ORIGINAL ARTICLE

CARDIOVASCULAR SURGERY AND INTERVENTIONS

DOI: 10.4274/e-cvsi.2026.2115
Cardiovasc Surg Int 2026;13(1):13-19

The predictive value of the inflammatory prognostic index (IPI) for early adverse outcomes following carotid artery surgery

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Received: November 19, 2025 **Accepted:** January 24, 2026 **Epub:** February 20, 2026 **Publication Date:** March 02, 2026

ABSTRACT

Objectives: The inflammatory prognostic index (IPI), a composite biomarker incorporating C-reactive protein (CRP), neutrophil-to-lymphocyte ratio (NLR), and serum albumin (ALB), has recently emerged as a marker of systemic inflammation. This study aimed to investigate the predictive capacity of IPI for early adverse outcomes following carotid artery surgery. A retrospective cohort study was conducted in a single tertiary center.

Patients and methods: Medical records of 286 patients who underwent carotid artery surgery between January 2021 and December 2024 were retrospectively reviewed. Patients were stratified into two groups according to early postoperative outcomes: those with adverse events (major cerebrovascular accident, myocardial infarction, or 30-day mortality) and those without. Preoperative CRP, NLR, and ALB levels were collected to calculate the IPI using the formula (CRP×NLR)/ALB. Comparative analyses were performed between groups. Multivariate logistic regression identified independent predictors of adverse outcomes, and receiver operating characteristic (ROC) analysis was used to determine the optimal IPI cut-off point.

Results: Adverse outcomes were observed in 20 patients (7%). The adverse group showed significantly higher IPI and CRP values, along with lower serum ALB levels, compared to the favorable group. In multivariate analysis, IPI emerged as the only independent predictor of adverse outcomes. ROC analysis identified an IPI threshold of 0.27, which predicted adverse outcomes with high sensitivity but moderate specificity.

Conclusion: This study is the first to demonstrate that IPI is an independent predictor of early adverse outcomes following carotid artery surgery. Given its simplicity and cost-effectiveness, IPI may serve as a practical biomarker for preoperative risk stratification and clinical decision-making in vascular surgery

Keywords: Inflammatory prognostic index, biomarker, poor outcome, carotid artery surgery.

Carotid artery surgery (CAS), remains a cornerstone in the management of significant carotid artery stenosis, aiming to reduce the risk of ischemic stroke—one of the leading causes of morbidity and mortality worldwide.[1] Approximately one-third of ischemic strokes are attributed to carotid artery stenosis.[2] While CAS is an effective strategy for stroke prevention, it is not without risks. In high-risk patients, the incidence of postoperative stroke or death can exceed 10%.[3] To optimize the risk-benefit ratio of CAS, guidelines recommend maintaining perioperative (intraoperative and postoperative) stroke or death rates below 6% for symptomatic and 3% for asymptomatic patients.[4] Achieving these targets, however, is challenged by the lack of standardized tools capable of reliably predicting adverse outcomes following the procedure.

Atherosclerosis is widely recognized as a chronic inflammatory process.[5] The formation and progression of atherosclerotic plaques are largely governed by inflammatory and immune mechanisms.[5] Numerous biomarkers reflecting systemic inflammation and immune status have been shown to be associated with outcomes following oncologic and cardiovascular surgery.[6,7] Among these, complete blood count-derived markers such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-

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Cite this article as: Güven H, Çetintaş D. The predictive value of the inflammatory prognostic index (IPI) for early adverse outcomes following carotid artery surgery. Cardiovasc Surg Int. 2026;13(1):13-19

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lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) have demonstrated potential prognostic value for predicting outcomes in vascular surgery, including CAS.[6,8,9]

The inflammatory prognostic index (IPI), a novel composite marker combining C-reactive protein (CRP), NLR, and serum albumin (ALB) levels, has recently emerged as a promising prognostic tool.[10] Calculated as IPI = CRP×NLR/ALB, this index provides an integrative measure of a patient's inflammatory and immune status. Initially introduced by Dirican et al.[10] to predict prognosis in patients with non-small cell lung cancer, the IPI has since demonstrated prognostic potential in various clinical settings. For example, it has demonstrated prognostic potential in various cardiovascular conditions, such as new-onset atrial fibrillation, mortality after coronary artery bypass grafting, and 90-day outcomes after acute ischemic stroke (AIS).[11] Furthermore, the potential role of IPI in vascular surgery has been evaluated, for example, in predicting perioperative mortality after type A aortic dissection surgery.[12]

