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ORIGINAL RESEARCH

Prediction of in-hospital mortality in patients with aortic dissection: a new inflammation-related biomarker

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Abstract

Background: Acute aortic dissection (AAD) is a rare cardiovascular emergency condition associated with high mortality. Neutrophil lymphocyte ratio (NLR) is an inflammation-related biomarker. Studies on NLR suggest that NLR is instrumental in predicting prognosis in patients with AAD. Hyperglycemia is associated with impaired hemostasis, and may induce endothelial dysfunction, oxidative stress, and inflammatory response. The authors developed a neutrophil-lymphocyte glycemic index (NLGI). This study aims to explore the potential of the NLGI in improving the prediction of mortality risk in patients hospitalized with AAD. **Methods**: Designed as retrospective observational research, this study was conducted on patients admitted to and diagnosed with AAD at the emergency department (ED) between 2017 and 2024. Patients aged 18 years and older who were surgically treated and had a confirmed diagnosis of AAD by thoracoabdominal computed tomography (CT) angiography were included in the study. **Results**: A total of 73 patients were included in the study. The patients' ages ranged from 37 to 97 years, with a mean age of 67.53 $\pm$ 14.10 years. Of the patients, 72.6% were men. In-hospital mortality occurred in 33 patients. Receiver Operating Characteristics (ROC) analysis was performed to investigate the performance of the variables in predicting in-hospital mortality. NLGI (Area Under Curve (AUC): 0.725) had the best performance, followed by neutrophil glycemic index (NGI, AUC: 0.699) and NLR (AUC: 0.683). **Conclusions**: NLGI was the best-performing marker in predicting in-hospital mortality. As a predictor of poor prognosis, NLGI can guide clinicians in identifying the critical patient group.

Keywords

Acute aortic dissection; Cardiovascular; Glycemic index; Inflammation; Lymphocytes

1. Introduction

Acute aortic dissection (AAD) is a rare cardiovascular emergency condition associated with high mortality, which the mortality rate increasing by 1%–2% per hour from the onset of AAD symptoms [1, 2]. Given the high mortality rates, an earlier prediction of the risk of mortality in hospitalized patients with AAD may contribute to better disease prognosis [3, 4].

Recent studies have demonstrated that inflammatory mechanisms are involved in aortic media layer degeneration and vascular remodeling and contribute to tissue destruction in patients with aortic dissection [1, 4]. Neutrophils are the most abundant cells among leukocytes and play an important role in the inflammation process. Previous studies on patients with AAD have reported that an increased neutrophil count is associated with poor prognosis and mortality [3, 5, 6]. Furthermore, T lymphocyte activation in AAD induces a decrease in the number of lymphocytes in circulating blood [1]. Neutrophil lymphocyte ratio (NLR) is an inflammation-related biomarker, which is calculated by dividing the number of neutrophils measured in peripheral blood by the number of lymphocytes. Studies on NLR suggest that NLR is instrumental in predicting prognosis in patients with AAD [5–7].

Hyperglycemia is associated with impaired hemostasis, and may induce endothelial dysfunction, oxidative stress, and inflammatory response [8]. Impaired glucose metabolism is associated with progression of cardiovascular disease and poor prognosis [9–11]. Previous studies have reported that the leuko-glycemic index (LGI), which comprises leukocyte count and blood glucose, successfully predicted the prognosis in certain medical conditions, including myocardial infarction and stroke [12, 13]. The authors developed a neutrophil-lymphocyte glycemic index (NLGI) based on the success of NLR in predicting prognosis and mortality in AAD and the effects of hyperglycemia on adverse outcomes in AAD. This study aimed to investigate the performance of this new index in predicting mortality in hospitalized patients with AAD.

2. Materials and methods

2.1 Study design

Designed as retrospective observational research, this study was conducted on patients admitted to and diagnosed with AAD at the emergency department (ED) between 2017 and 2024. Prior to initiating the study, approval was obtained from the Clinical Research and Ethics Committee of Toros University (Date: 23 May 2024, No: 91). Ethical rules and principles as prescribed by the World Medical Association Declaration of Helsinki-Ethical Principles for Medical Research Involving Human Subjects were followed at all stages of the study. Written and verbally informed consent was obtained from the participants.

2.2 Selection of participants

Patients with a prediagnosis of AAD based on the hospital data among patients admitted to the ED between 2017 and 2024 were retrospectively evaluated. Patients aged 18 years and older who were surgically treated and had a confirmed diagnosis of AAD by thoracoabdominal computed tomography (CT) angiography were included in the study. Pregnant women, patients aged below 18 years, history of diabetes, history of chronic aorta dissection, who presented to the ED with cardiac arrest, history of immunodeficiency, and missing data were excluded from the study.

