



Received: 2025.07.08

Accepted: 2025.09.19

Available online: 2025.10.01

Published: 2025.11.18

Predictive Value of IBI for In-Hospital Death in Elderly Patients with Non-ST-Segment Elevation Myocardial Infarction

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Financial support: This work was financially supported by the National Natural Science Foundation of China (81703213) and the Natural Science Youth Foundation of Jiangsu Province of China (BK20151034)

Conflict of interest: None declared

Background: Non-ST-segment elevation myocardial infarction (NSTEMI) has a with high incidence rate and a high mortality rate in elderly patients, and inflammation plays an important role. As a useful inflammatory marker, the relationship between the inflammatory burden index (IBI) and in-hospital death of elderly patients with NSTEMI remains unclear. The aim of this study was to investigate the predictive value of IBI for in-hospital death in elderly patients with NSTEMI.

Material/Methods: This single-center study retrospectively enrolled patients diagnosed with NSTEMI between February 2021 and February 2025. All patients were ≥ 75 years old and did not receive percutaneous coronary intervention (PCI) treatment during hospitalization. Patients were divided into 2 groups according to whether cardiogenic death occurred during hospitalization. IBI was calculated as the product of C-reactive protein and the neutrophil-to-lymphocyte ratio.

Results: This study enrolled a total of 418 patients, with a mean age of 79.60 ± 3.67 years. During the hospitalization period, cardiogenic death occurred in 43 (10.3%) patients. After adjusting for possible confounding factors, multivariate logistic regression analysis showed that IBI (OR=2.22, 95% CI: 1.64-3.00) was an independent risk factor for in-hospital death in elderly patients with NSTEMI. Restricted cubic spline suggested a non-linear dose-response relationship between IBI and in-hospital death. The results of ROC showed that the area under the curve of IBI was 0.760.

Conclusions: In elderly patients with NSTEMI, IBI demonstrated an independent association with in-hospital mortality, with modest discriminatory performance. There is a non-linear dose-response relationship between IBI and in-hospital death in elderly patients with NSTEMI.

Keywords: Age Groups • Cardiology • Death • Inflammation

Full-text PDF: <https://www.medscimonit.com/abstract/index/idArt/950592>

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Introduction

With the aging of the population, the treatment of elderly patients (≥ 75 years old) with non-ST-segment elevation myocardial infarction (NSTEMI) has become an important challenge in the field of cardiology [1]. Clinically, due to the decline in physiological reserve, complex comorbidities, and poor treatment tolerance, elderly NSTEMI patients exhibit a relatively high risk of in-hospital death [2]. Although primary percutaneous coronary intervention (PCI) can significantly reduce the risk of in-hospital death, real-world data show that only one-fourth of elderly NSTEMI patients receive PCI treatment [3,4]. In a clinical trial of frail elderly patients with NSTEMI, the routine invasive strategy provided no benefits [5]. Therefore, it is necessary to explore more risk factors for elderly NSTEMI patients who have not undergone PCI treatment to identify high-risk patients at an early stage.

Inflammation plays a crucial role in the pathogenesis, disease progression, and prognosis of elderly patients with NSTEMI [6]. In recent years, the inflammatory burden index (IBI), as an emerging inflammatory biomarker, has been widely applied in the field of risk stratification for various diseases [7-13]. Compared with single inflammatory markers, IBI, which is calculated as the product of C-reactive protein (CRP) and the neutrophil-to-lymphocyte ratio, can more comprehensively and stably assess the body's inflammatory status, demonstrating higher accuracy in reflecting the degree of inflammation and predicting the prognosis [9-11]. Previous studies have confirmed that there is an independent association between IBI levels and the prevalence of cardiovascular diseases [11,12]. In another study, IBI was proven to be an independent predictor of all-cause mortality in patients with osteoarthritis [13]. However, in elderly patients with NSTEMI, the relationship between IBI and in-hospital death remains unclear. This study aims to evaluate the predictive efficacy of IBI for cardiogenic death during hospitalization in elderly NSTEMI patients. In line with the latest NSTEMI guideline [14], we set the age threshold at ≥ 75 years in this study to better capture a population with a higher burden of frailty, multimorbidity, and competing risks.