However, while IPI has been investigated mainly in the context of long-term survival or in relation to other inflammatory indices, its role in predicting early adverse outcomes (within 30 days) following CAS has not yet been investigated. Although a recent study highlighted the prognostic potential of SII for early side effects following CAS,[8] the potential of IPI to predict adverse outcomes after CAS, including similar studies focused on inflammation, has not been investigated. This knowledge gap underlines the necessity of further exploring the IPI as a predictive tool for early complications after CAS.

The primary objective of this study is to investigate the role of preoperative IPI levels in predicting early adverse outcomes (defined as stroke, myocardial infarction [MI], or death within 30 days) in patients undergoing CAS.

PATIENTS AND METHODS

Study Design and Patient Selection

This retrospective observational cohort study included 286 patients who underwent CAS for significant carotid artery stenosis at the department of cardiovascular surgery of a tertiary referral hospital in Türkiye between January 2021 and December 2024. The study enrolled adult patients who underwent elective, first-time, isolated CAS with complete and accessible medical records. Exclusion criteria included the absence or incompleteness of medical data, emergency procedures, re-operations, combined carotid and coronary artery surgeries, and the presence of active infections, malignancies, hematological or immunological disorders, as well as end-stage renal or hepatic diseases. Based on postoperative outcomes, patients were categorized into two groups: The adverse outcome group (n=20), comprising those who experienced adverse events such as major cerebrovascular accident (CVA), MI, or 30-day mortality, and the favorable outcome group (n=266). Clinical data, including demographic characteristics, comorbidities, laboratory test results, derived indices, intraoperative details, and postoperative complications, were retrieved from the hospital's electronic medical records system, documented for analysis, and subsequently compared between these two groups. Patients receiving preoperative antiplatelet therapy (e.g., aspirin or clopidogrel) were enrolled, and bleeding/hematoma rates were compared between patients receiving antiplatelet therapy and those not receiving antiplatelet therapy.

Ethical approval for the study was obtained from the University of Health Sciences Türkiye, Bursa City Hospital Scientific Research Ethics Committee (approval number: 2025-2/6, date: 22.01.2025). The study was performed in accordance with the ethical principles of the Declaration of Helsinki.

Preoperative Management

The indications for surgical intervention were defined as the presence of ≥50% internal carotid artery (ICA) stenosis in symptomatic patients and ≥70% ICA stenosis in asymptomatic patients, following the criteria established by the North American Symptomatic Carotid Endarterectomy (CAS) Trial and the Society for Vascular Surgery guidelines.[13,14] The degree of ICA stenosis was initially assessed through color Doppler ultrasound and subsequently confirmed using advanced angiographic techniques, including computed tomography angiography and digital subtraction angiography. All patients scheduled for surgery were prescribed 100 mg/day of oral acetylsalicylic acid (ASA) as an antiplatelet therapy prior to the procedure.

Surgical Technique

All surgical procedures were performed under general anesthesia with intraoperative monitoring using near-infrared spectroscopy. An oblique incision was made along the anterior border of the sternocleidomastoid muscle. After exposing the carotid bifurcation, the common carotid artery (CCA), ICA, and external carotid artery (ECA) were isolated using vascular tapes. These arteries were visually inspected, and clamps were applied sequentially (ICA, CCA, and ECA) following intravenous administration of heparin at a dose of 150 IU/kg, targeting an activated clotting time of ≥250 seconds. Depending on the surgeon's preference, different surgical techniques were employed, including conventional CAS with either Dacron or saphenous vein patch closure, primary closure (without patch), traditional or modified eversion CAS, and saphenous vein graft bypass or interposition for the ICA. Upon removal of the atherosclerotic lesion, the clamps were released in reverse order (ECA, CCA, and ICA). Protamine was administered intravenously to neutralize heparin. Hemostasis was achieved, and the wound was closed in standard fashion. The effect of surgical techniques on clinical outcomes was evaluated by statistical analysis

