, 99.1%), and their demographic characteristics, hemodynamic parameters, and clinical outcomes were compared. No significant differences were found between the two groups in terms of age, sex, or BMI. However, baseline disease severity, assessed by the total SOFA score at T0, was significantly higher in the MGC group. Similarly, APACHE II scores were significantly higher in patients who developed MGC. Regarding hemodynamic parameters, lactate levels at T1 were significantly higher in the MGC group, whereas no differences were observed at T0, T2, or T3. Among inflammatory and hematologic markers, the PLR at T3 and C-reactive protein levels at T3 were significantly higher in the MGC group. In contrast, the NLR, MLR, procalcitonin (PCT), and presepsin levels measured at both T0 and T3 showed no significant differences between the two groups.

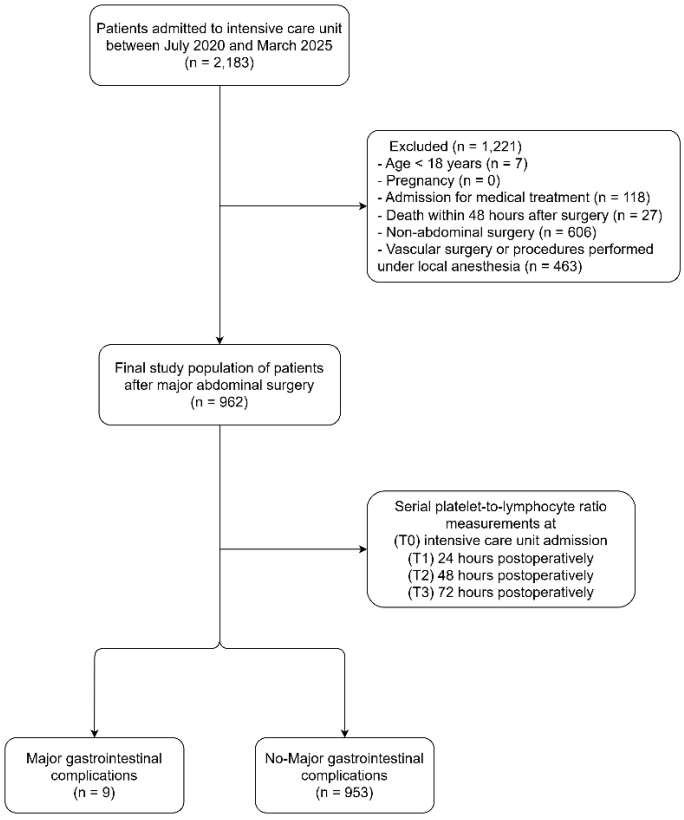

Figure 1. Flow diagram of patient enrollment and study design.

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Regarding clinical outcomes (Table 1), mechanical ventilation was required more frequently in the MGC group than in the no-MGC group (n = 4 (44.4%) vs. n = 65 (6.8%), $p < 0.001$). The length of hospital stays was significantly longer in the MGC group (48.3 ± 31.2 vs. 18.8 ± 26.8 days, $p = 0.002$), while the length of ICU stays and the duration of ventilation or CRRT did not differ significantly between the groups. All nine patients who developed major GI complications experienced postoperative morbidities (100%), compared with 12.2% of patients in the no-MGC group ($p < 0.001$). Among the postoperative complications, intra-abdominal fluid collection was notably more common in the MGC group, affecting four patients (44.4%), compared with 59 patients (6.2%) in the no-MGC group ($p < 0.001$). Infectious complications showed a similar pattern, occurring in four patients (44.4%) in the MGC group versus 73 patients (7.7%) in the no-MGC group ($p < 0.001$). Postoperative bleeding was also substantially more frequent in the complication group (three patients, 33.3%), whereas it was observed in only 20 patients (2.1%) in the no-MGC group ($p < 0.001$).

Table 1. Comparison of patient characteristics, hemodynamic parameters, and clinical outcomes according to the presence of major gastrointestinal complications (MGCs).

