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Original Article

Preoperative inflammatory burden index as a predictor of postoperative outcomes in open abdominal aortic aneurysm repair

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Abstract

Aim: The Inflammatory Burden Index(IBI), calculated using C-reactive protein (CRP), neutrophil, and lymphocyte counts, has emerged as a promising marker of systemic inflammation. However, its prognostic utility in vascular surgical procedures has not been well established. This investigation examined the relationship between preoperative IBI values and postoperative complications in individuals undergoing open abdominal aortic aneurysm (AAA) repair.

Material and Methods: We conducted a retrospective case-control analysis involving 86 patients who received elective open AAA repair from January 2017 through May 2022. Participants were categorized into two cohorts based on whether they experienced postoperative wound discharge. We employed receiver operating characteristic (ROC) curve methodology to identify the optimal IBI threshold for predicting superficial surgical site infection (SSSI). We then compared clinical characteristics, laboratory parameters, and perioperative outcomes between patients with high versus low IBI levels.

Results: SSSI occurred in 30.2% of patients and was significantly associated with elevated IBI ($p = 0.033$). ROC analysis identified an IBI threshold of 24.271 as optimal for SSSI prediction (AUC: 0.646, 95% CI: 0.516--0.775). Individuals with IBI ≥24.271 demonstrated notably lower albumin concentrations, decreased ejection fraction, elevated CRP and neutrophil levels, extended hospitalization duration, slower resumption of oral intake, and higher SSSI incidence ($p < 0.05$ for all comparisons). As an inflammatory marker, IBI is noteworthy for its predictive value in terms of postoperative outcomes.

Conclusion: High preoperative IBI was associated with increased postoperative SSSI and delayed recovery in patients undergoing open AAA repair. IBI may serve as a practical, cost-effective biomarker for preoperative risk stratification and can be evaluated in terms of perioperative management strategies to improve outcomes.

Keywords: Inflammatory burden index, open repair, surgical site infection, aortic aneurysm, abdominal, predictive tests, postoperative outcomes

INTRODUCTION

Abdominal aortic aneurysm (AAA) is a significant vascular condition with prevalence rates varying by geographic region and demographic factors. According to the European Society for Vascular Surgery (ESVS) 2024 Clinical Practice Guidelines on the Management of Abdominal Aorto-Iliac Artery Aneurysms, the prevalence of AAA in the elderly population is approximately 1-2% in recent years, with higher rates observed in specific populations [1]. In the guidelines it was well described that the prevalence is negligible before the age of 55-60 years but increases steadily with age. In cases where systemic or anatomical factors preclude endovascular aortic repair (EVAR), open aneurysm repair (OAR) remains an important option in young and healthy patients. In recent years, the management

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of AAA has undergone dynamic changes, with many surgeons and surgical centers increasingly favoring EVAR over open surgical repair. The literature contains extensive comparative analyses between EVAR and open repair methodologies [2,3]. Despite this shift, modern outcomes following open surgical AAA repair demonstrate excellent results. Due to its economic advantages and favorable long-term prognosis, open repair maintains its position as an essential therapeutic alternative. Although mortality rates are reported as 3% in high-volume centers, most studies have found mortality rates to be between 5% and 8% [3].

Systemic inflammatory biomarkers, including white blood cell (WBC) counts and C-reactive protein (CRP) concentrations, demonstrate positive correlations with aneurysm diameter [4,5]. Accumulating evidence suggests that systemic inflammatory responses substantially affect postoperative recovery and complication rates. Increased inflammatory marker levels have been linked to poor outcomes in cardiovascular pathologies [6,7].

Postoperative complications following open abdominal aortic aneurysm repair represent a major determinant of patient morbidity and recovery [8,9]. These complications may result from multiple mechanisms, including factors related to the surgical technique itself---such as operative approach, tissue handling, and wound management---as well as patient-specific characteristics, including comorbid conditions and baseline physiological status [10]. Multiple investigations have employed preoperative inflammatory measurements particularly CRP levels---as predictors of early and late surgical outcomes [11,12].

Superficial surgical site infection (SSSI) is defined as an infection occurring within 30 days after surgery, affecting only the skin and subcutaneous tissue of the incision, and meeting standard criteria such as purulent drainage, positive culture of incisional fluid/tissue, deliberate opening of the incision with local signs of infection, or clinician diagnosis [13]. In populations undergoing vascular surgery, the incidence of surgical site infections (SSIs) ranges from approximately 2.5% to over 10%, depending on the type of procedure and patient factors [14]. However, certain patient populations and surgical approaches may be associated with higher rates due to specific risk factors and definitions used.