Postoperative Management and Follow-up

Following surgery, all patients were initially monitored in the intensive care unit (ICU). In the absence of major complications, they were transferred to the general ward on the first postoperative day. Dual antiplatelet therapy, consisting of 100 mg/day ASA and 75 mg/day clopidogrel, was initiated six hours postoperatively in the absence of significant bleeding. Discharged patients were scheduled for routine follow-up visits at one week, then at one, two, three, six, and twelve months, and annually thereafter. At each visit, patients underwent physical examination and routine Doppler ultrasonography. Patients were monitored for at least 30 days postoperatively, except for those who experienced mortality.

Laboratory Analysis

Blood samples were collected after a minimum of six hours of fasting, using vacutainers for peripheral venous access. The collected samples were placed in tubes containing anticoagulants and promptly transported to the laboratory. Automated biochemical and hematological analyzers were utilized to measure the laboratory

parameters, which included glucose, hemoglobin A1c, total cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, ALB, CRP, hemoglobin, hematocrit, platelet count, white blood cell count, neutrophil count, and lymphocyte count. In addition, several derived indices were calculated: The NLR, PLR, systemic immune-inflammation index (SII), and IPI. Given that composite inflammatory indices are derived from basic laboratory parameters, alternative multivariable model structures were explored during the analysis phase. These included models incorporating clinical variables combined with a single inflammatory index. The final model was selected based on clinical relevance and overall model performance. SII was calculated using the formula "platelet×NLR", while IPI was determined as "CRP×NLR/ALB".

Statistical Analysis

Data analysis was conducted using IBM SPSS Statistics version 24 (IBM Corp., Armonk, NY, USA). The normality of distribution for continuous variables was assessed using the Shapiro-Wilk test. Continuous variables that followed a normal distribution were compared between groups using the Student's t-test, while the Mann-Whitney U test was employed for variables that did not follow a normal distribution. Categorical variables were analyzed using the chi-square test. Variables identified as significant in univariate analysis ($p<0.1$) were further evaluated using multivariate logistic regression to identify independent predictors of poor outcomes. Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cut-off value(s) for significant variables, with sensitivity and specificity values calculated based on the area under the curve (AUC). To assess potential multicollinearity among variables included in the multivariable logistic regression model, variance inflation factor (VIF) values were calculated. A VIF value <5 was considered indicative of the absence of significant multicollinearity.

Given the shared components among inflammatory indices, IPI was retained in the final model due to its superior discriminatory performance.

Normally distributed continuous variables were presented as mean ± standard deviation, whereas non-normally distributed variables were reported as median (minimum-maximum). Categorical variables were expressed as numbers (percentages). A p-value of less than 0.05 was considered statistically significant.

RESULTS

In this study, the overall population had a mean age of 65.9±8.7 years (ranging from 42 to 88 years), with 36% of the participants being female. Among the cohort, 63% had a history of previous cerebrovascular events (CVE), including amaurosis fugax, transient ischemic attack, and minor or major stroke. Contralateral ICA occlusion was present in 13 patients (4.5%). The overall incidence of the composite 30-day adverse outcome was 7% (20 out of 286 patients). The primary poor outcomes assessed in this study were major CVA, MI, and mortality, which were observed in 13, 2, and 5 patients, respectively.

Comparative analysis of the preoperative demographic and clinical characteristics between the adverse outcome group and the favorable outcome group demonstrated no statistically significant differences for any of the assessed parameters, indicating that the groups were comparable in terms of baseline characteristics (Table 1).

When intraoperative and postoperative variables were analyzed, the median lengths of stay in the ICU and the overall hospital stay were significantly longer in the adverse outcome group compared to the favorable outcome group. Prolonged ICU and hospital stays were observed as a consequence of major postoperative complications and were not included as predictive variables in the risk assessment analyses.

No significant differences were identified between the two groups for any other intraoperative or postoperative variables assessed (Table 2).