Variables	MGC (n=9)	No-MGC (n=953)	*p-value*
Age, years	67.2[21-113]	66.8[19-98]	*0.168*
Sex, male, n (%)	4(44.4%)	533(55.9%)	*0.490*
BMI (Kg/m²)	22.6 ± 4.1	23.4 ± 3.5	*0.528*
Total SOFA score at T0 (mean, ±SD)	6 ± 4.4	2.5 ± 2.2	*0.045*
APACHE II score	14.4 ± 6.9	10.1 ± 6	*0.030*
Hemodynamic value (mean, ±SD)			
Lactate at T0, mmol/L (mean, ±SD)	3.8 ± 2.5	2.3 ± 1.8	*0.104*
Lactate at T1, mmol/L (mean, ±SD)	5.3 ± 3.8	1.9 ± 1.3	*0.031*
Lactate at T2, mmol/L (mean, ±SD)	2.4 ± 1.9	1.4 ± 0.9	*0.152*
Lactate at T3, mmol/L (mean, ±SD)	1.5 ± 0.7	1.2±0.8	*0.282*
NLR at T0 (mean, ±SD)	8.9 ± 4.2	12.7 ± 9.3	*0.215*
NLR at T3 (mean, ±SD)	18.3 ± 18.4	10.9 ± 7.9	*0.264*
PLR at T0 (mean, ±SD)	249.1 ± 212	237.7 ± 170.8	*0.843*
PLR at T3 (mean, ±SD)	306 ± 270.5	179.3 ± 92.7	*<0.001*
MLR at T0 (mean, ±SD)	0.9 ± 0.6	0.7 ± 0.6	*0.456*
MLR at T3 (mean, ±SD)	1 ± 0.6	0.8 ± 0.8	*0.426*
CRP at T0 (mean, ±SD)	5 ± 7.6	3.2 ± 7.5	*0.486*
CRP at T3 (mean, ±SD)	20 ± 10.2	13.4 ± 7.9	*0.020*
Procalcitonin at T0, ng/mL	20.7 ± 41	6.2 ± 39.7	*0.275*
Procalcitonin at T3, ng/mL	76.5 ± 218.6	7.2 ± 96.9	*0.370*
Presepsin at T0	868.2 ± 430.9	714 ± 1026.2	*0.653*
Presepsin at T3	1024.9 ± 416.7	865.3 ± 971.4	*0.623*
Clinical outcomes			
Use of mechanical ventilation, n(%)	4(44.4%)	65(6.8%)	*<0.001*
Use of CRRT, n (%)	1(11.1%)	32(3.4%)	*0.203*
Length of mechanical ventilation, day	1 ± 2.3	0.3 ± 1.5	*0.183*
Length of CRRT, day	1 ± 3	0.1 ± 1	*0.416*
Length of ICU stay, day	5.9 ± 5.1	1.5 ± 1.4	*0.156*
Length of hospital stay, day	48.3 ± 31.2	18.8 ± 26.8	*0.002*
Postoperative morbidities	59(11%)	60(12.7%)	*0.434*
Postoperative morbidities	9(100%)	116(12.2%)	*<0.001*
Intraabdominal fluid collection	4(44.4%)	59(6.2%)	*<0.001*
Infectious complication	4(44.4%)	73(7.7%)	*<0.001*
Postoperative bleeding	3(33.3%)	20(2.1%)	*<0.001*
Pleural effusion	1(11.1%)	28(2.9%)	*0.154*
Re-admission	1(11.1%)	33(3.5%)	*0.216*

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APACHE II: acute physiology and chronic health evaluation II, BMI: body mass index, CRP:C-reactive protein, CRRT: continuous renal replacement therapy, GI: gastrointestinal, ICU: intensive care unit, MLR: monocyte-to-lymphocyte ratio, NLR: neutrophil-to-lymphocyte ratio, PLR: platelet-to-lymphocyte ratio, SD: standard deviation, SOFA: sequential organ failure assessment, T0: within 1 hour after admission to intensive care unit, T1: within 24 hours after admission to intensive care unit, T2: within 48 hours after admission to intensive care unit, T3: within 72 hours after admission to intensive care unit.