The Inflammatory Burden Index (IBI) is a practical biomarker that reflects systemic inflammatory activity, calculated using CRP, neutrophil (Ne), and lymphocyte (Lym) counts. Initially proposed by Xei et al. for assessing inflammation and prognosis in oncology patients [15], its utility in predicting surgical outcomes has not yet been fully explored in the context of vascular surgery or AAA repair. In this study, we aimed to evaluate the predictive value of IBI in estimating postoperative morbidity in patients undergoing elective open surgical repair of AAA.

MATERIAL AND METHODS

Patient Population and Study Design

This retrospective observational case-control investigation study examined patients who underwent surgical treatment for AAA at our center from January 2017 through May 2022. During this timeframe, 93 patients underwent AAA surgery. We excluded 7 patients who required urgent surgical intervention for ruptured AAA or those lacking adequate follow-up information, resulting in 86 patients who were incorporated into the final analysis. The decision to perform surgery is based on current guideline recommendations and individual patient risk assessments. Elective surgical repair was indicated in patients with a maximum aneurysm diameter $\geq$ 5.5 cm in men or $\geq$ 5 cm in women, and a rapid increase in diameter (> 0.5 cm within six months). We conducted retrospective chart reviews to document preoperative clinical characteristics, intraoperative management approaches, and postoperative results.

The primary endpoint was the occurrence of SSSI after surgery, while secondary outcomes included in-hospital mortality, duration of hospital stay, and postoperative recovery parameters. Since this was a retrospective study, no randomization or blinding (masking) procedures were applicable. This investigation employed a non-probability consecutive sampling method, including all eligible patients within the study period. The sample size was determined by the total number of patients who met the inclusion criteria, as no prior comparable data were available for a formal power analysis. The Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital Ethics Committee granted approval for this study protocol (Date: 02.08.2022, Approval No: 2022.06.37). All procedures adhered to the principles outlined in the Declaration of Helsinki.

Patients were evaluated preoperatively using thoracoabdominal computed tomography angiography and categorized as suprarenal, pararenal, juxtarenal, or infrarenal AAA. We documented baseline clinical parameters, past medical history, concurrent illnesses, echocardiographic assessments, laboratory results, operative characteristics, and postoperative adverse events. Data collection utilized both the hospital's electronic medical record system and individual patient charts.

IBI was calculated for each patient using preoperative laboratory values and defined as: IBI: CRP × Ne / Lym. CRP levels were measured in mg/L, and neutrophil and lymphocyte counts were expressed in 10^9/L.

Hypertension was defined as a systolic blood pressure ≥ 140 mmHg and/or a diastolic blood pressure ≥ 90 mmHg. Diabetes mellitus was diagnosed based on a random plasma glucose level ≥ 200 mg/dL (11.1 mmol/L). Patients with an estimated glomerular filtration rate below 60 mL/min/1.73 m² were considered to have chronic kidney disease. Chronic obstructive pulmonary disease (COPD) was diagnosed in patients with an FEV1/FVC ratio < 0.70 despite bronchodilator therapy, accompanied by symptoms of dyspnea or cough, and a significant history of exposure to noxious stimuli. Hyperlipidemia was defined as a total cholesterol level ≥ 240 mg/dL, a triglyceride level ≥ 200 mg/dL, or the use of lipid-lowering medication. A history of myocardial infarction, positive coronary angiography, or prior coronary artery bypass grafting was considered indicative of cardiac disease. Preoperative left ventricular ejection fraction was assessed via echocardiography using the biplane Simpson method. The need for inotropic support was defined as postoperative administration of one or more inotropic agents.

Surgery Procedure

All operations were conducted using general anesthesia via a midline transperitoneal surgical approach. When femoral artery anastomosis was required, we performed either unilateral or bilateral horizontal groin incisions to expose the femoral bifurcation. For procedures necessitating suprarenal cross-clamping, we delivered cold Custodiol solution (4 - -8°C) directly into the renal artery ostia through ostial cannulation at 15 mL/min throughout the clamping duration. Dacron grafts were employed in all operations. Antimicrobial prophylaxis followed established protocols for preoperative, intraoperative, and postoperative phases [16].