Material and Methods

Study Population

This single-center retrospective study consecutively enrolled patients diagnosed with NSTEMI at the First Affiliated Hospital of Nanjing Medical University between February 2021 and February 2025. NSTEMI was defined in accordance with the latest guidelines [14,15]. Briefly, NSTEMI was characterized by a rise and/or fall in cardiac injury biomarkers – primarily high-sensitivity cardiac troponin – with at least 1 value above the 99th percentile

upper reference limit, accompanied by clear evidence of ischemia and no persistent ST-segment elevation on ECG. The study protocol was reviewed and approved by the Institutional Review Board (IRB) of the First Affiliated Hospital of Nanjing Medical University (2025-SR-272). This research was conducted in accordance with the Declaration of Helsinki. Given that the study posed no risks to the patients, the IRB waived the requirement for informed consent. The inclusion criteria were as follows: (1) aged ≥ 75 years; (2) not receiving PCI treatment; (3) having complete clinical data. The exclusion criteria were: (1) a history of myocardial infarction (MI); (2) comorbid inflammatory diseases or autoimmune diseases; (3) severe renal insufficiency (estimated glomerular filtration rate < 30 mL/min/1.73 m²); (4) active bleeding in vital organs; (5) patients with malignant tumors or hematological diseases. We excluded patients with a history of MI to ensure a more homogeneous index-pathophysiology at presentation and to minimize confounding due to prior infarct-related remodeling, revascularization, and secondary prevention therapies. Patients were divided into 2 groups according to whether cardiogenic death occurred during hospitalization. Given the retrospective design, we provide a brief post hoc power/precision justification for the primary endpoint (in-hospital cardiogenic death). We assumed an event rate of 10%, set $\alpha=0.05$, and targeted 80-90% power. The effect size for IBI was specified as an adjusted odds ratio per 1-SD (or per log-unit) increase. Based on these assumptions, we evaluated power for the available sample and confirmed that the final sample size is adequate to detect the prespecified effect size and to yield precise estimates of model performance (including sensitivity and specificity with 95% CI).

Clinical Data Collection

The clinical data of all enrolled patients were collected based on their medical records, encompassing information such as age, sex, body mass index (BMI), previous medical history, and medications administered. The lymphocyte count, neutrophil count, and CRP levels measured for the first time after the patients' admission were recorded. To more effectively reflect myocardial injury and cardiac function status, the recorded high-sensitivity troponin T (hs-TNT) and B-type natriuretic peptide (BNP) were peak values during hospitalization. The neutrophil-to-lymphocyte ratio (NLR) was defined as the ratio obtained by dividing the neutrophil count by the lymphocyte count. The IBI was defined as the product of CRP and NLR [9-11]. The medications recorded included aspirin, P2Y₁₂ inhibitors, β -blockers, statins, sodium-glucose cotransporter 2 inhibitors, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers.

Statistical Analysis

To assess the normality of continuous variables, the Kolmogorov-Smirnov test was employed. Normally distributed

and non-normally distributed continuous data were statistically analyzed using the independent samples t-test and Mann-Whitney U test, respectively. The comparison of categorical variables between the 2 groups was performed using the chi-square test. Logistic regression analysis was used to explore risk factors associated with in-hospital death. Specifically, based on prior literature and pathophysiology, the following potentially relevant variables were incorporated into multivariate regression analysis via a stepwise forward method: IBI, age, sex, hypertension, diabetes mellitus, systolic blood pressure, heart rate, Killip class, baseline antithrombotic therapy, left ventricular ejection fraction (LVEF), hs-TNT, and BNP. The receiver operating characteristic curve (ROC) was used to evaluate the predictive efficacy of the IBI for in-hospital death, and the DeLong test was used to compare the differences between the areas under the curve (AUCs). The primary threshold selection rule was the Youden Index, with sensitivity/specificity-targeted alternative cut-offs considered in scenario analyses. We estimated sensitivity, specificity, and their 95% CIs at the selected thresholds [16]. Restricted cubic splines (RCS) were used to explore the dose-response relationship between IBI and in-hospital death. SPSS (Version 27.0, Chicago, USA) and R (Lucent Technologies, New Jersey, USA) were used for statistical analysis. Statistical significance was defined as a P value <0.05 .