2.3 Data collection and measurement

Demographics of the patients, vital parameters evaluated at the time of admission to ED and laboratory results at the time of admission were recorded on the data collection form. The inflammation-related biomarkers NLR, platelet-lymphocyte ratio (PLR), lymphocyte-monocyte ratio (LMR), systemic immune-inflammatory index (SII), LGI, neutrophil glycemic index (NGI) and NLGI were calculated and recorded on the data collection form. Inflammation-related biomarkers are calculated using the following formulas:

NLR = Neutrophil count/Lymphocyte count
PLR = Platelet count/Lymphocyte count
LMR = Lymphocyte count/monocyte count
SII = Neutrophil count × Platelet count/Lymphocyte count
LGI = Leukocyte count × Glucose (mg/dL)/1000
NGI = Neutrophil count × Glucose (mg/dL)/1000
NLGI = NLR × Glucose (mg/dL)/1000

Patients were classified as Stanford Type A and B based on the results of the thoracoabdominal CT angiography. The primary outcome was considered in-hospital mortality. In-hospital mortality was assessed from the time of admission to the ED and was defined as death in the ED or during hospital stay. In-hospital mortality data of the patients were recorded in the data collection form.

2.4 Statistical analysis

The Statistical Package for the Social Sciences software (SPSS, version 26.0; IBM, Chicago, IL, USA) was used for the statistical analyses. The normal distribution hypothesis of study data was tested by histogram, scatter, and box-plot graphs; Kolmogorov-Smirnov test; Skewness and Kurtosis. Descriptive statistics were presented as mean ± standard deviation (SD), median (25th and 75th percentiles) and frequency and percentage (%). Based on whether the normal distribution hypothesis was met, independent samples *T*-test or Mann-Whitney U test was used for the analysis of continuous and categorical variables, and Chi-squared test was used for categorical variables.

Multivariable logistic regression analysis was used to sequentially assess whether the inflammation-related biomarkers were independent factors in predicting in-hospital mortality by unadjusted and adjusted models. The receiver operating characteristics (ROC) curve and area under the curve (AUC) analysis with 95% confidence interval (CI) were used for the analysis of the diagnostic performance of inflammation-related biomarkers in predicting in-hospital mortality. Furthermore, the Youden's index was used to calculate the cut-off points of the variables. Specificity, sensitivity, positive likelihood ratio (+LR), and negative likelihood ratio (−LR) of the variables were calculated using the most appropriate cut-off point. A p level of < 0.05 was considered statistically significant (Type 1 Error level).

3. Results

Age of the 73 patients included in the study ranged between 37–97 years, and the mean age was 67.53 ± 14.10 years. Of the patients, 72.6% (n = 53) were men and 27.4% (n = 20) were women. In-hospital mortality occurred in 33 patients. The mean age was higher in the nonsurvivors group ($p < 0.042$), and no statistical relation between mortality and sex ($p = 0.583$) was observed. No statistically significant difference between the survivors and nonsurvivors in regard to white blood cells (WBC) ($p = 0.089$), hemoglobin ($p = 0.978$) and platelet ($p = 0.327$) values were observed. The neutrophil ($p = 0.019$), NLR ($p = 0.008$), SII ($p = 0.030$), LGI ($p = 0.009$), NGI ($p = 0.001$), NLGI ($p = 0.001$), lymphocyte ($p = 0.026$) and LMR ($p = 0.008$) levels were lower in nonsurvivors compared to the survivors. Table 1 presents the relationship between demographics, clinical and laboratory results, and in-hospital mortality.

Multivariate logistic regression analysis was used to investigate the independent effects of inflammation-related biomarkers in predicting in-hospital mortality. It was observed that NLR (set OR (odds ratio): 1.18, 95% CI 1.023–1.361, $p = 0.023$), LMR (adjusted OR: 0.423, 95% CI 0.21–0.855, $p = 0.017$), LGI (adjusted OR: 4.143, 95% CI 1.466–11.709, $p = 0.007$), NGI (adjusted OR: 5.84, 95% CI 1.641–20.784, $p = 0.006$) and NLGI (adjusted OR: 3.726, 95% CI 1.458–9.525, $p = 0.006$) were the independent variables in predicting in-hospital mortality in patients with AAD. In the logistic regression analysis, D-dimer was not found to be a statistically significant predictor of in-hospital mortality ($p = 0.438$).