Table 1. Preoperative basic demographic and clinical characteristics

Variable	Adverse outcome group (n=20)	Favorable outcome group (n=266)	p-value
Age (year)	67.1±6.4	65.8±8.9	0.380
Gender (female)	7 (35%)	96 (36%)	0.922
Weight (kg)	81.3±15.3	76.6±14.5	0.201
Height (cm)	170.0±4.8	170.5±5.1	0.658
BMI (kg/m²)	28.1±5.2	26.4±5.1	0.162
Smoking	8 (40%)	92 (35%)	0.624
Antiplatelet therapy (%)	9 (45%)	82 (39 %)	0.312
HT	14 (70%)	159 (60%)	0.367
DM	9 (45%)	112 (42%)	0.800
HL	8 (40%)	96 (36%)	0.726
CAD	11 (55%)	127 (48%)	0.531
PAD	6 (30%)	52 (20%)	0.262
COPD	2 (10%)	16 (6%)	0.479
Previous CVE	15 (75%)	164 (62%)	0.234
Contralateral ICA occlusion	2 (10%)	11 (4%)	0.225

BMI: Body mass index; CAD: Coronary artery disease; COPD: Chronic obstructive pulmonary disease; CVE: Cerebrovascular event; DM: Diabetes mellitus; HL: Hyperlipidemia; HT: Hypertension; ICA: Internal carotid artery; PAD: Peripheral artery disease.

Table 2. Intraoperative and postoperative data

Variable	Adverse outcome group (n=20)	Favorable outcome group (n=266)	p-value
Clamp time (min)	24 (13-75)	24 (10-90)	0.905
Operation time (min)	58 (45-142)	60 (32-197)	0.762
Duration of ICU stay (hour)	180 (48-360)	18 (4-72)	<0.001
Duration of hospital stay (day)	12 (2-32)	4 (2-10)	<0.001
Vocal cord paralysis	1 (5%)	7 (3%)	0.536
Bleeding/hematoma	0 (0%)	14 (5%)	0.293
Major CVA	13 (65%)	0 (0%)	NS
MI	2 (10%)	0 (0%)	NS
30-day mortality	5 (25%)	0 (0%)	NS
Surgical techniques			0.896
CEA with patch	10 (%50)	130 (48.9%)	
CEA without patch	6 (30%)	77 (28.9%)	
Eversion	3 (15%)	37 (13.9%)	
Saphenous vein graft	1 (5%)	22 (8.3%)	

CVA: Cerebrovascular accident; ICU: Intensive care unit; MI: Myocardial infarction; CEA: Carotid Endarterectomy; NS: Not specified.

Evaluation of the laboratory test results and derived indices revealed that the mean serum ALB level was significantly lower, while the median values of CRP and the IPI were significantly higher in the adverse outcome group compared to the favorable outcome group. No other laboratory parameters showed statistically significant differences between the two groups (Table 3).

Following the univariate analysis, variables with p values less than 0.1 were considered potential risk factors and were included in the multivariate logistic regression analysis. In this regression model, only IPI retained statistical significance as an independent predictor of poor outcome, while other variables were excluded due to lack of significance. VIF analysis demonstrated no significant multicollinearity among the included variables, with all VIF values remaining below the predefined threshold (Table 4).

Table 3. Laboratory parameters

Variable	Adverse outcome group (n=20)	Favorable outcome group (n=266)	p-value
Glucose (mg/dL)	116.9±25.1	117.4±22.1	0.941
Hemoglobin A1c (%)	6.0±1.0	6.1±1.0	0.686
Total cholesterol (mg/dL)	207.7±46.0	196.3±49.8	0.334
LDL cholesterol (mg/dL)	131.1±24.3	130.9±34.9	0.968
Triglyceride (mg/dL)	178.4±68.3	170.6±62.4	0.627
Albumin (g/L)	37.0±4.9	42.1±6.4	<0.001
CRP (mg/L)	4.5 (1.4-16.3)	2.8 (0.3-31.6)	0.004
Hemoglobin (g/dL)	12.7±2.0	12.6±1.9	0.952
Hematocrit (%)	38.4±6.2	38.5±8.1	0.966
Platelet count (10^3/µL)	274.2±104.7	264.9±97.8	0.702
WBC count (10^3/µL)	9.3±3.0	8.2±2.5	0.116
Neutrophil count (10^3/µL)	6.7±2.5	5.6±1.9	0.085
Lymphocyte count (10^3/µL)	1.3±0.7	1.4±0.8	0.393
NLR	5.5 (2.2-16.6)	4.0 (1.1-25.6)	0.061
PLR	248 (73-694)	203 (33-1461)	0.325
SII	1452 (406-4802)	1067 (189-6930)	0.070
IPI	0.73 (0.13-3.43)	0.26 (0.02-4.00)	<0.001