Results

Baseline Data Comparison Between Groups

A total of 418 elderly patients with NSTEMI were enrolled in this study, and cardiogenic in-hospital deaths occurred in 43 (10.3%) (Figure 1). The mean age of the patients was 79.60 ± 3.67 years, and males accounted for 66.03% of the total. The analysis of baseline data showed that, compared with the surviving elderly NSTEMI patients, the patients with cardiogenic death had significantly higher levels of the IBI, CRP, NLR, neutrophil count, and BNP, and a significantly higher proportion of patients with KILLIP >1 . In contrast, the left ventricular ejection fraction (LVEF) and lymphocyte count were significantly lower ($P<0.05$) (Table 1).

Association Between the IBI and In-Hospital Death in Elderly Patients with NSTEMI

Considering that the values were relatively large, IBI, hs-TNT, and BNP were logarithmically transformed and then subjected to logistic regression analysis. All variables were included in the univariate logistic regression analysis. The results demonstrated that the IBI, LVEF, KILLIP >1 , lymphocyte count, CRP, and BNP were associated with cardiogenic death during hospitalization in elderly patients with NSTEMI (Table 2). To avoid

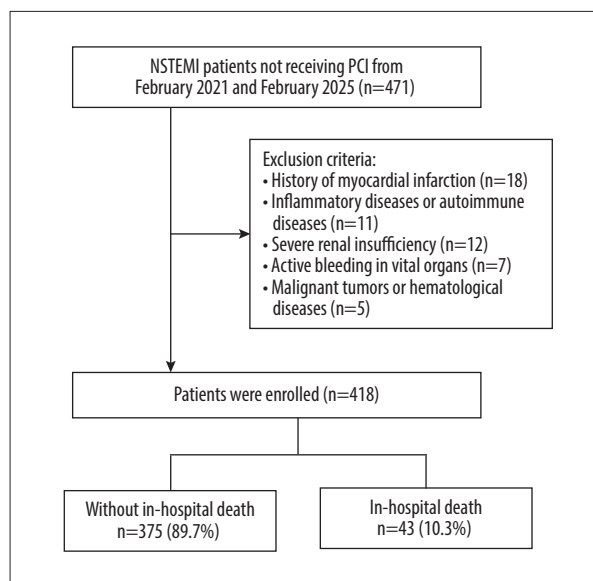


Figure 1. Study flowchart. NSTEMI, non-ST-segment elevation myocardial infarction. This figure was generated using Microsoft PowerPoint, Microsoft, Redmond, WA, USA.

the interference of collinearity, the relevant variables were incorporated into the multivariate logistic regression analysis using the stepwise forward method. The results showed that IBI (OR=2.22, 95% CI: 1.64-3.00), BNP, and LVEF were independent predictive risk factors for cardiogenic death during hospitalization in elderly patients with NSTEMI (Table 3). The results of RCS suggested a non-linear dose-response relationship between IBI and in-hospital death (p for nonlinearity <0.001 , P for overall <0.001), showing that the higher the IBI, the greater the risk of in-hospital death (Figure 2).

ROC Curves for In-Hospital Death in Elderly Patients with NSTEMI

The results of ROC showed that the area under the curve (AUC) of IBI was 0.760 (cut-off value: 36.85, 95% CI: 0.689-0.832, $P<0.001$), the AUC of CRP was 0.676 (cut-off value: 2.41, 95% CI: 0.600-0.753, $P<0.001$), and the AUC of NLR was 0.710 (cut-off value: 9.12, 95% CI: 0.639-0.782, $P<0.001$). The DeLong test demonstrated that the discriminatory ability of IBI for cardiogenic death during hospitalization in elderly NSTEMI patients was significantly superior to that of CRP ($z=3.967$, $P<0.001$), while there was no statistically significant difference with NLR ($z=1.398$, $P=0.162$) (Figure 3, Table 4).

Discussion

This study explored the relationship between IBI and in-hospital death in elderly patients with NSTEMI. It was found that IBI can independently predict in-hospital mortality in elderly

Table 1. Baseline patient characteristics.