A partial correlation analysis was conducted to assess the association between PLR at T3 and various hemodynamic parameters (Table 2). PLRs at T3 were positively correlated with C-reactive protein levels at T3 (partial correlation coefficient = 0.079, $p = 0.021$), PCT levels at T3 (partial correlation coefficient = 0.130, $p < 0.001$), MLRs at T3 (partial correlation coefficient = 0.166, $p < 0.001$), and NLRs at T3 (partial correlation coefficient = 0.527, $p < 0.001$). However, no significant correlations were observed between PLRs and lactate or presepsin levels at T3.

Table 2. Partial correlation coefficients between platelet-to-lymphocyte ratio at T3 and other parameters after adjustment for age, sex, and BMI.

Parameters	Partial Correlation Coefficients	*p*-value
CRP at T3	0.079	*0.021*
Lactate at T3	-0.045	*0.218*
Presepsin at T3	0.069	*0.058*
PCT at T3	0.130	*<0.001*
MLR at T3	0.166	*<0.001*
NLR at T3	0.527	*<0.001*

BMI: body mass index, CRP:C-reactive protein, MLR: monocyte-to-lymphocyte ratio, NLR: neutrophil-to-lymphocyte ratio, PCT: procalcitonin, PLR: platelet-to-lymphocyte ratio, T3: within 72 hours after admission to intensive care unit.

A univariate analysis was performed to identify potential risk factors for postoperative MGCs (Table 3). The SOFA score at T0, lactate level at T1, C-reactive protein level at T3, and PLR at T3 were identified as significant predictors. In the multivariate analysis, an elevated PLR at T3 remained an independent predictor of postoperative MGCs [odds ratio [OR] = 1.006, 95% confidence interval [CI]: 1.002–1.009, $p = 0.002$].

Table 3. Predictors of major gastrointestinal complications in postoperative patients identified by univariate and multivariate logistic regression analyses.

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Age	1.002(0.953-1.055)	*0.924*		
Sex	1.586(0.423-5.944)	*0.494*		
Total SOFA score	1.433(1.202-1.709)	*<0.001*		
APACHE II score	1.034(0.996-1.074)	*0.076*		
Lactate at T1	1.026(1.044-1.536)	*0.016*		
CRP at T3	1.044(1.004-1.085)	*0.029*	1.043(0.998-1.089)	*0.060*
PLR at T3	1.006(1.003-1.010)	*<0.001*	1.006(1.002-1.009)	*0.002*

APACHE II: acute physiology and chronic health evaluation II, CI: confidence index, CRP:C-reactive protein, GI: gastrointestinal, OR: odds ratio, PLR: platelet-to-lymphocyte ratio, SOFA: sequential organ failure assessment, T1: within 24 hours after admission to intensive care unit, T3: within 72 hours after admission to intensive care unit.

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A univariate analysis was performed to identify the predictors of hospital readmission after discharge (Table 4). The analysis revealed that lower PLRs at T3 were significantly associated with an increased risk of readmission. This association persisted in the multivariate analysis, in which the PLR at T3 emerged as the sole independent predictor of hospital readmission (OR = 0.993, 95% CI: 0.987–0.998, *p = 0.011)*.

Table 4. Predictors of hospital readmission in postoperative patients identified by univariate and multivariate logistic regression analyses.

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Age	1.024(0.995-1.054)	*0.111*		
Sex	1.128(0.568-2.239)	*0.731*		
Lactate at T1	0.709(0.473-1.064)	*0.097*		
NLR at T0	1.024(0.995-1.055)	*0.108*		
PLR at T3	0.993(0.988-0.999)	*0.016*	0.993(0.987-0.998)	*0.011*

CI: confidence index, NLR: neutrophil-to-lymphocyte ratio, OR: odds ratio, PLR: platelet-to-lymphocyte ratio, T0: within 1 hour after admission to intensive care unit, T1: within 24 hours after admission to intensive care unit, T3: within 72 hours after admission to intensive care unit.

4. Discussion

The study findings demonstrated that the PLR measured at 72 hours after surgery was an independent predictor of both MGCs and hospital readmission in patients admitted to the ICU following major abdominal surgery. Notably, the direction of the association differed between the two outcomes; a higher PLR at 72 hours postoperatively was associated with an increased risk of MGCs, whereas a lower PLR at the same time point was associated with an increased risk of hospital readmission. These findings suggest that the PLR may reflect both the magnitude of the postoperative inflammatory burden as well as dynamic alterations in the balance between inflammatory responses, immune function, and overall recovery status during the postoperative period.