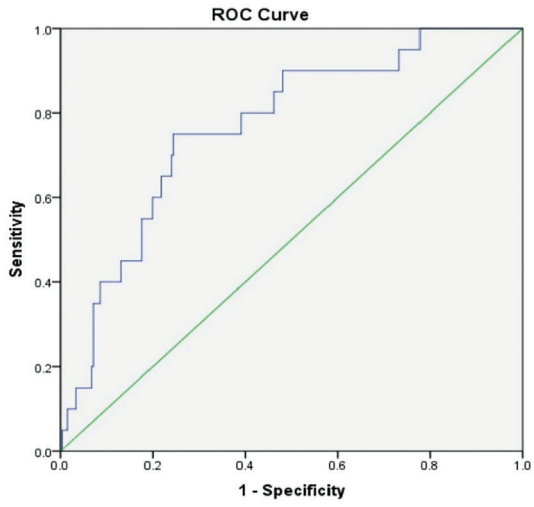
CRP: C-reactive protein; IPI: Inflammatory prognostic index; LDL: Low-density lipoprotein; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; SII: Systemic immune-inflammation index; WBC: White blood cell.

ROC curve analysis identified an IPI cut-off value of 0.27 as the optimal threshold for predicting poor outcomes, with a sensitivity of 85% and a specificity of 48% (AUC=0.767 [95% confidence interval: 0.666-0.869]) (Figure 1).

DISCUSSION

Our study demonstrates that the IPI is a significant prognostic biomarker for predicting early adverse outcomes following CAS. IPI, which integrates CRP, NLR, and serum ALB levels, provides a comprehensive reflection of the patient's inflammatory and immune response. Our findings revealed that higher IPI values were significantly associated with poor outcomes (p<0.001). Specifically, the median IPI value was 0.73 (0.13-3.43 in the adverse outcome group, compared to 0.26 (0.02-4.00) in the good outcome group.

Our analysis showed that traditional risk factors, including age, gender, body mass index, smoking status, hypertension (HT), diabetes mellitus, hyperlipidemia, coronary artery disease, peripheral artery disease, and chronic obstructive pulmonary disease, did not significantly differentiate between patients with poor and good outcomes (p>0.05).



Optimum cut-off value	Area under the curve (AUC)	Sensitivity	Specificity	P value
0.27	0.767 (0.666–0.869)	85%	48%	<0.001

Figure 1. Receiver operating characteristic (ROC) curve analysis.

Table 4. Multivariate logistic regression analysis to determine the independent predictors

Variable	Beta	Standard error	Wald	Expected beta	95% CI for expected beta (upper-lower)	p-value
Neutrophil	0.444	0.865	0.264	1.560	0.286-8.494	0.607
NLR	0.000	0.171	0.000	1.000	0.716-1.397	0.999
SII	0.000	0.000	0.085	1.000	0.999-1.001	0.771
CRP	0.073	0.107	0.459	1.075	0.871-1.327	0.498
Albumin	0.227	0.125	3.324	1.255	0.983-1.603	0.068
IPI	-0.189	0.056	11.329	0.828	0.742-0.924	0.001

CI: Confidence interval; CRP: C-reactive protein; IPI: Inflammatory prognostic index; NLR: Neutrophil-to-lymphocyte ratio; SII: Systemic immune-inflammation index. Variance inflation factor values for all variables included in the model were <5.

For instance, the prevalence of HT was 14 (70%) in the adverse outcome group versus 14 (70%) in the good outcome group (p=0.367). This finding indicates that conventional risk factors may be limited in predicting individual patient outcomes, highlighting the potential of inflammatory biomarkers to bridge this gap. The comparison of bleeding and hematoma rates according to preoperative antiplatelet therapy was included as a secondary safety analysis, given the routine use of antiplatelet agents in patients undergoing CAS. The proportion of patients receiving preoperative antiplatelet therapy was 31% (82/266) in the favorable outcome group and 45% (9/20) in the adverse outcome group (p=0.312, Table 1). In our cohort, preoperative antiplatelet therapy was not associated with an increased risk of bleeding or hematoma formation (p=0.293, Table 2). This finding should be interpreted as descriptive and exploratory, and it does not suggest any modification of current perioperative antiplatelet management strategies.