Variables	Total (n=418)	Survival (n=375)	Death (n=43)	P
Age, years	79.60±3.67	79.54±3.68	80.12±3.56	0.329
Male, n (%)	276 (66.03)	250 (66.67)	26 (60.47)	0.416
BMI, kg/m ²	21.83±3.53	21.77±3.37	22.30±4.72	0.476
Heart rate, bpm	79.71±14.38	79.47±14.34	81.74±14.82	0.327
SBP, mmHg	117.11±20.86	117.42±20.59	114.42±23.20	0.372
DBP, mmHg	72.67±14.38	72.90±14.20	70.65±15.83	0.333
Total cholesterol, mmol/L	4.42±1.04	4.44±1.04	4.26±1.02	0.295
Triglycerides, mmol/L	1.48±0.96	1.45±0.90	1.69±1.39	0.288
LDL-C, mmol/L	2.77±0.92	2.77±0.91	2.81±1.08	0.811
HDL-C, mmol/L	1.04±0.24	1.04±0.24	1.06±0.24	0.596
LVEF, %	43.38±6.38	43.65±6.37	40.95±6.04	0.008
CRP, mg/L	3.35 (1.01, 8.20)	3.23 (0.94, 6.90)	6.80 (3.08, 9.81)	<.001
WBC, 10 ⁹ /L	10.30±3.16	10.20±3.15	11.19±3.20	0.052
Neutrophil, 10 ⁹ /L	8.18±4.21	8.02±4.29	9.54±3.16	0.025
Lymphocyte, 10 ⁹ /L	1.70±1.26	1.77±1.30	1.12±0.46	<.001
Platelet, 10 ⁹ /L	215.16±58.67	216.67±58.67	201.95±57.69	0.119
IBI	36.61±53.84	32.42±49.58	73.18±73.20	<.001
NLR	5.70 (3.14, 9.06)	5.42 (2.97, 8.74)	9.26 (5.33, 13.01)	<.001
BNP, ng/L	2486.7 (1486.7, 4718.9)	2380.7 (1427.1, 4395.0)	4247.0 (2156.0, 7917.7)	<.001
hs-TnT, ng/L	3769.5 (1538.9, 6502.3)	3680.0 (1541.8, 6267.5)	4622.0 (1511.5, 8596.0)	0.150
Hypertension, n (%)	198 (47.37)	174 (46.40)	24 (55.81)	0.242
Diabetes mellitus, n (%)	123 (29.43)	109 (29.07)	14 (32.56)	0.634
Stroke, n (%)	65 (15.55)	58 (15.47)	7 (16.28)	0.889
Smoking, n (%)	172 (41.15)	154 (41.07)	18 (41.86)	0.920
KILLIP >1, n (%)	103 (24.64)	86 (22.93)	17 (39.53)	0.017
Length of stay, days	7.67±2.57	7.62±2.20	8.12±2.27	0.230
P2Y12 inhibitors, n (%)	398 (95.22)	358 (95.47)	40 (93.02)	0.738
Aspirin, n (%)	383 (91.63)	344 (91.73)	39 (90.70)	1.000
Statins, n (%)	390 (93.30)	351 (93.60)	39 (90.70)	0.690
SGLT2i, n (%)	87 (20.81)	77 (20.53)	10 (23.26)	0.677
ACEI/ARB, n (%)	210 (50.24)	189 (50.40)	21 (48.84)	0.846
β-blockers, n (%)	313 (74.88)	280 (74.67)	33 (76.74)	0.766
Spironolactone, n (%)	59 (14.11)	50 (13.33)	9 (20.93)	0.175

BMI – body mass index; LVEF – left ventricular ejection fraction; HDL-C – high-density leptin cholesterol; LDL-C – low-density leptin cholesterol; CRP – C-reactive protein; FBG – fasting blood glucose; NLR – neutrophil/lymphocyte; IBI – inflammatory burden index; hs-TnT – high-sensitivity troponin T; BNP – B-type natriuretic peptide; ACEI – angiotensin-converting enzyme inhibitors; ARB – angiotensin receptor blocker; SGLT2i – sodium-glucose cotransporter 2 inhibitors.

Table 2. Univariate regression analysis for in-hospital death in elderly patients with NSTEMI.

