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Prognostic value of the Naples prognostic score for predicting major adverse cardiac events and long-term outcomes in patients with NSTEMI

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ABSTRACT

Objectives: The Naples prognostic score (NPS) is determined using four parameters: Serum albumin, total cholesterol, the neutrophil-to-lymphocyte ratio, and the lymphocyte-to-monocyte ratio. Since both inflammation and nutritional status have a critical impact on the onset and advancement of cardiovascular diseases, this score has been suggested as a beneficial prognostic tool. In this research, we sought to assess the predictive value of NPS, measured at the time of hospital admission, for major adverse cardiac events (MACE) and long-term outcomes in patients with non-ST-segment elevation myocardial infarction (NSTEMI).

Patients and methods: A cohort of 125 individuals with NSTEMI, identified between January 1 and June 1, 2019, was retrospectively evaluated. According to their NPS values, the cohort was stratified into two groups: Low NPS (0-2 points; n=73) and high NPS (3,4 points; n=52). Over an average follow-up period of 60 months, the manifestation of MACE and overall mortality was systematically documented.

Results: MACE was observed in 31 patients, with a markedly greater frequency in the high-NPS group (n=22; p<0.001). Long-term mortality occurred in 18 individuals, of whom 15 belonged to the high-NPS category (p<0.001). ROC curve analysis determined an optimal NPS threshold of 2.5 for predicting both MACE and overall mortality. Survival analysis using the Kaplan-Meier method revealed a considerable decrease in survival among patients with elevated NPS (p<0.001).

Conclusion: The NPS, which incorporates inflammatory and nutritional components, functions as a prognostic determinant of MACE and long-term mortality in NSTEMI cases.

Keywords: Naples prognostic score, non-ST-segment elevation myocardial infarction, major adverse cardiac events, risk stratification, mortality.

Acute myocardial infarction (AMI) continues to be a leading cause of death across the globe and represents a substantial public health burden. From a clinical perspective, AMI is categorized into two principal subtypes based on electrocardiographic results: ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI).[1] While both entities share similar pathophysiological mechanisms, they differ in clinical presentation, management strategies, and long-term prognosis. From a physiological standpoint, AMI occurs due to an abrupt disruption of coronary blood flow, which may present as complete occlusion—as typically seen in STEMI and occasionally in NSTEMI—or as partial obstruction, more frequently observed in NSTEMI.[2] This condition typically results from erosion or disruption of atherosclerotic plaques within the coronary arteries.[3] In recent years, the prevalence of NSTEMI has risen, primarily

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as a consequence of the extensive implementation of high-sensitivity troponin tests in patients with acute chest pain.[4] Risk stratification in individuals with NSTEMI may facilitate the detection of those at higher risk for adverse cardiac occurrences. Early recognition of high-risk patients enables the application of tailored interventions that may contribute to improved clinical outcomes. The Naples prognostic score (NPS), originally designed to estimate postoperative mortality within the cohort of colorectal cancer cases undergoing surgery,[5] integrates serum albumin concentration (Alb), total cholesterol (TC), neutrophil-to-lymphocyte ratio (NLR), and lymphocyte-to-monocyte ratio (LMR), thereby enabling a combined evaluation of malnutrition and inflammation. Given the critical role of inflammation and malnutrition in the pathophysiology of certain cardiovascular diseases, the prognostic potential of the NPS in these patient populations warrants further investigation.[6] Previous studies have assessed the predictive significance of the NPS in STEMI patients treated with primary percutaneous coronary intervention (PCI). Several studies have demonstrated that, in this patient population, the NPS is a an indicator of mortality risk during hospitalization and in long-term follow-up.[7,8] It is well established that, compared to STEMI patients, those with NSTEMI are generally older, have a greater burden of comorbidities, and exhibit a higher propensity for inflammation.[9] Studies investigating the NPS in this patient population remain limited in the literature.[10]

Aim: The objective of this research was to explore the prognostic utility of the NPS, obtained at admission, in forecasting major adverse cardiac events (MACE) and extended overall mortality in individuals with NSTEMI.