In our study, there is no statistically significant difference was found between patients in the adverse outcome group (n=20) and the good outcome group (n=266) in terms of carotid clamp time (median 24 [13-75] min *vs.* median 24 [10-90] min, p=0.905) and operative time (median 58 [45-142] min *vs*. median 60 [32-197] min, p=0.762). We also evaluated the impact of different surgical techniques (conventional carotid endarterectomy, patch closure, eversion techniques, and saphenous vein grafting) on clinical outcomes. Although no significant difference was found between techniques (0.896, Table 2), the potential impact of this factor on outcomes cannot be excluded. Future studies should further examine the prognostic value of surgical techniques in conjunction with IPI. However, regarding other perioperative and postoperative outcomes, ICU stay (median 180 [48-360] hours) and hospital stay (median 12 [2-32] days) were found to be statistically significantly longer in the adverse outcome group. This suggests that major complications that constitute the definition of adverse outcome (major CVA 65%, MI 10%, 30-day mortality 25%- seen only in the adverse outcome group) result in patients staying in the ICU and hospital for longer periods. The significantly longer ICU and hospital stays observed in the adverse outcome group should be interpreted as downstream consequences of major postoperative complications rather than as preoperative or intraoperative predictors. This temporal distinction is essential to avoid misinterpretation of length-of-stay variables as causative factors.

Chronic systemic inflammation is known to play a pivotal role in the pathogenesis of atherosclerosis and vascular diseases.[15] Numerous studies have reported the prognostic value of inflammatory markers in predicting adverse outcomes following CAS.[16,17] Although major CVA and MI have different pathophysiological mechanisms, both are influenced by the inflammatory nature of atherosclerosis. As composite indices integrate multiple inflammatory and nutritional parameters, they were interpreted as summary markers rather than independent substitutes for their individual components. As an indicator of systemic inflammation and immune status, IPI may reflect the common inflammatory pathways of these complications.[18] Therefore, IPI is thought to offer prognostic value for both conditions. In our univariate analysis, ALB level was found to be significantly lower in the adverse outcome group (p<0.001). ALB is a negative acute phase reactant that decreases in the inflammatory response and has been reported to affect the prognosis of AIS.[19] The median value of CRP was found to be significantly higher in the adverse outcome group (p=0.004). CRP is a well-known positive acute phase reactant of inflammation and is associated with the risk of cardiovascular events.[19,20] According to the univariate analysis results, many other laboratory parameters such as glucose, HbA1c, total cholesterol, LDL cholesterol, triglyceride, hemoglobin, hematocrit, platelet count, leukocyte count, lymphocyte count, NLR, PLR and SII did not show any statistically significant difference between the two groups (p>0.05).

The most critical finding is the multivariate analysis result. In multivariate analysis, only IPI was found to be a significant and independent predictor of adverse outcome (p=0.001), while other markers with univariate significance or p<0.1, including CRP and ALB, lost their statistically significance. This result indicates that IPI (as an index combining CRP, NLR and ALB) can independently predict early poor outcomes after CAS better than markers such as CRP or ALB alone or other derived indices such as SII, and the association of the components of IPI with inflammatory status supports the linkage of inflammation with postsurgical outcomes.

ROC curve analysis was used to evaluate the prognostic performance of IPI in predicting early poor outcomes after CEA. The AUC value obtained was 0.767, indicating that IPI has a moderate discriminatory power. The determined cut-off value of 0.27 can correctly identify the majority of patients with poor outcomes with a sensitivity of 85%. The identified IPI cut-off value demonstrated high sensitivity but limited specificity, indicating that while it may be useful as a screening tool for early risk identification, it is not suitable as a standalone marker for guiding major perioperative clinical decisions. Instead, IPI should be integrated with established clinical, radiological, and procedural factors to support comprehensive perioperative risk assessment. However, the specificity of this cut-off value was found to be 48%, indicating that approximately half of the patients without poor outcomes could be classified as false positives.