Variables	OR	95% CI	P
Male, n (%)	1.31	0.68~2.50	0.417
Hypertension, n (%)	1.46	0.77~2.75	0.244
Diabetes mellitus, n (%)	1.18	0.60~2.32	0.634
Stroke, n (%)	1.06	0.45~2.50	0.889
Smoking, n (%)	1.03	0.54~1.96	0.920
KILLIP >1, n (%)	2.20	1.14~4.24	0.019
P2Y12 inhibitor, n (%)	0.63	0.18~2.25	0.481
Aspirin, n (%)	0.88	0.29~2.62	0.816
Statins, n (%)	0.67	0.22~2.02	0.474
SGLT2i, n (%)	1.17	0.55~2.48	0.677
ACEI/ARB, n (%)	0.94	0.50~1.77	0.846
β-blockers, n (%)	1.12	0.53~2.36	0.766
Spirinolactone, n (%)	1.72	0.78~3.80	0.180
IBI	2.08	1.56~2.78	<.001
WBC, 10 ⁹ /L	1.09	1.00~1.20	0.054
Neutrophil, 10 ⁹ /L	1.06	1.00~1.12	0.071
Lymphocyte, 10 ⁹ /L	0.34	0.19~0.63	<.001
Platelet, 10 ⁹ /L	1.00	0.99~1.00	0.119
CRP, mg/L	1.15	1.07~1.24	<.001
NLR	1.07	1.03~1.11	0.001
Age, years	1.04	0.96~1.13	0.329
BMI, kg/m ²	1.04	0.96~1.14	0.350
Heart rate, bpm	1.01	0.99~1.03	0.327
SBP, mmHg	0.99	0.98~1.01	0.371
DBP, mmHg	0.99	0.97~1.01	0.332
BNP, ng/L	1.75	1.26~2.44	<.001
hs-TnT, ng/L	1.21	0.85~1.73	0.293
Triglycerides, mmol/L	0.84	0.61~1.16	0.294
Total cholesterol, mmol/L	1.22	0.93~1.58	0.145
LDL-C, mmol/L	1.04	0.74~1.46	0.811
HDL-C, mmol/L	1.41	0.39~5.08	0.596
LVEF, %	0.94	0.89~0.98	0.009

BMI – body mass index; LVEF – left ventricular ejection fraction; HDL-C – high-density leptin cholesterol; LDL-C – low-density leptin cholesterol; CRP – C-reactive protein; FBG – fasting blood glucose; NLR – neutrophil/lymphocyte; IBI – inflammatory burden index; hs-TnT – high-sensitivity troponin T; BNP – B-type natriuretic peptide; ACEI – angiotensin-converting enzyme inhibitors; ARB – angiotensin receptor blocker; SGLT2i – sodium-glucose cotransporter 2 inhibitors; NSTEMI – non-ST-segment elevation myocardial infarction.

Table 3. Multivariate regression analysis for in-hospital death in elderly patients with NSTEMI.

Variables	OR	95% CI	P
IBI	2.22	1.64~3.00	<.001
BNP	1.84	1.26~2.69	0.002
LVEF	0.94	0.90~1.00	0.034

Adjusted variables including IBI, age, sex, hypertension, diabetes mellitus, systolic blood pressure, heart rate, Killip class, baseline antithrombotic therapy, LVEF, high-sensitivity troponin T, and BNP. IBI – inflammatory burden index; BNP – B-type natriuretic peptide; LVEF – left ventricular ejection fraction; NSTEMI – non-ST-segment elevation myocardial infarction.