In-hospital mortality is defined as death occurring during the hospital stay. Postoperative dialysis requirement was determined jointly by the nephrology and surgical teams and was defined as the need for at least one dialysis session or continuous veno-venous hemodiafiltration. Gastrointestinal function recovery was categorized as: Regimen 1 (R1) representing clear liquid tolerance, Regimen 2 (R2) indicating soft or full-liquid diet tolerance, and Regimen 3 (R3) denoting solid food tolerance. The primary outcome of the study was the occurrence of superficial surgical site infection (SSSI) within 30 days after surgery. Superficial surgical site infections were defined according to the Centers for Disease Control and Prevention criteria and corresponded to CDC wound class I (clean), involving only the skin and subcutaneous tissue, and manifested as clinically relevant postoperative wound discharge requiring evaluation and treatment. Secondary outcomes included length of hospital stay, time to return to regular oral intake, and other postoperative recovery parameters [17]. Wound culture specimens were collected from all patients experiencing this complication, with antimicrobial treatment coordinated through our infectious disease specialists. Hospital discharge was permitted following negative culture results or complete resolution of drainage.

SSSI served as the clinical indicator of postoperative complications. We divided patients into two cohorts according to SSSI presence or absence. Following demonstration of statistically significant IBI differences between cohorts, we performed receiver operating characteristic (ROC) curve analysis to establish the optimal IBI threshold for SSSI prediction. Using this threshold, we categorized patients as Group low IBI (IBI < 24.271) and Group high IBI (IBI ≥ 24.271).

Statistical Analysis

We conducted statistical analyses using IBM SPSS Statistics version 23 (released in 2015; IBM Corp., Armonk, NY, USA). Continuous data were reported as mean ± standard deviation for normally distributed data or median (interquartile range) for non-normally distributed data, whereas categorical data were displayed as frequencies and percentages. We assessed data distribution normality through the Kolmogorov Smirnov test. Clinical and laboratory parameter comparisons utilized Student's t-test for normally distributed data and Mann--Whitney U test for non-normally distributed data. Categorical variable comparisons employed the chi-square test or Fisher's exact test based on appropriateness. Statistical significance was established at p-value < 0.05. Exact p-values were reported in all tables.

RESULTS

Our study cohort comprised 86 patients who received surgical treatment for AAA. The average patient age was 67.5 ± 10.5 years, with 74 patients (86%) being male. Infrarenal cross-clamping was applied in 78 patients (90.7%). Early mortality occurred in 2 patients (2.3%), and SSSI was observed in 26 patients (30.2%).

Demographic and preoperative characteristics are summarized in Table 1. Operative and postoperative characteristics are presented in Table 2.

Coronary artery disease was significantly more frequent in the SSSI+ patient group compared to the SSSI- patient group (96.2% vs. 69.2%; p = 0.001). COPD was also more prevalent in the SSSI+ patient group, affecting 14 patients (53.8% vs. 26.2%; p = 0.015). Preoperative CRP levels were significantly higher in the SSSI+ patient group than in the SSSI- patient group (16.6 ± 15.6 mg/dL vs. 11.0 ± 15.2 mg/dL; p = 0.028). Similarly, IBI was significantly elevated in the SSSI+ patient group compared with the SSSI- patient group (52.3 ± 62.7 vs. 32.2 ± 49.0; p = 0.033). No statistically significant differences were observed between the groups with respect to other demographic or preoperative variables (Table 3).

ROC curve analysis identified an IBI cutoff value of 24.271 as the optimal threshold for predicting SSSI, with an area under the curve (AUC) of 0.646 (95% CI: 0.516–0.775; p = 0.033) (Figure 1). At this threshold, IBI demonstrated a sensitivity of 65.4% and a specificity of 63.3% for predicting SSSI.

Ejection fraction was significantly lower in Group high IBI compared to Group low IBI (54.3 ± 10.8 vs. 59 ± 5.5; p = 0.02). Neutrophil count was higher in Group high IBI (5.6 ± 1.8 $\times 10^9$/L vs. 4.8 ± 1.2 $\times 10^9$/L; p = 0.031), while Lym count was lower (1.83 ± 0.58 $\times 10^9$/L vs. 2.23 ± 0.73 $\times 10^9$/L; p = 0.008). Albumin levels were also significantly lower in Group high IBI (40.1 ± 4.4 vs. 42.3 ± 3.4; p = 0.02). CRP levels were significantly higher in Group high IBI (23.2 ± 17.8 mg/dL vs. 4 ± 3.1 mg/dL; p < 0.001) (Table 4). The two groups showed no other statistically significant differences regarding intraoperative variables. Table 5 presents these findings in detail.