While postoperative inflammation is necessary for wound healing, excessive or prolonged inflammatory responses may lead to tissue injury, delayed recovery, and postoperative complications. Activated platelets play a central role in inflammatory and coagulation pathways by releasing mediators such as platelet-derived growth factor and thromboxane A_2 and by interacting with endothelial cells to promote microthrombosis and microcirculatory dysfunction. Platelet activation via Toll-like receptors in response to bacterial components further amplifies systemic inflammation through cytokine and chemokine release [23–26]. Consequently, excessive platelet activation may disrupt the balance between coagulation and inflammation, resulting in sustained inflammatory responses and delayed recovery. In addition to hemostasis, platelets also function as immune regulatory cells, modulating the secretion of interleukin (IL)-8, CCL2, and CXCL12 and releasing growth factors that promote angiogenesis and tissue remodeling, while platelet-derived transforming growth factor-β suppresses NKG2D expression on natural killer cells and Th17 activity, thereby attenuating antitumor and antibacterial immunity [27–30]. Acute stress conditions such as surgery, trauma, and sepsis are often accompanied by apoptosis-induced lymphopenia, a key mechanism of immunoparalysis associated with increased susceptibility to infection and mortality, which may persist for several weeks and contribute to impaired immune function [31–42]. Consistent with these mechanisms, patients in our study with elevated PLRs at 72 hours after ICU admission tended to exhibit persistent lymphopenia, suggesting exaggerated inflammatory activation and impaired immune recovery. Under such conditions, prolonged inflammation may disrupt wound healing and promote matrix metalloproteinase activation, leading to growth factor degradation and reduced extracellular matrix stability [43]. These findings are consistent with those of Solanki et al.,

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who reported that excessive postoperative inflammation after hepatectomy was associated with bile leakage and delayed liver regeneration [2].

In this context, the association between elevated PLRs at 72 hours and GI complications observed in our study suggests that excessive systemic inflammation accompanied by persistent lymphopenia may lead to immune dysregulation, impaired intestinal mucosal defense, and microcirculatory dysfunction, ultimately contributing to postoperative ileus or GI bleeding. Thus, serial postoperative PLR measurements may help monitor dynamic changes in inflammatory and immune status in critically ill patients who undergo major abdominal surgery. Patients with elevated PLRs at 72 hours after ICU admission may require closer clinical surveillance. Early imaging, such as abdominal radiography or CT, may facilitate prompt evaluation when decreased bowel motility or abdominal distension is suspected. Timely diagnostic evaluation and hemostatic intervention may be warranted when clinical signs suggest GI bleeding. Taken together, the PLR may reflect both hyper-inflammatory activation and immune suppression, depending on its postoperative dynamics.

Our study found that different PLRs at the same postoperative time point (72 hours) predicted distinct clinical risks. While an elevated PLR was associated with acute postoperative complications, a decreased PLR independently predicted hospital readmission after discharge. These findings suggest that the PLR should not be interpreted as a linear prognostic marker but rather as a dynamic biomarker reflecting different pathophysiological states depending on the direction of its deviation. Although a low PLR is often considered to indicate minimal inflammation, the 72-hour postoperative time point (T3) represents a transitional phase between acute inflammation and recovery. At this stage, a reduced PLR may reflect thrombocytopenia, lymphocyte rebound, or delayed physiological recovery, indicating impaired recovery from surgical stress. Thus, even patients who appear clinically stable at discharge may remain vulnerable to delayed adverse events and unplanned readmission. Taken together, extreme deviations in PLR at 72 hours may signal suboptimal postoperative recovery, supporting the bidirectional prognostic value of PLR for identifying high-risk critically ill patients after major abdominal surgery. Additionally, PLR measurements offer several practical advantages in critically ill postoperative patients. The PLR can be readily calculated from routine CBC results, which are universally available in most hospitals without additional cost. Because CBC parameters rapidly reflect acute immunological changes, serial measurements may help detect immune dysfunction or delayed immune recovery and thereby support more precise clinical decision-making. Unlike biomarkers such as lactate or presepsin levels, which require specialized assays, or complex scoring systems such as APACHE II or SOFA, the PLR is simple and easily applied in routine practice. Therefore, serial PLR monitoring may facilitate the early identification of high-risk patients and support timely clinical intervention after major surgery.