PATIENTS AND METHODS

Study Design and Population

In this retrospective, single-center observational study, 125 patients diagnosed with NSTEMI and treated with coronary angiography and/or PCI between January 1 and June 1, 2019, were included. Retrospective follow-up was conducted, with a mean duration of 60 months. Clinical data—including baseline characteristics, laboratory results, imaging parameters, and follow-up outcomes— were obtained from digital health records and patient charts of the hospital.

Exclusion Criteria

Patients with neoplastic diseases, those receiving chemotherapy, individuals with evidence of any acute or chronic inflammatory condition, active infection, recent (within the past 3 months) glucocorticoid therapy, or use of immunosuppressive agents were removed from the study cohort. Additional exclusion criteria included severe hepatic dysfunction (e.g., cirrhosis, chronic hepatitis), marked kidney impairment (described as glomerular filtration rate <30 mL/min/1.73 m² or chronic hemodialysis), severe anemia (hemoglobin <8 g/dL), cardiogenic shock requiring inotropic support or resulting in in-hospital mortality, severe valvular heart disease, and incomplete or missing data. A total of 62 patients meeting one or more of these criteria were excluded from the final analysis (Figure 1).

NPS Calculation and Risk Grouping

The NPS was determined using laboratory parameters measured following hospital admission. Scoring was performed according to previously established components and cut-off values reported in earlier studies:[5,7,8]

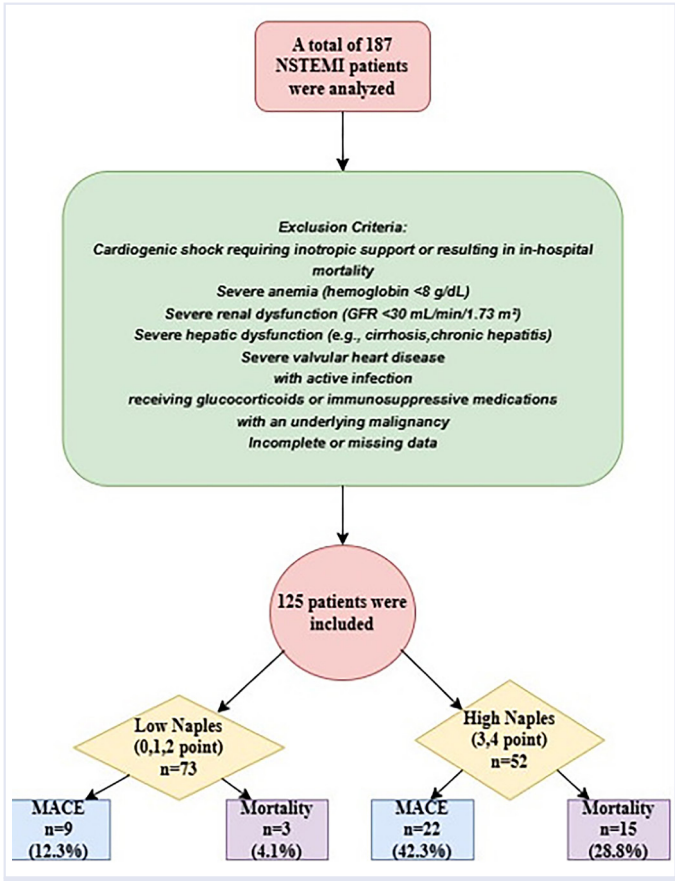

Figure 1. Study design flowchart.
MACE: Major adverse cardiac events; GFR: Glomerular filtration rate; NSTEMI: Non-ST-segment elevation myocardial infarction.

1) TC ≤180 mg/dL

2) Alb <4 g/dL

3) NLR >2.96

4) LMR ≤4.44.

One point was assigned to patients for each parameter meeting the specified thresholds (score range: 0-4). According to their total NPS, patients were classified into two groups: Low NPS (0, 1, or 2 points) and high NPS (3 or 4 points).