Univariate and multivariate logistic regression analyses were performed to evaluate the impact of inflammation-related biomarkers on the prediction of in-hospital mortality in patients with Type A and Type B AAD. Accordingly, NLR (adjusted OR: 1.158, 95% CI: 1.011–1.327, $p = 0.034$), LMR (adjusted OR: 0.577, 95% CI: 0.350–0.953, $p = 0.032$),

TABLE 1. Relationship with in-hospital mortality and patient's demographic characteristics, clinic, and laboratory findings.

	In-hospital mortality		
	Alive (n = 40)	Deceased (n = 33)	*p* values
Sex; n (%)			
Male	28 (30%)	25 (24.2%)	0.583
Female	12 (70%)	8 (75.8%)	
Dissection Classification; n (%)			
Stanford Type A	20 (50%)	26 (78.8%)	0.011
Stanford Type B	20 (50%)	7 (21.2%)	
Age (yr)	64.50 ± 14.77	71.21 ± 12.48	0.042
SBP (mmHg)	126.50 (120–130)	99 (90–107.5)	<0.001
DBP (mmHg)	80 (77–82)	65 (54.5–73.5)	<0.001
MAP (mmHg)	94.67 (91.42–100.17)	76.67 (66.83–84.67)	<0.001
Pulse rate (beats/min)	81.13 ± 8.83	116.82 ± 18.55	<0.001
WBC count (cells/mm^3)	11.32 ± 3.70	12.85 ± 3.88	0.089
Hemoglobin (mg/dL)	12.75 (11.6–14.05)	13.10 (10.30–14.70)	0.978
Platelet count (cells/mm^3)	216 (173–275)	202 (152.50–249.5)	0.327
Neutrophil count (cells/mm^3)	8.63 ± 3.40	10.63 ± 3.63	0.019
Lymphocyte count (cells/μL)	1.47 (1.02–2.09)	1.03 (0.84–1.61)	0.026
Monocyte count	0.53 (0.39–0.80)	0.63 (0.40–0.96)	0.176
NLR	5.43 (2.96–9.68)	8.07 (5.84–16.27)	0.008
PLR	158.2 (109.73–208.38)	165.77 (114.18–258.76)	0.261
LMR	2.71 (1.82–4.05)	1.63 (1.20–2.94)	0.008
SII	1206.19 (663.61–2222.78)	1681.77 (984.32–3291.90)	0.030
Glucose (mg/dL)	118 (96.75–136.25)	134 (117–193.50)	0.008
LGI	1.40 (0.95–1.86)	1.81 (1.41–2.87)	0.009
NGI	1.08 ± 0.53	1.66 ± 0.82	0.001
NLGI	0.69 (0.38–1.20)	1.20 (0.84–2.48)	0.001
AST (IU/L)	23 (17–29.75)	28 (18–45.50)	0.136
ALT (IU/L)	18.5 (13–25)	22 (15–30.5)	0.260
LDH (IU/L)	273.75 ± 54.44	270.42 ± 72.57	0.824
CRP	2.75 (2–3.56)	4 (2.75–6.55)	0.088
Lactate (mmol/L)	1.85 (1.05–2)	3 (2.05–3.8)	<0.001
Troponin	83 (12.75–141)	69 (21–199)	0.390
D-dimer	2.75 (2–3.56)	4 (2.75–6.55)	0.002

Values are presented as mean ± SD, median (25th and 75th quartile) or n (%).
ALT: alanine aminotransferase; AST: aspartate aminotransferase; CRP: C-reactive protein; DBP: Diastolic blood pressure; LDH: lactate dehydrogenase; LGI: leuko-glycemic index; LMR: lymphocyte-monocyte ratio; MAP: mean arterial pressure; NGI: neutrophil glycemic index; NLGI: neutrophil-lymphocyte glycemic index; NLR: neutrophil lymphocyte ratio; PLR: platelet to lymphocyte ratio; SBP: systolic blood pressure; SII: systemic immune-inflammatory index; WBC: white blood cells count.

LGI (adjusted OR: 2.499, 95% CI: 1.045–5.979, p = 0.040), NGI (adjusted OR: 3.161, 95% CI: 1.144–8.732, p = 0.026) and NLGI (adjusted OR: 3.223, 95% CI: 1.154–9.002, p = 0.026) were identified as independent predictors of in-hospital mortality in Type A AAD, whereas only NLGI (adjusted OR: 3.005, 95% CI: 1.003–9.006, p = 0.049) was associated with in-hospital mortality in Type B AAD. The statistical figures for multivariate logistic regression analysis are given in Tables 2 and 3.