In-hospital mortality occurred in two patients (2.3%) during hospitalization. Review of medical records revealed that deaths were attributable to early postoperative complications, including cardiac failure and respiratory complications; no mortality was directly related to preoperative IBI. Length of hospital stay was significantly longer in Group high IBI compared to Group low IBI (10.5 ± 7.1 days vs. 7 ± 3.7 days; p = 0.041), and the duration to transition to Regimen 3 diet was also significantly longer in Group high IBI compared to Group low IBI (166 ± 120.1 hours vs. 106.6 ± 41.4 hours; p = 0.004). SSSI occurred more frequently in Group high IBI compared to Group low IBI (17 patients [43.6%] vs. 9 patients [19.1%]; p = 0.014). No other statistically significant differences were observed between the two groups in terms of postoperative variables (Table 6).

Table 1. Baseline demographic, clinical, laboratory data of the study population

Variable	Mean ± SD / n (%)	Range (Min–Max)
Age (years)	67.5 ± 10.5	26 – 88
Male sex	74 (86.0%)	–
Weight (kg)	79.8 ± 15.2	45 – 128
Height (cm)	170.5 ± 9.2	140 – 190
Re-operation	1 (1.2%)	–
Prior CABG	21 (24.4%)	–
CAD	62 (72.1%)	–
Hypertension	74 (86.0%)	–
Hyperlipidemia	35 (40.7%)	–
Diabetes mellitus	32 (37.2%)	–
COPD	30 (34.9%)	–
Ejection fraction (%)	56.9 ± 8.6	22 – 67
WBC ($\times 10^9$/L)	8.1 ± 1.9	3.3 – 13.8
Hematocrit (%)	40.2 ± 5.5	28.6 – 52.4
Platelets ($\times 10^9$/L)	243.8 ± 72.0	94 – 539
Neutrophils ($\times 10^9$/L)	5.2 ± 1.6	2.3 – 11.2
Lymphocytes ($\times 10^9$/L)	2.05 ± 0.69	0.56 – 3.84
Creatinine (mg/dL)	0.94 ± 0.22	0.5 – 2.02
Albumin (g/L)	41.3 ± 4.0	28.8 – 48.9
Total cholesterol (mg/dL)	175.3 ± 45.4	95 – 292
Triglycerides (mg/dL)	163 ± 125	49 – 752
HDL cholesterol (mg/dL)	41.2 ± 8.9	22 – 66
LDL cholesterol (mg/dL)	100.5 ± 38.1	29 – 189
CRP (mg/dL)	12.7 ± 15.5	0.6 – 86.6

CABG: Coronary artery bypass grafting, CAD: Coronary artery disease, COPD: Chronic obstructive pulmonary disease, CRP: C-reactive protein, DM: Diabetes mellitus, EF: Ejection fraction, HCT: Hematocrit, HDL: High-density lipoprotein, HL: Hyperlipidemia, HT: Hypertension, IBI: Inflammatory burden index, LDL: Low-density lipoprotein, Lym: Lymphocytes, Ne: Neutrophils, PLT: Platelet; WBC: White blood cell count

Table 2. Baseline intraoperative and postoperative data of the study population

Variable	Mean ± SD / n (%)	Range (Min–Max)
Tube graft	29 (33.7%)	–
ABI	29 (33.7%)	–
ABF	17 (19.8%)	–
ABI + ABF combination	11 (12.8%)	–
Aneurysm repair	79 (91.9%)	–
EVAR explantation	7 (8.1%)	–
Infrarenal clamping	78 (90.7%)	–
Suprarenal clamping	8 (9.3%)	–
Inotropic support	20 (23.5%)	–
ICU stay (days)	1.8 ± 2.5	1 – 20
Total hospital stay (days)	8.6 ± 5.7	1 – 35
Early revision – bleeding	11 (12.8%)	–
Early revision – occlusion	9 (10.5%)	–
Early revision – bowel surgery	2 (2.3%)	–
Regimen 1 time (hours)	61.4 ± 45.5	0 – 240
Regimen 2 time (hours)	95 ± 61.7	0 – 360
Regimen 3 time (hours)	133 ± 90	0 – 620
Superficial surgical site infection	26 (30.2%)	–
Postoperative dialysis	4 (4.7%)	–
In-hospital mortality	2 (2.3%)	–