In the present study, the PLR measured 72 hours after surgery was a useful marker for identifying patients at high risk for MGC. Importantly, the 72-hour postoperative time point represents a critical phase characterized by substantial physiological and immunological changes. Previous studies have shown that inflammatory markers such as C-reactive protein levels begin to increase within 24–48 hours after surgery and typically peak around 72 hours postoperatively, accompanied by key immunological alterations including decreased monocyte HLA-DR expression, reduced natural killer cell activity, and increased proportions of PD-1-positive T cells [44]. Interleukin-10, which rises in the early postoperative phase (24–48 hours), suppresses monocyte HLA-DR expression and impairs antigen-presenting capacity, thereby promoting immunosuppression [45]. When such immune suppression persists beyond 72 hours, it may contribute to immunoparalysis associated with infection, organ dysfunction, and prolonged hospitalization. Notably, this period also coincides with the time when early signs of complications, such as postoperative ileus, ischemic injury, anastomotic leakage, and infection, most commonly emerge. Accordingly, changes in the PLR during this window may sensitively reflect intestinal mucosal injury, worsening inflammation, and impaired immune recovery. This interpretation is consistent with previous reports showing that postoperative inflammatory responses peak on postoperative days 2–3, when markers such as C-reactive protein levels, the NLR, and the PLR

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demonstrate their greatest changes and predictive value [46]. These findings support our observation that the PLR measured at 72 hours after surgery may serve as an early indicator of GI complication risk. Importantly, temporal changes in PLR should not be viewed merely as hematologic fluctuations but as signals reflecting postoperative immune recovery. A markedly elevated PLR may indicate excessive inflammatory activation with immune suppression, whereas a reduced PLR may suggest delayed immune recovery or underlying physiological vulnerability.

Despite the clinical relevance of our findings, several limitations should be acknowledged. First, although 962 postoperative ICU patients were included, MGCs occurred in only nine patients (0.9%), which may have limited statistical power and affected the precision of the estimated association between PLRs and MGCs. Second, the retrospective study design introduces the possibility of selection bias. Although adjustments were made for major clinical covariates, residual or unmeasured confounding, particularly differences in ICU management or treatment intensity, may not have been fully considered. Third, the PLR may be influenced by multiple clinical factors, including medications, fluid resuscitation, blood transfusion, nutritional status, and systemic inflammation, which could not be fully controlled in this study. Finally, the study population was heterogeneous with respect to surgical type and complexity. As a single-center study conducted at a tertiary referral hospital, the cohort mainly consisted of severely ill patients requiring postoperative ICU admission, which may limit the generalizability of the findings. Additionally, this study did not establish an optimal PLR cutoff value or a standardized clinical decision-making algorithm. Therefore, multicenter prospective studies with larger event numbers are required to validate the prognostic value of the PLR and establish clinically applicable cutoff values and decision-making strategies for postoperative management.

5. Conclusions

In conclusion, the PLR measured at 72 hours after surgery was associated with MGCs and hospital readmission in critically ill patients following major abdominal surgery. Both elevated and decreased PLRs reflected distinct pathophysiological states, suggesting that the PLR may serve as a dynamic marker of postoperative inflammatory and immune status. Given its simplicity and low cost, the PLR may help identify high-risk patients and support postoperative clinical decision-making. Further studies are needed to define optimal PLR cutoff values and standardize the clinical use of PLRs.

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Abbreviations

APACHE II	Acute Physiology and Chronic Health Evaluation II
BMI	Body Mass Index
CBC	Complete Blood Count
CRRT	Continuous Renal Replacement Therapy
CT	Computed Tomography
ICU	Intensive Care Unit
GI	Gastrointestinal
MGC	Major Gastrointestinal Complications
MLR	Monocyte-to-Lymphocyte Ratio
NLR	Neutrophil-to-Lymphocyte Ratio
PLR	Platelet-to-Lymphocyte Ratio
SOFA	Sequential Organ Failure Assessment
WBC	White Blood Cell Count

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