ROC analysis was performed to investigate the performance of the variables in predicting in-hospital mortality. NLGI (AUC: 0.725, 95% CI: 0.609–0.841, p = 0.001) had the best performance, followed by NGI (AUC: 0.699, 95% CI: 0.579–0.820, p = 0.004) and NLR (AUC: 0.683, 95% CI: 0.561–0.804, p = 0.008). In the ROC analysis performed for patients with Type A AAD, NLGI demonstrated the best performance in predicting in-hospital mortality (AUC: 0.721, 95% CI: 0.569–0.843, p = 0.004), while it also showed good discriminatory power for predicting in-hospital mortality in patients with Type B AAD, with an AUC of 0.750. Tables 4 and 5 show the performance characteristics of the variables in predicting in-hospital mortality and Figs. 1,2,3 include the ROC curve.

4. Discussion

To the best of the authors' knowledge, this study is the first to examine the impact of the Neutrophil-Lymphocyte Glycemic Index (NLGI) in predicting in-hospital mortality in patients with Acute Aortic Dissection (AAD). The study demonstrates that NLGI is an effective index for predicting in-hospital mortality in AAD patients. It was observed that patients with elevated levels of NLR, LMR, SII, LGI, NGI and NLGI at the time of admission to the emergency department had a higher in-hospital mortality rate. Multivariate logistic regression analysis was performed to assess whether these inflammation-related biomarkers serve as independent predictors of in-hospital mortality. The analysis revealed that NLR, LMR, LGI, NGI and NLGI are independent predictors of in-hospital mortality in patients with AAD, whereas D-dimer was not an independent predictor. Furthermore, subgroup analyses for Type A and Type B AAD demonstrated that NLGI is an independent predictor for both types.

Previous studies suggested that the inflammatory response played an important role in the occurrence and progression of aortic dissection [14]. Inflammation in the aortic wall has been considered to play a role in the occurrence and progression of aortic dissection, despite the fact that the effect of inflammatory mechanisms in the pathophysiology of aortic dissection has not been definitively elucidated. It was suggested that inflammatory cells induced apoptosis in smooth muscle cells in the aortic wall, leading to development of aortic dissection [6, 15]. Previous studies have reported the occurrence of inflammatory cells, including macrophages, neutrophils and lymphocytes in the media and adventitia layers of the dissected aorta. Moreover, neutrophils release matrix metalloproteases, interleukin-6, free oxygen radicals, serine free oxygen radicals and histone proteases, which exacerbate vascular inflammation, widen the dissection and accelerate rupture [16, 17]. Lymphocytes contribute to the progress of aortic dissection by inducing vascular smooth muscle apoptosis and matrix metalloproteinase (MMP) secretion. Previous studies reported a decrease in total lymphocyte count and an increase in neutrophil count in peripheral blood samples of patients with AAD [15, 17]. A study by Onuk *et al.* [18] on patients with AAD found that elevated neutrophil and decreased lymphocyte counts were associated with increased mortality rate. In another study on patients with Type A AD, the in-hospital mortality was associated with increased neutrophil count and lymphopenia [5]. Herein, elevated neutrophil count and lymphopenia were

TABLE 2. ORs of the prognostic factors for predicting in-hospital mortality in patients with AAD.

Parameters	Model 1		Model 2		Model 3	
	OR (95% CI)	p value	OR (95% CI)	p value	OR (95% CI)	p value
Neutrophil	1.179 (1.024–1.359)	0.022	1.198 (1.025–1.400)	0.024	1.227 (0.989–1.521)	0.063
Lymphocyte	0.456 (0.216–0.960)	0.039	0.459 (0.199–1.054)	0.066	0.340 (0.109–1.061)	0.063
NLR	1.153 (1.044–1.274)	0.005	1.154 (1.034–1.288)	0.011	1.180 (1.023–1.361)	0.023
LMR	0.614 (0.425–0.886)	0.009	0.586 (0.388–0.887)	0.012	0.423 (0.210–0.855)	0.017
SII	1.000 (1.000–1.001)	0.029	1.000 (1.000–1.001)	0.174	1.001 (1.000–1.001)	0.064
LGI	2.813 (1.387–5.705)	0.004	2.860 (1.299–6.296)	0.009	4.143 (1.466–11.709)	0.007
NGI	3.644 (1.601–8.294)	0.002	3.709 (1.497–9.189)	0.005	5.840 (1.641–20.784)	0.006
NLGI	2.844 (1.444–5.602)	0.003	3.057 (1.437–6.506)	0.004	3.726 (1.458–9.525)	0.006
D-dimer	1.018 (0.974–1.063)	0.438	*		*	

**Not applicable.*
Model 1: unadjusted model.
Model 2: adjusted for age, sex, and dissection type.
Model 3: each marker was adjusted for age, sex, dissection type, lactate, and D-dimer.
AAD: Acute aortic dissection; CI: confidence interval; LGI: leuko-glycemic index; LMR: lymphocyte-monocyte ratio; NGI: neutrophil glycemic index; NLGI: neutrophil-lymphocyte glycemic index; NLR: neutrophil lymphocyte ratio; OR: Odds ratio; SII: systemic immune-inflammatory index.