ABF: Aortobifemoral bypass; ABI: Aortobiiliac bypass; EVAR: Enovascular Aortic Repair; ICU: Intensive care unit; R-time: Regimen time

Table 3. Comparison of baseline and operative characteristics between SSSI- and SSSI+

Variable	SSSI (–) n = 60 (69.8%)	SSSI (+) n = 26 (30.2%)	p-value
Age (years)	66.3 ± 10.7 (Median: 67)	70.1 ± 8.2 (Median: 70.5)	0.114
Male sex	53 (88.3%)	21 (80.8%)	0.353
Prior CABG	11 (18.3%)	10 (38.5%)	0.046*
CAD	37 (61.7%)	25 (96.2%)	0.001*
Hypertension	49 (81.7%)	25 (96.2%)	0.075
Hyperlipidemia	23 (38.3%)	12 (46.2%)	0.498
Diabetes mellitus	19 (31.7%)	13 (50.0%)	0.106
COPD	16 (26.7%)	14 (53.8%)	0.015*
Ejection fraction (%)	58 ± 7.6 (Median: 60)	54.1 ± 10.3 (Median: 60)	0.093
WBC ($\times 10^9$/L)	8.09 ± 1.9 (Median: 7.8)	8.4 ± 1.9 (Median: 8)	0.454
Hematocrit (%)	40.7 ± 5.9 (Median: 41.5)	39.3 ± 4.4 (Median: 39.5)	0.279
Platelets ($\times 10^9$/L)	237.4 ± 61.5 (Median: 227)	258.5 ± 91.5 (Median: 231.5)	0.469
Neutrophils ($\times 10^9$/L)	5.1 ± 1.5 (Median: 4.8)	5.4 ± 1.6 (Median: 5.3)	0.386
Lymphocytes ($\times 10^9$/L)	2.06 ± 0.6 (Median: 1.95)	2.02 ± 0.7 (Median: 1.89)	0.814

CABG: Coronary artery bypass grafting; CAD: Coronary artery disease; COPD: Chronic obstructive pulmonary disease; CRP: C-reactive protein; DM: Diabetes mellitus; EF: Ejection fraction; HCT: Hematocrit; HDL: High-density lipoprotein; HL: Hyperlipidemia; HT: Hypertension; IBI: Inflammatory Burden Index; LDL: Low-density lipoprotein; Lym: Lymphocytes; Ne: Neutrophils; PLT: Platelets; SSSI: Superficial Surgical Site Infection; WBC: White blood cells; * Statistically significant ($p < 0.05$)

Table 3. Comparison of baseline and operative characteristics between SSSI- and SSSI+

Variable	SSSI (–) n = 60 (69.8%)	SSSI (+) n = 26 (30.2%)	p-value
Creatinine (mg/dL)	0.93 ± 0.22 (Median: 0.92)	0.95 ± 0.21 (Median: 0.89)	0.843
Albumin (g/L)	41.3 ± 4.0 (Median: 41.6)	41.3 ± 4.1 (Median: 42)	0.932
Total cholesterol (mg/dL)	175.2 ± 46.7 (Median: 170)	175.4 ± 43.1 (Median: 175)	0.982
Triglycerides (mg/dL)	149.7 ± 95.2 (Median: 118)	192.6 ± 174.5 (Median: 133.5)	0.629
HDL (mg/dL)	41.7 ± 9.3 (Median: 42)	40 ± 8.1 (Median: 41)	0.434
LDL (mg/dL)	101 ± 41 (Median: 98)	99.3 ± 31.4 (Median: 96.5)	0.851
CRP (mg/dL)	11 ± 15.2 (Median: 5)	16.6 ± 15.6 (Median: 12.6)	0.028*
IBI	32.2 ± 49 (Median: 15.6)	52.3 ± 62.7 (Median: 30.5)	0.033*

CABG: Coronary artery bypass grafting; CAD: Coronary artery disease; COPD: Chronic obstructive pulmonary disease; CRP: C-reactive protein; DM: Diabetes mellitus; EF: Ejection fraction; HCT: Hematocrit; HDL: High-density lipoprotein; HL: Hyperlipidemia; HT: Hypertension; IBI: Inflammatory Burden Index; LDL: Low-density lipoprotein; Lym: Lymphocytes; Ne: Neutrophils; PLT: Platelets; SSSI: Superficial Surgical Site Infection; WBC: White blood cells; * Statistically significant ($p < 0.05$)