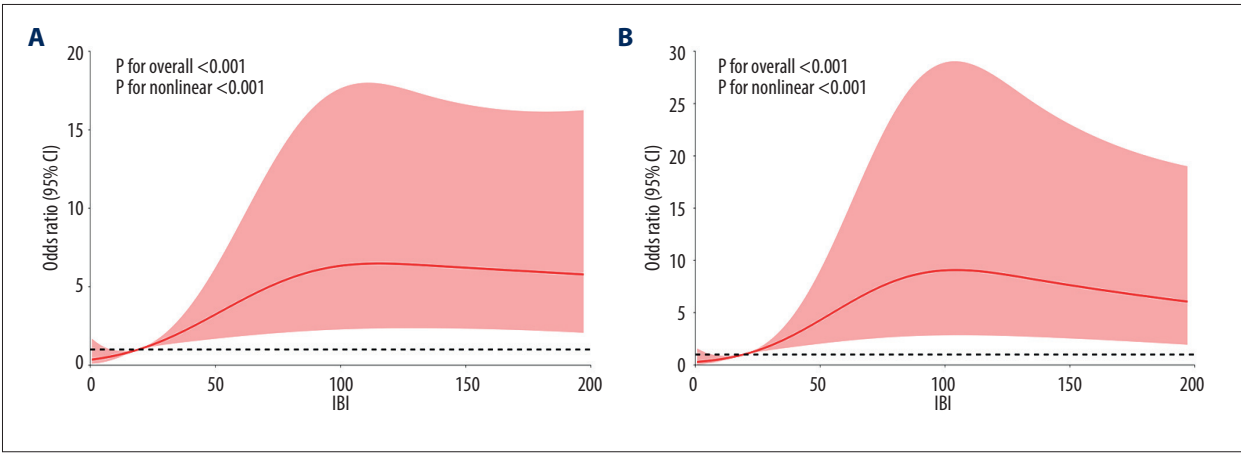


Figure 2. Dose-response relationship between IBI and in-hospital death in elderly patients with NSTEMI. (A) Unadjusted dose-response relationship between IBI and in-hospital death; (B) Adjusted dose-response relationship between IBI and in-hospital death. IBI – inflammatory burden index; NSTEMI – non-ST-segment elevation myocardial infarction. This figure was drawn using R 4.3.1 and generated using Microsoft PowerPoint, Microsoft, Redmond, WA, USA.

patients with NSTEMI, and there is a non-linear dose-response relationship exists between IBI and in-hospital death risk.

In the elderly population, NSTEMI is characterized by a high incidence rate and a high mortality rate [1]. In a prior study, Luo et al reported an in-hospital mortality of 10.7% among older NSTEMI patients aged ≥ 75 years, which is consistent with our findings [17]. This phenomenon is not only related to the frailty, complex coronary artery lesions, progressive decline of multiple organ functions, and poor treatment tolerance, but also is closely linked to inflammatory imbalance [17-19]. IBI, as an emerging inflammatory assessment index, has been widely used in the risk stratification of various diseases in recent years [7-13]. A recent study found that IBI is closely associated with contrast-induced acute kidney injury in patients with ST-segment elevation myocardial infarction (STEMI) during hospitalization [11]. In another study, IBI was confirmed to be an independent risk marker for all-cause mortality in patients with osteoarthritis [13]. Given the unique pathophysiological characteristics of elderly NSTEMI patients, the present study specifically focused on this special group of patients and made innovative discoveries: elevated IBI is an independent

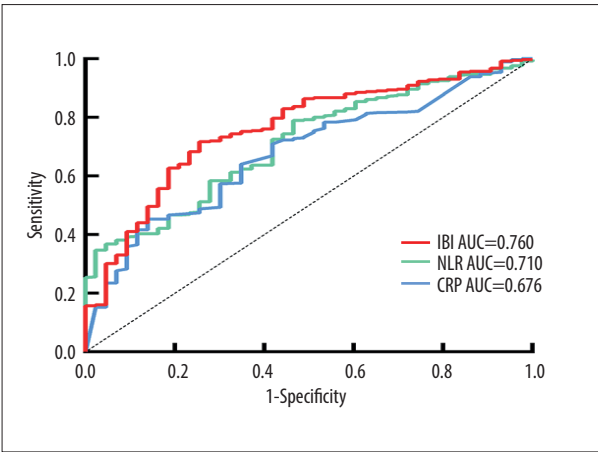


Figure 3. Receiver operating characteristic analysis (ROC) of IBI for in-hospital death in elderly patients with NSTEMI. IBI – inflammatory burden index; CRP – C-reactive protein; NLR – neutrophil-to-lymphocyte ratio; NSTEMI – non-ST-segment elevation myocardial infarction. This figure was generated using GraphPad Prism 9, GraphPad Software, USA.

Table 4. ROC curve analysis of IBI for in-hospital death in elderly patients with NSTEMI.

	AUC	95% CI	P	Cut-off	Sensitivity	Specificity
IBI	0.760	0.689–0.832	<.001	36.85	0.744	0.717
NLR	0.710	0.639–0.782	<.001	9.12	0.535	0.789
CRP	0.676	0.600–0.753	<.001	2.41	0.860	0.453

CI – confidence interval; AUC – area under curve; IBI – inflammatory burden index; CRP – C-reactive protein; NSTEMI – non-ST-segment elevation myocardial infarction.