TABLE 3. ORs of the prognostic factors for predicting in-hospital mortality in subgroups.

Parameters	Model 1		Model 2	
	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Type A AAD				
Lymphocyte	0.407 (0.155–1.065)	0.067	*	
NLR	1.158 (1.011–1.327)	0.034	1.161 (1.006–1.341)	0.042
LMR	0.577 (0.350–0.953)	0.032	0.524 (0.302–0.909)	0.021
SII	1.001 (1.000–1.001)	0.055	*	
LGI	2.499 (1.045–5.979)	0.040	2.516 (1.011–6.262)	0.047
NGI	3.161 (1.144–8.732)	0.026	3.227 (1.114–9.35)	0.031
NLGI	3.223 (1.154–9.002)	0.026	3.124 (1.082–9.016)	0.035
Type B AAD				
Lymphocyte	0.446 (0.102–1.944)	0.282	*	
NLR	1.146 (0.978–1.344)	0.092	*	
LMR	0.641 (0.340–1.207)	0.169	*	
SII	1.000 (0.999–1.001)	0.433	*	
LGI	3.2951 (0.847–12.819)	0.085	*	
NGI	4.271 (0.901–20.236)	0.067	*	
NLGI	2.833 (1.046–7.669)	0.040	3.005 (1.003–9.006)	0.049

**Not applicable.*
Model 1: unadjusted model.
Model 2: adjusted for age, sex, and dissection type.
AAD: Acute aortic dissection; CI: confidence interval; LGI: leuko-glycemic index; LMR: lymphocyte-monocyte ratio; NGI: neutrophil glycemic index; NLGI: neutrophil-lymphocyte glycemic index; NLR: neutrophil lymphocyte ratio; OR: Odds ratio; SII: systemic immune-inflammatory index.

TABLE 4. Cutoff points and performance characteristics of inflammatory markers in predicting in-hospital mortality.

Parameters	AUC	95% CI	Cutoff	Sensitivity	Specificity	+LR	−LR	*p* values
Neutrophil	0.644	(0.516–0.771)	10.840	0.515	0.750	2.06	0.65	0.036
Lymphocyte	0.653	(0.526–0.779)	1.410	0.697	0.550	1.55	0.55	0.026
NLR	0.683	(0.561–0.804)	6.296	0.758	0.575	1.78	0.42	0.008
LMR	0.681	(0.557–0.805)	1.647	0.515	0.850	3.43	0.57	0.008
SII	0.648	(0.521–0.775)	2868.116	0.333	0.925	4.44	0.72	0.030
LGI	0.678	(0.554–0.802)	2.007	0.424	0.900	4.24	0.64	0.009
NGI	0.699	(0.579–0.820)	1.756	0.424	0.900	4.24	0.64	0.004
NLGI	0.725	(0.609–0.841)	0.800	0.788	0.600	1.97	0.35	0.001

AUC: Areas under the curve; CI: Confidence interval; LMR: lymphocyte-monocyte ratio; +LR: positive likelihood ratio; −LR: negative likelihood ratio; NGI: Neutrophil glycemic index; NLGI: Neutrophil-lymphocyte glycemic index; NLR: Neutrophil lymphocyte ratio; LGI: leuko-glycemic index; SII: systemic immune-inflammatory index.

TABLE 5. Cutoff points and performance characteristics of inflammatory markers in predicting in-hospital mortality in subgroups.

Parameters	AUC	95% CI	Cutoff	Sensitivity	Specificity	+LR	−LR	*p* values
Type A AAD								
NLR	0.685	0.526–0.843	>5.880	0.769	0.650	2.20	0.36	0.024
LMR	0.692	0.539–0.820	≤1.750	0.539	0.900	5.38	0.51	0.017
LGI	0.681	0.527–0.810	>1.354	0.846	0.550	1.88	0.28	0.025
NGI	0.683	0.529–0.812	>1.055	0.808	0.550	1.79	0.35	0.022
NLGI	0.721	0.569–0.843	>0.720	0.808	0.600	2.02	0.32	0.004
Type B AAD								
NLGI	0.750	0.547–0.895	>1.677	0.571	0.950	11.43	0.45	0.043

AUC: Areas under the curve; CI: Confidence interval; LMR: lymphocyte-monocyte ratio; +LR: positive likelihood ratio; −LR: negative likelihood ratio; AAD: Acute aortic dissection; NGI: Neutrophil glycemic index; NLGI: Neutrophil-lymphocyte glycemic index; NLR: Neutrophil lymphocyte ratio; LGI: leuko-glycemic index.