Table 4. Comparison of baseline characteristics between IBI groups

Variable	IBI < 24.271 n = 47 (54.7%)	IBI ≥ 24.271 n = 39 (45.3%)	p-value
Age (years)	67.2 ± 8.6 (Median: 66)	67.8 ± 11.8 (Median: 70)	0.194
Male sex	41 (87.2%)	33 (84.6%)	0.727
Weight (kg)	80.8 ± 16.2 (Median: 82)	78.6 ± 14.0 (Median: 78)	0.503
Height (cm)	169.6 ± 8.0 (Median: 172)	171.5 ± 10.5 (Median: 172)	0.353
Re-operation	1 (2.6%)	0 (0%)	0.453
Prior CABG	12 (25.5%)	9 (23.1%)	0.792
CAD	31 (66.0%)	31 (79.5%)	0.164
Hypertension	40 (85.1%)	34 (87.2%)	0.782
Hyperlipidemia	22 (46.8%)	13 (33.3%)	0.205
Diabetes mellitus	14 (29.8%)	18 (46.2%)	0.118
COPD	16 (34.0%)	14 (35.9%)	0.857
Ejection fraction (%)	59 ± 5.5 (Median: 60)	54.3 ± 10.8 (Median: 60)	0.020*
WBC ($\times 10^9$/L)	7.9 ± 1.7 (Median: 7.7)	8.4 ± 2.1 (Median: 8.3)	0.275
Hematocrit (%)	40.5 ± 5.6 (Median: 41.7)	39.9 ± 5.3 (Median: 39.5)	0.573
Platelets ($\times 10^9$/L)	232 ± 71.5 (Median: 219)	258 ± 70.8 (Median: 247)	0.108
Neutrophils ($\times 10^9$/L)	4.8 ± 1.2 (Median: 4.6)	5.6 ± 1.8 (Median: 5.4)	0.031*
Lymphocytes ($\times 10^9$/L)	2.23 ± 0.73 (Median: 2.22)	1.83 ± 0.58 (Median: 1.76)	0.008*
Creatinine (mg/dL)	0.93 ± 0.23 (Median: 0.93)	0.94 ± 0.20 (Median: 0.89)	0.955
Albumin (g/L)	42.3 ± 3.4 (Median: 42.6)	40.1 ± 4.4 (Median: 40.7)	0.020*
Total cholesterol (mg/dL)	183.4 ± 46.1 (Median: 183)	165.3 ± 43.0 (Median: 157.5)	0.087
Triglycerides (mg/dL)	175.6 ± 131.4 (Median: 124.5)	147.7 ± 118.3 (Median: 114.5)	0.364
HDL (mg/dL)	42.6 ± 9.7 (Median: 42.5)	39.4 ± 7.6 (Median: 40)	0.104
LDL (mg/dL)	104.7 ± 38.4 (Median: 101)	95.5 ± 37.7 (Median: 91.5)	0.277
CRP (mg/dL)	4 ± 3.1 (Median: 3.4)	23.2 ± 17.8 (Median: 16)	< 0.001*

CABG: Coronary artery bypass grafting, CAD: Coronary artery disease, COPD: Chronic obstructive pulmonary disease, CRP: C-reactive protein, EF: Ejection fraction, HDL: High-density lipoprotein IBI: Inflammatory burden index, LDL: Low-density lipoprotein, WBC: White blood cells; * Statistically significant ($p < 0.05$)

Table 5. Comparison of operative characteristics between IBI groups

Variable	IBI < 24.271 n = 47 (54.7%)	IBI ≥ 24.271 n = 39 (45.3%)	p-value
Tube graft	13 (27.7%)	16 (41.0%)	0.192
ABI	18 (38.3%)	11 (28.2%)	0.324
ABF	12 (25.5%)	5 (12.8%)	0.141
Aneurysm repair	42 (89.4%)	37 (94.9%)	0.352
EVAR explantation	5 (10.6%)	2 (5.1%)	0.352
Infrarenal clamping	43 (91.5%)	35 (89.7%)	0.781
Suprarenal clamping	4 (8.5%)	4 (10.3%)	0.781

ABF: Aortobifemoral bypass, ABI: Aortobiiliac bypass, EVAR: Endovascular aortic repair, IBI: Inflammatory burden index, ICU: Intensive care unit; * Statistically significant ($p < 0.05$)