Study Endpoints

During the follow-up period, patients were evaluated based on the occurrence of MACE and overall long-term mortality. MACE was described as the combined of cardiovascular death, non-fatal myocardial infarction (MI), and non-fatal cerebrovascular events. Mortality information was retrieved from hospital archives and the national social security registry. Patients who experienced death during follow-up were classified according to their respective risk groups.

Ethical Consideration

The investigation was authorized by the Institutional Ethics Committee of Mardin Artuklu University (approval no: 2025/4-37, April 22, 2025), and the research was executed out in line with the principles of the Declaration of Helsinki (2024).

Statistical Analysis

Statistical analyses were executed using SPSS software, version 26.0 (Chicago, IL, USA). Descriptive statistics were applied to summarize baseline demographic and clinical variables. Normality of data distribution was evaluated using histograms and analytical tests. For group comparisons, continuous variables were examined with the autonomous samples t-test when normally distributed, and with the Mann-Whitney U test when distributional assumptions were not met. Categorical variables were examined using either the chi-square test or Fisher's exact test, as suitable. Data are demonstrated as mean ± standard deviation for normally distributed continuous variables, median (interquartile range) for non-normally distributed data, and percentages for categorical variables. The prognostic performance of the NPS in relation to MACE and long-term mortality was investigated using receiver operating characteristic (ROC) curve analysis. Cox proportional hazards regression, both univariate and multivariate, was applied to determine independent determinants of long-term outcomes. Survival probabilities were illustrated by Kaplan-Meier curves, and between-group differences were evaluated using the log-rank test. Statistical importance was set at a two-sided p-value <0.05.

RESULTS

After exclusions, the final study cohort consisted of 125 patients (Figure 1). Of these, 79 patients (63.2%) were male. Based on their NPS scores, Patients were grouped into two categories: Low NPS (0, 1, or 2 points; n=73) and High NPS (3 or 4 points; n=52). Demographic, laboratory, imaging, and procedural characteristics of the individuals are demonstrated in Table 1. There were no notable differences detected between the two groups regarding demographic, imaging, and procedural parameters. Among the laboratory variables, lymphocyte value (p<0.001) and Alb (p=0.005) were lower in the high NPS group, whereas neutrophil value (p<0.001) was higher (Table 1).

During the follow-up period, MACE was observed in 31 patients, 22 of whom belonged to the high NPS group (p<0.001). Additionally, overall death was observed in 18 patients, 15 of whom were in the high NPS group (p<0.001) (Table 1).

Cox regression analyses, both univariate and multivariate, were conducted to determine autonomous predictors of long-term mortality. The univariate evaluation demonstrated that ejection fraction (EF) (p=0.011) and Naples score (p=0.002) were markedly related to mortality. In the subsequent multivariate analysis, conducted to determine the most robust predictor among them, the Naples score remained independently and importantly related with long-term mortality (hazard ratio: 6.762; 95% confidence interval [CI]: 1.937-23.602; p=0.003) (Table 2).

The diagnostic accuracy of the Naples score in forecasting overall death and MACE was evaluated using ROC curve analyses. For all-cause mortality, the area under the curve (AUC) was 0.763 (95% CI: 0.639-0.888; p<0.001). A cut-off value of 2.5 for the Naples score demonstrated 83% sensitivity and 66% specificity in predicting long-term mortality (Figure 2A). For MACE prediction, the AUC was 0.730 (95% CI: 0.622-0.837; p<0.001), and a Naples score cut-off value of 2.5 yielded 71% sensitivity and 69% specificity (Figure 2B).

According to Kaplan-Meier estimates, individuals with higher Naples scores showed a markedly greater risk of long-term mortality throughout the 60-month follow-up period (Log-rank =14.382, p<0.001) (Figure 3A). Additionally, Kaplan-Meier analysis revealed a markedly higher incidence of MACE among patients with high Naples scores during the same follow-up period (Log-rank =14.216, p<0.001) (Figure 3B).