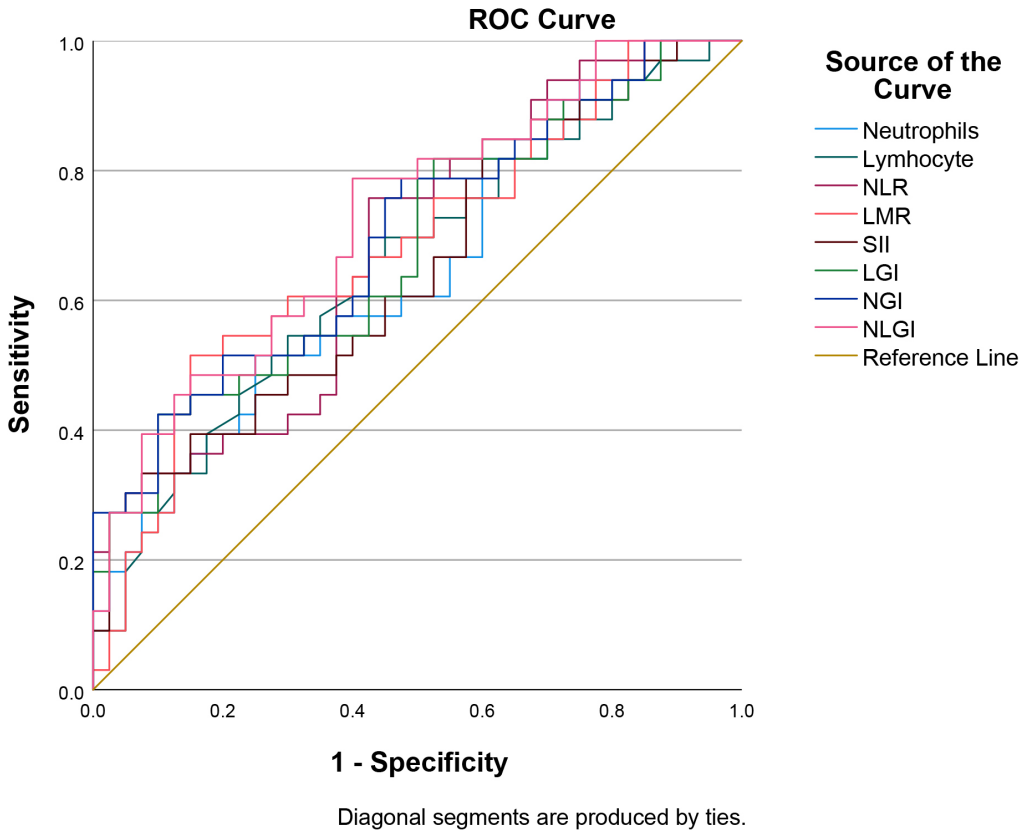

FIGURE 1. ROC curve of inflammation-related biomarkers in predicting in-hospital mortality in patients with AAD. AAD: Acute aortic dissection; NGI: Neutrophil glycemic index; NLR: Neutrophil lymphocyte ratio; LGI: leuko-glycemic index; LMR: Lymphocyte-monocyte ratio; ROC: Receiver operating characteristic; SII: systemic immune-inflammatory index; NEU: Neutrophil; LYM: Lymphocyte; NLGI: Neutrophil-lymphocyte glycemic index.