Table 6. Comparison of postoperative characteristics between IBI groups

Variable	IBI < 24.271 n = 47 (54.7%)	IBI ≥ 24.271 n = 39 (45.3%)	p-value
ICU stay (days)	1.4 ± 0.9 (Median: 1)	2.3 ± 3.5 (Median: 1)	0.152
Hospital stay (days)	7 ± 3.7 (Median: 6)	10.5 ± 7.1 (Median: 7)	0.041*
Early revision – bleeding	5 (10.6%)	6 (15.4%)	0.512
Early revision – occlusion	5 (10.6%)	4 (10.3%)	0.954
Early revision – bowel surgery	1 (2.1%)	1 (2.6%)	> 0.99
Regimen 1 time (hours)	53.6 ± 34.5 (Median: 48)	70.7 ± 55.0 (Median: 48)	0.226
Regimen 2 time (hours)	81.3 ± 39.3 (Median: 72)	111.6 ± 78.4 (Median: 72)	0.154
Regimen 3 time (hours)	106.6 ± 41.4 (Median: 96)	166 ± 120.1 (Median: 126)	0.004*
SSSI	9 (19.1%)	17 (43.6%)	0.014*
Postoperative dialysis	1 (2.1%)	3 (7.7%)	0.325
In-hospital mortality	1 (2.1%)	1 (2.6%)	> 0.99

IBI: Inflammatory burden index, ICU: Intensive care unit, R-time: Regimen time, SSSI: Superficial surgical site infection; * Statistically significant ($p < 0.05$)

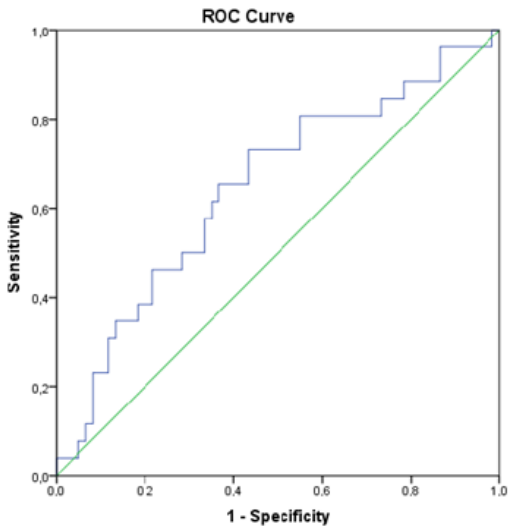

Figure 1. Receiver operating characteristic (ROC) curve for IBI predicting superficial surgical site infection following open AAA repair

DISCUSSION

Our investigation demonstrated that preoperative IBI is a valuable prognostic marker for postoperative outcomes following open abdominal aortic aneurysm repair. Patients with higher preoperative IBI values exhibited significantly increased rates of superficial surgical site infection, delayed return to a regular diet, and prolonged length of hospital stay. To the best of our knowledge, this study represents one of the first investigations to evaluate the association between IBI and postoperative outcomes in vascular surgery. These findings suggest that an increased preoperative inflammatory burden, as quantified by IBI, may be associated with impaired postoperative recovery and wound healing in patients undergoing open AAA repair.

IBI is a novel index that combines two readily available laboratory parameters---CRP and the neutrophil-to-lymphocyte ratio (NLR)---into a single, practical measure of systemic inflammation. Its utilization has expanded across multiple

medical specialties in recent years, particularly within oncology, where it has demonstrated superior prognostic capability compared to isolated inflammatory parameters [15]. The results obtained to elucidate the relationship between prognosis and inflammation in vascular diseases are noteworthy [18]. Consequently, the predictive capability exhibited by IBI merits additional exploration concerning its relevance to vascular surgical patient cohorts, particularly in relation to postoperative outcomes following AAA repair.

Multiple prognostic scoring systems, including Glasgow Aneurysm Score (GAS) and V-POSSUM, have been developed to estimate mortality risk in AAA repair [19]. These models, initially constructed for ruptured AAA scenarios, incorporate preoperative demographic information and intraoperative variables. With EVAR reducing mortality rates in AAA treatment, clinical attention has transitioned toward reducing morbidity associated with open surgical approaches. For elective procedures, incorporating physiological biomarkers like IBI may improve predictive performance for postoperative complications. Integration of IBI into risk assessment algorithms could facilitate adoption of intensive perioperative management strategies---including rigorous glycemic control, tailored antimicrobial protocols, and enhanced bowel preparation---for open AAA repair.