Mean Naples scores in patients with and without all-cause mortality were compared using a bar chart. The mean Naples score was 3.16 in the mortality group and 2.13 in the non-mortality group, with a statistically meaningful discrepancy between the two (p<0.001) (Figure 4).

DISCUSSION

This research was designed to investigate the prognostic utility of the NPS among NSTEMI patients, and the main findings are as follows:

1) Higher NPS values are positively linked to an elevated risk of MACE.

2) Higher NPS values are connected with an elevated risk of long-term overall death.

3) Following its use in cancer patients and certain cardiovascular disease populations, the NPS also offers a novel perspective in the risk classification of patients with NSTEMI.

The significance of the NPS lies in its components: It incorporates NLR and LMR as markers of inflammation, along with serum albumin and TC serving as markers of malnutrition, thereby enabling simultaneous assessment of both inflammatory status and nutritional condition.[5] The coexistence of inflammation and malnutrition may exacerbate adverse clinical outcomes and exert a synergistic effect on prognosis. Inflammation is a crucial factor in the initiation and progression of coronary artery disease (CAD). Contributing to the formation, progression, and rupture of atherosclerotic plaques, as well as promoting the activation of procoagulant pathways.[11] Neutrophils contribute significantly to atherosclerotic plaque instability. They are key regulators of the inflammatory response following MI, and elevated neutrophil counts have been connected with an elevated risk of cardiovascular mortality.[12] In contrast to neutrophils, lymphocytes play a regulatory role in inflammation and are therefore considered to have an anti-atherosclerotic effect. While anemia and thrombosis tend to exacerbate inflammation, lymphocytes may help attenuate its severity.[13] Thus, NLR has been identified as a biomarker of systemic inflammation and considered a possible predictor of both risk and prognosis in CAD.[14,15] NLR has been studied in many heart diseases before, and important findings have been discovered.[16,17]

As essential immune cells participating in the development of inflammation and atherosclerosis, lymphocytes and monocytes additionally affect the prognosis of MI patients. Low lymphocyte values and elevated monocyte values have been connected with adverse cardiovascular outcomes in patients with CAD.[18,19] Monocytes exhibit procoagulant properties in the setting of inflammation and MI, primarily through the formation of thrombotic monocyte-platelet aggregates.[20] Considering all these factors, it has been suggested that a composite inflammatory marker reflecting the balance between lymphocytes and monocytes may yield further value in cardiovascular risk analysis. Accordingly, the LMR has been regarded as a indicator of systemic inflammation. Numerous studies have revealed that the LMR is connected with the severity of cardiovascular disease and mortality.[21,22]