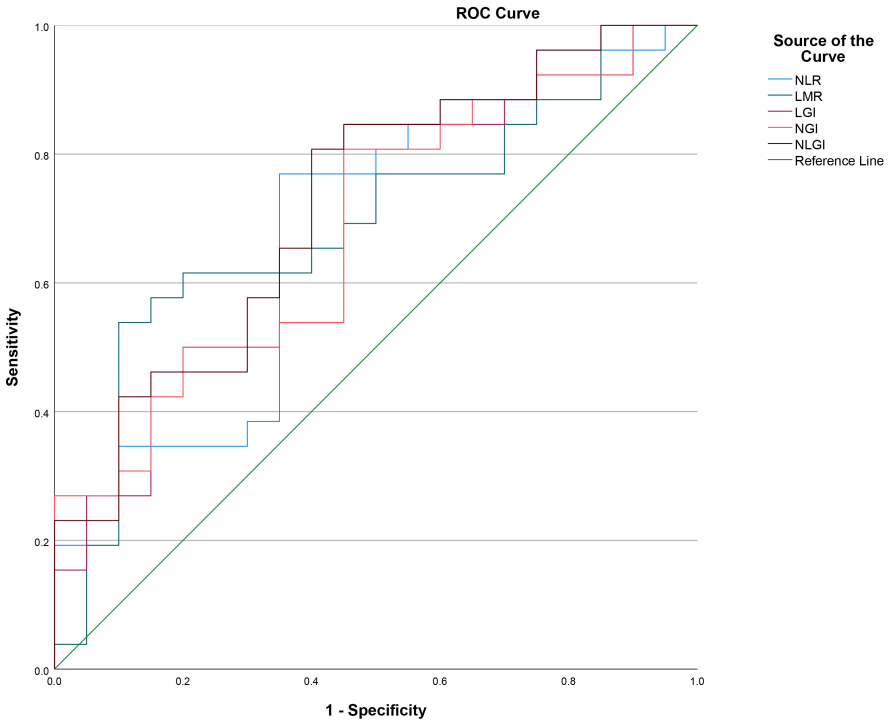

FIGURE 2. ROC curve of inflammation-related biomarkers in predicting in-hospital mortality in patients with Type A AAD. AAD: Acute aortic dissection; NGI: Neutrophil glycemic index; NLGI: Neutrophil-lymphocyte glycemic index; NLR: Neutrophil lymphocyte ratio; LGI: leuko-glycemic index; LMR: Lymphocyte-monocyte ratio; ROC: Receiver operating characteristic.

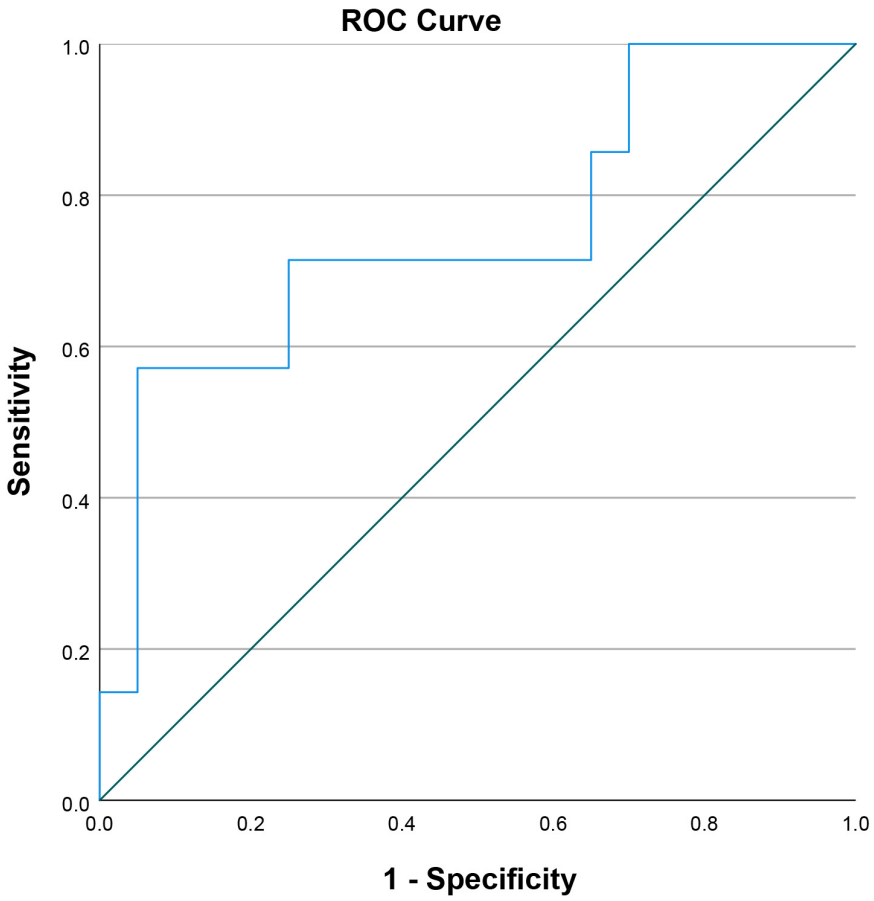

FIGURE 3. ROC curve of inflammation-related biomarkers in predicting in-hospital mortality in patients with Type B AAD. NLGI is represented by the blue line; the green line indicates the reference line. AAD: Acute aortic dissection; NLGI: Neutrophil-lymphocyte glycemic index; ROC: Receiving operating characteristics.

associated with increased in-hospital mortality consistent with the reports by previous studies.