Systemic broad-spectrum antimicrobial prophylaxis during arterial reconstruction procedures is well-established in clinical practice guidelines [20]. Nevertheless, questions have emerged regarding whether alterations in interstitial fluid distribution during open AAA repair might compromise antibiotic effectiveness [21]. Perioperative fluid resuscitation, vasopressor therapy, and inotropic medication administration may additionally diminish prophylactic antibiotic efficacy [21]. A retrospective study by Greenblatt et al. documented a 30-day readmission rate of 12.8% after open AAA repair, with wound complications accounting for 17.6% of these readmissions [22]. Our investigation confirms a distinct relationship between elevated IBI and surgical site infection occurrence.

Additionally, patients with wound discharge were more likely to have lower preoperative albumin levels, as well as comorbidities such as coronary artery disease and COPD. It is well established that CRP and NLR are elevated in heart failure and coronary artery disease [23]. These patients may have underlying endothelial dysfunction and compromised tissue perfusion, which could impair wound healing. Thus, IBI appears to reflect the overall risk profile of the patient, functioning as more than just a laboratory marker.

An additional significant observation was the relationship between elevated IBI and extended gastrointestinal recovery time, reflected by a longer time to transition to R3. Postoperative ileus and delayed bowel function recovery following open abdominal procedures are recognized consequences of systemic inflammatory response and physiologic stress [24]. Patients with elevated IBI may exhibit an exaggerated inflammatory response, leading to intestinal edema and reduced motility. Alternatively, some patients may have subclinical chronic peritonitis or mesenteric inflammation that delays bowel recovery even in the absence of infection.

Patients undergoing vascular surgery have higher readmission rates compared to those in other surgical specialties [25]. In the prospective RELIEF trial by Bain et al., elevated CRP levels were linked to significantly longer hospital stays, highlighting the impact of postoperative inflammation on patient-centered outcomes [26]. In our study, wound discharge and delayed gastrointestinal recovery were key contributors to prolonged hospitalization. Therefore, IBI may serve as a valuable indicator of postoperative complications and can be integrated into preoperative risk stratification to potentially reduce healthcare costs and optimize resource utilization.

The incidence of SSSI in the present study was higher than the general range reported in the literature for vascular surgery. This difference can be attributed to several factors specific to our study. First, our definition of SSSI included all clinically relevant postoperative wound discharge requiring evaluation and treatment, rather than only culture-confirmed or deep surgical site infections. This broader definition captures a wider spectrum of wound complications. Second, all patients underwent open abdominal aortic aneurysm repair via a midline transperitoneal approach, which is associated with inherently higher wound complication rates compared to less invasive procedures or endovascular approaches. Third, patient-specific factors such as the high prevalence of comorbidities may have contributed to impaired wound healing. Accordingly, the reported SSSI rate should be interpreted within the context of the study design, patient population characteristics, and the specific surgical approach employed.

Limitations

This investigation possesses several limitations. First, its retrospective design and single-center nature may limit the generalizability of the findings, and the sample size was relatively small. Second, although routinely obtained preoperative laboratory parameters were analyzed, certain potential confounding factors that may influence postoperative outcomes---such as nutritional status, perioperative glycemic control, and other unmeasured metabolic or inflammatory conditions---were not systematically evaluated. Third, this study focused exclusively on superficial surgical site infections, which were the most frequently observed infectious complications in the cohort; deeper surgical site infections, including graft or deep tissue infections, were not assessed and therefore could not be analyzed. In addition, the inflammatory burden index was the only composite inflammatory marker evaluated, and the optimal cutoff value was determined using receiver operating characteristic analysis. Finally, whether IBI is specific to abdominal aortic aneurysm pathology or reflects

a more general inflammatory response remains uncertain. Larger-scale, prospective, randomized controlled investigations are required to determine the most suitable IBI cutoff value for patients receiving AAA repair.

CONCLUSION

Elevated preoperative IBI is associated with increased postoperative outcomes in patients undergoing open AAA repair. These patients were more likely to develop surgical site infections, experienced delayed return to normal diet, and had longer hospital stays. We therefore suggest that IBI may serve as a practical predictive marker for postoperative outcomes in open AAA repair.

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Patient Consent for Publication: This study was conducted in accordance with the Declaration of Helsinki. Due to the retrospective nature of the study, informed consent forms were waived. Confidentiality of personal data was meticulously protected, and patient identities were anonymized.

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