risk factor for in-hospital death in elderly NSTEMI patients, and there is a non-linear dose-response relationship between IBI and in-hospital death. In the coronary plaques of elderly patients, the infiltration degree of inflammatory cells is significantly higher than that in younger people [20]. The active enzymes released by neutrophils further damage the integrity of the vascular endothelium, thus significantly increasing the risk of adverse events [21]. In the acute phase of MI, the excessive activation of inflammatory cells triggers an “inflammatory storm”, releasing many damage-related factors, which exacerbate the apoptosis and necrosis of myocardial cells [22,23]. Due to insufficient secretion of anti-inflammatory factors and impaired self-repair ability, elderly patients are more likely to progress to cardiogenic shock, cascading damage of multiple organ functions, and even death [24,25]. CRP, as a classic inflammatory marker, is closely related to the severity of the disease and poor prognosis in patients with MI [26]. Some studies have shown that in the early stage of the onset of acute MI, a significant increase in CRP levels is positively correlated with the expansion of the infarct area and the severity of cardiac function impairment [27,28]. Relevant studies have shown that in elderly patients with MI, CRP remains at a continuously high level, and this significantly increases the risk of adverse events such as cardiovascular death in the hospital [29,30]. In this study, we found that IBI can predict the in-hospital death of elderly NSTEMI patients more effectively than CRP alone, which is likely because IBI integrates more dimensional information, thus providing additional prognostic value. NLR reflects the balance between the body’s inflammatory and immune status and also has important clinical significance in the field of MI [31,32]. After the occurrence of acute MI, the phenomenon of increased neutrophils and decreased lymphocytes lead to an increase in NLR [33]. A cross-sectional retrospective study found that NLR is an important indicator for the risk stratification and prognosis of patients with NSTEMI [34]. In our study, although the AUC of IBI for in-hospital death was larger than that of NLR, there was no statistically significant difference between them. In addition to the fact that NLR serves as a valuable inflammatory biomarker for MI, the findings of our study might also be influenced by the limited sample size. In summary, although IBI outperformed CRP, its difference from NLR was not statistically significant.

Therefore, the incremental value of IBI over readily available clinical markers remains uncertain. It is imperative that future studies with larger sample sizes be conducted to further elucidate these relationships and findings.

Considering the high death risk of elderly NSTEMI patients, there is an urgent need for accurate prognostic assessment indicators in clinical practice. The results of this study can help clinicians screen out high-risk patients at an early stage, thereby guiding the formulation of individualized treatment strategies. For example, for patients with elevated IBI, hemodynamic monitoring can be strengthened, antithrombotic and anti-inflammatory treatment regimens can be optimized, or multidisciplinary collaborative intervention measures can be initiated earlier to improve the prognosis of patients. In addition, the convenient calculation method of IBI, which is based on routine blood routine and inflammatory indicators, gives it broad application potential in clinical practice. However, although IBI is independently associated with in-hospital mortality, its discriminatory performance is only moderate (AUC=0.76). In practice, IBI should serve as a complement to established clinical assessment rather than a standalone decision-making tool.

Limitations

First, as a retrospective study, its capacity for causal inference is limited and it is prone to selection bias, information bias, and unmeasured confounding. Second, our findings are primarily applicable to older NSTEMI patients aged ≥75 years, a group characterized by greater frailty and competing risks. Accordingly, caution is warranted when extrapolating these results to younger populations. Third, our study focused exclusively on in-hospital cardiogenic death. Whether IBI is associated with post-discharge cardiogenic mortality remains to be determined and will require future follow-up data for further clarification. Fourth, advanced age is often associated with frailty. Because this study was retrospective and frailty assessment is not routinely performed at our center, relevant data were unavailable. Consequently, the absence of a standardized measure of frailty limited our ability to control for confounding. Future prospective studies should incorporate validated frailty scales to enhance the adequacy of risk adjustment

and the interpretability of results. Fifth, in our study, the IBI cut-off was set at 36.85, yielding a sensitivity of 74.4% and a specificity of 71.7%. Depending on the intended clinical application, higher sensitivity may be prioritized for rule-out and higher specificity for rule-in. It should be noted that these are in-sample estimates and may be subject to optimism; stronger evidence requires external validation in independent cohorts.

Conclusions

In elderly patients with NSTEMI, IBI demonstrated an independent association with in-hospital mortality, with modest discriminatory performance. There is a non-linear dose-response relationship between IBI and in-hospital death in elderly patients with NSTEMI.

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Patient Permission/Consent Declarations

This was a retrospective study. Given that the study posed no risks to the patients, the IRB waived the requirement for informed consent.

Data Availability Statement

The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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