NLR was shown to be more successful in predicting aortic dissection compared to neutrophil and lymphocyte counts. Numerous studies in relevant literature agree that NLR is a successful marker in predicting the prognosis of patients with aortic dissection [5, 15, 17, 19]. A study by Zhang *et al.* [14] reported that NLR was successful in predicting in-hospital mortality with an AUC of 0.695. Furthermore, the same study suggested that NLR was an independent prognostic factor in multivariate logistic regression analysis in predicting in-hospital mortality. Another study by Onuk *et al.* [18] reported that higher NLR was a predictor of in-hospital mortality in patients with AAD. Upon ROC analysis, it was observed that the AUC of NLR for predicting in-hospital mortality was 0.672. Additionally, NLR was an independent predictor of in-hospital mortality upon multivariate regression analysis [18]. In the present study, NLR showed moderate performance in predicting in-hospital mortality with an AUC of 0.683. The present study demonstrated the independent effect of NLR in predicting in-hospital mortality based on multivariate logistic regression analysis. These results are consistent with literature.

Quiroga *et al.* [13] suggested LGI, a combined index of leukocytes and blood glucose, as a prognostic marker in patients with myocardial infarction. Previous studies suggested that LGI was an indicator of systemic inflammation and served as a successful index in predicting mortality and prognosis in different clinical conditions [13, 20].

Impaired glucose metabolism and hyperglycemia have been associated with numerous adverse effects in an individual. These include oxidative stress, endothelial dysfunction, and activation of the inflammatory response [8, 9]. Stress hyperglycemia is defined as newly detected elevated blood glucose that typically resolves following the remission of the acute illness. Elevated blood glucose levels lead to an increased concentration of circulating inflammatory cytokines and a decreased glucose uptake capacity in peripheral tissues. The augmented production of counter-regulatory hormones—glucagon, catecholamines, cortisol and growth hormone—enhances insulin resistance, thereby impairing insulin action. The resulting increase in insulin resistance promotes lipolysis, contributing to a catabolic state that leads to lipotoxicity. Consequently, glucosuria, ketonuria, osmotic diuresis, dehydration, hemodynamic instability, and impaired tissue perfusion ensue. Ultimately, these processes result in a negative nitrogen balance, which diminishes wound healing and reduces resistance to infections [10]. Lin *et al.* [11] discovered that hyperglycemia at admission in patients with aortic dissection was associated with an increase in ventilation time. In another study, Mutailifu *et al.* [21] reported that hyperglycemia at admission was associated with in-hospital mortality in patients with AAD. The authors of the present study developed NLGI

based on the effects of hyperglycemia and the success of NLR in predicting prognosis in patients with AAD. In the present study, NLGI performed better compared to both NLR and LGI in predicting in-hospital mortality. Furthermore, it was shown NLGI was an independent factor in predicting in-hospital mortality upon multivariate logistic regression analysis.

This study has several limitations that warrant consideration. Primarily, its retrospective and single-center design may introduce selection bias, potentially affecting the representativeness of the study population. Furthermore, the relatively small sample size restricts the generalizability of the findings to broader patient cohorts, which may in turn impact the validity and reliability of the conclusions drawn. Future investigations should aim to replicate these results using larger populations and adopt multicenter, prospective study designs to enhance external validity. Additionally, the exclusion of patients with incomplete data may reduce the applicability of the results to certain subgroups. Continuous monitoring of NLGI at multiple time points should also be considered to assess its potential prognostic value more comprehensively. To overcome these limitations, future research should focus on improving data acquisition methods and utilizing more robust and comprehensive clinical datasets.

5. Conclusions

The findings of this study indicate that the Neutrophil-Lymphocyte Glucose Index (NLGI), along with other inflammation-related biomarkers, holds significant prognostic value in predicting in-hospital mortality among patients diagnosed with acute aortic dissection (AAD). NLGI has emerged as a strong and independent predictor within this patient population. The clinical implementation of these biomarkers facilitates the early identification of high-risk individuals and contributes to the development of appropriate therapeutic strategies. The practicality and cost-effectiveness of calculating NLGI enhance its applicability, particularly in emergency department settings, thereby potentially improving healthcare efficiency and patient outcomes. Accordingly, future studies should aim to further evaluate the clinical utility of NLGI and validate these findings across broader and more heterogeneous patient populations.

AVAILABILITY OF DATA AND MATERIALS

The data that support the findings of this study are available on request from the corresponding author.

AUTHOR CONTRIBUTIONS

MSS, MAE—Conceptualization, Methodology, Data Curation, Visualization. MSS, ME—Formal analysis. MAE, ME—Investigation. MSS, MAE, ME—Writing–original draft preparation, writing–review and editing. MAE—Resources, Supervision.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Clinical Research Ethics Committee of the Toros University. Date: 23 May 2024, number: 91. Written and verbally informed consent was obtained from the participants.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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