These findings reveal that IPI has a high sensitivity and low specificity profile. Such a marker may have clinical value as a screening tool, especially for the early identification of high-risk patients. However, the high false positive rate due to low specificity may limit the use of IPI as a sole decision-making tool and suggest that it should be supported by more specific tests.

Although postoperative adverse events following CAS are predominantly influenced by mechanical and procedural factors—including plaque manipulation, embolic phenomena, and perioperative hypoperfusion—systemic inflammation may contribute indirectly by reflecting overall patient vulnerability and impaired physiological reserve. In this context, the IPI should not be interpreted as a mechanistic driver of postoperative complications, but rather as a composite marker of systemic inflammatory and nutritional status that may complement traditional risk assessment strategies.

Furthermore, while more specific biomarkers of plaque-related inflammation (such as hs-CRP, matrix metalloproteinases, or lipoprotein-associated phospholipase A_2) may provide greater biological specificity, these parameters are not routinely measured in standard clinical practice. The use of IPI, derived from widely available laboratory parameters, therefore represents a pragmatic approach to preoperative risk stratification rather than a substitute for plaque-specific inflammatory assessment.

The simplicity and cost-effectiveness of IPI, which is derived from routinely measured blood parameters, make it a practical tool in clinical practice. Identifying high-risk patients based on preoperative IPI values can enable proactive perioperative management strategies, such as intensive monitoring or the consideration of anti-inflammatory therapies in patients with high IPI.

This study has several limitations. First, its retrospective design introduces inherent biases, such as selection and information bias, which may affect the generalizability of the findings. Second, being a single-center study conducted in a tertiary referral hospital, the results may not be directly applicable to other populations or healthcare settings. Third, the inflammatory markers used to calculate the IPI, including CRP, NLR, and serum ALB, were derived from routine laboratory measurements, which may be subject to inter-laboratory variability. Fourth, the study focused exclusively on early (30-day) adverse outcomes, potentially overlooking the long-term prognostic value of IPI. Fifth, the exclusion of patients with active infections, malignancies, hematological or immunological disorders, and end-stage renal or hepatic diseases may limit the applicability of the findings to broader patient populations. Sixth, although multivariate analysis was used to adjust for potential confounding factors, the possibility of residual confounding cannot be completely ruled out. Finally, despite demonstrating high sensitivity, IPI had moderate specificity, leading to a substantial number of false positives, which limits its use as a standalone diagnostic tool. The relatively small number of adverse events limits the statistical power of multivariate analyses and raises the possibility of model overfitting. Therefore, the results of the regression analysis should be interpreted with caution and regarded as exploratory rather than definitive.

In this study, we demonstrated that higher preoperative IPI values were significantly associated with early adverse outcomes following CAS. Specifically, higher preoperative IPI values were associated with an increased risk of major CVE, MI, and 30-day mortality. Our findings suggest that IPI, a composite marker derived from routinely measured inflammatory parameters (CRP, NLR, and ALB), provides a simple and cost-effective tool for risk stratification in patients undergoing CAS. However, due to its moderate specificity, IPI may serve as a practical adjunctive biomarker for preoperative risk stratification and risk awareness, rather than a sole determinant of clinical decision-making. Further prospective multicenter studies with larger sample sizes are needed to validate these findings and explore the potential benefits of integrating IPI into clinical decision-making protocols.

Ethics

Ethics Committee Approval: Ethical approval for the study was obtained from the University of Health Sciences Türkiye, Bursa City Hospital Scientific Research Ethics Committee (approval number: 2025-2/6, date: 22.01.2025). The study was performed in accordance with the ethical principles of the Declaration of Helsinki.

Informed Consent: Retrospective study.

Acknowledgments

We would like to thank Beytullah Kınabaş for his technical support.

Footnotes

Authorship Contributions

Concept: H.G., D.Ç.; Design: H.G., D.Ç.; Data Collection or Processing: H.G., D.Ç.; Analysis or Interpretation: H.G., D.Ç.; Literature Search: H.G., D.Ç.; Writing: H.G., D.Ç.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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