Table 1. Basic demographic and laboratory characteristics of the patients

Variables	Low Naples (n=73)	High Naples (n=52)	p-value
Gender (female), n (%)	29 (39.7)	17 (32.7)	0.422
Age, (years)	60.3±11.1	62.2±11.0	0.333
TIMI -1, n (%) -2, n (%) -3, n (%)	 20 (27.4) 38 (52.1) 15 (20.5)	 9 (17.3) 27 (51.9) 16 (30.8)	0.271
GRACE	1.97±0.72	2.03±0.62	0.598
Culprit artery, n (%) -LAD -Cx -RCA	 33 (45.2) 24 (32.9) 16 (21.9)	 23 (44.2) 15 (28.8) 14 (26.9)	0.786
HT, n (%)	50 (68.5)	38 (73.1)	0.580
DM, n (%)	19 (26.0)	10 (19.2)	0.375
Dyslipidemia, n (%)	20 (27.4)	18 (34.6)	0.387
CKD, n (%)	5 (6.8)	4 (7.7)	0.857
Smoking, n (%)	23 (31.5)	22 (42.3)	0.215
Previous MI, n (%)	11 (15.1)	7 (13.5)	0.801
Previous PCI, n (%)	13 (17.8)	5 (9.6)	0.198
Percutaneous coronary intervention, n (%)	50 (68.5)	42 (80.8)	0.125
LVEF, (%)	60 (55-60)	60 (53-60)	0.207
WBC, ($x10^3$/uL)	9.2 (8.1-10.8)	10.5 (8.3-12.5)	0.069
Lymphocyte, ($x10^3$/uL)	2.67±1.04	1.89±0.65	**<0.001**
Neutrophil, ($x10^3$/uL)	5.94±2.51	7.99±3.35	**<0.001**
Monocyte, ($x10^3$/uL)	0.68±0.29	0.72±0.23	0.387
Hemoglobin, (gr/L)	13.7±1.8	13.9±1.8	0.406
Platelet (10^3/uL)	259.3±57.0	260.2±87.0	0.948
Glucose, (mg/dL)	112 (101-210)	112 (99-136)	0.679
BUN, (mg/dL)	37.2±13.6	38.0±8.7	0.739
Creatinine, (mg/dL)	0.99±0.26	0.97±0.19	0.703
Albumin, (gr/dL)	3.76±0.48	3.54±0.40	**0.005**
CRP, (mg/dL)	6.4 (4.0-12.0)	8.2 (5.2-14.3)	0.137
Total cholesterol, (mg/dL)	175.8±34.4	189.2±41.1	0.065
HDL-C, (mg/dL)	36.1±7.1	36.4±6.5	0.844
LDL-C, (mg/dL)	108.3±27.2	117.4±36.6	0.143
Triglyceride, (mg/dL)	156.5±64.7	177.5±58.0	0.059
MACE, n (%)	9 (12.3)	22 (42.3)	**<0.001**
Mortality, n (%)	3 (4.1)	15 (28.8)	**<0.001**

TIMI: Thrombolysis in myocardial infarction; GRACE: The global registry of acute coronary events; LAD: Left anterior descending; Cx: Circumflex; RCA: Right coronary artery; HT: Hypertension; DM: Diabetes mellitus; CKD: Chronic kidney disease; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; LVEF: Left ventricular ejection fraction; WBC: White blood cells (10^6/L), BUN: Blood urea nitrogen; CRP: C-reactive protein; HDL-C: High-density lipoprotein cholesterol (mg/dL); LDL-C: Low-density lipoprotein cholesterol (mg/dL); MACE: Major adverse cardiac events. Data are presented as mean ± standard deviation or n (%). Statistical significance is considered at a p-value of less than 0.05.

In NSTEMI patients, nutritional status plays a crucial role in clinical outcomes; malnutrition is commonly observed and linked to higher rates of morbidity and mortality.[23] Malnutrition reflects not only inflammation but also frailty, a condition characterized by increased vulnerability and dysfunction across multiple physiological systems. Serum albumin levels can be used as a marker for assessing nutritional status. Hypoalbuminemia is not only a marker of malnutrition but also an indicator of systemic inflammation, resulting from the pro-inflammatory effects of different cytokines that suppress Albs.[24] Serum albumin levels reflect protein and calorie intake and indicate the degree of inflammation and disease severity during acute illness.[25] In addition, albumin plays critical roles in maintaining oncotic pressure, transporting various molecules, scavenging free radicals, and inhibiting platelet aggregation.[26] Hypoalbuminemia causes impaired endothelial function and increased blood viscosity.[27]

Table 2. Independent predictors of 5-year mortality in univariate and multivariate Cox regression analysis models

Variables	Univariate analysis			Multivariate analysis		
	HR	95% CI	p	HR	95% CI	p
Age	1.038	0.996-1.083	0.080			
Gender	1.063	0.412-2.742	0.900			
DM	1.312	0.468-3.680	0.606			
HT	3.561	0.819-15.488	0.090			
LVEF	0.934	0.886-0.984	**0.011**	0.952	0.903-1.003	0.067
CRP	3.318	0.959-11.483	0.058			
Naples group	7.415	2.146-25.614	**0.002**	6.762	1.937-23.602	**0.003**

DM: Diabetes mellitus; HT: Hypertension; LVEF: Left ventricular ejection fraction; CRP: C-reactive protein; CI: Confidence interval; HR: Hazard ratio. Statistical significance is considered at a p-value of less than 0.05.

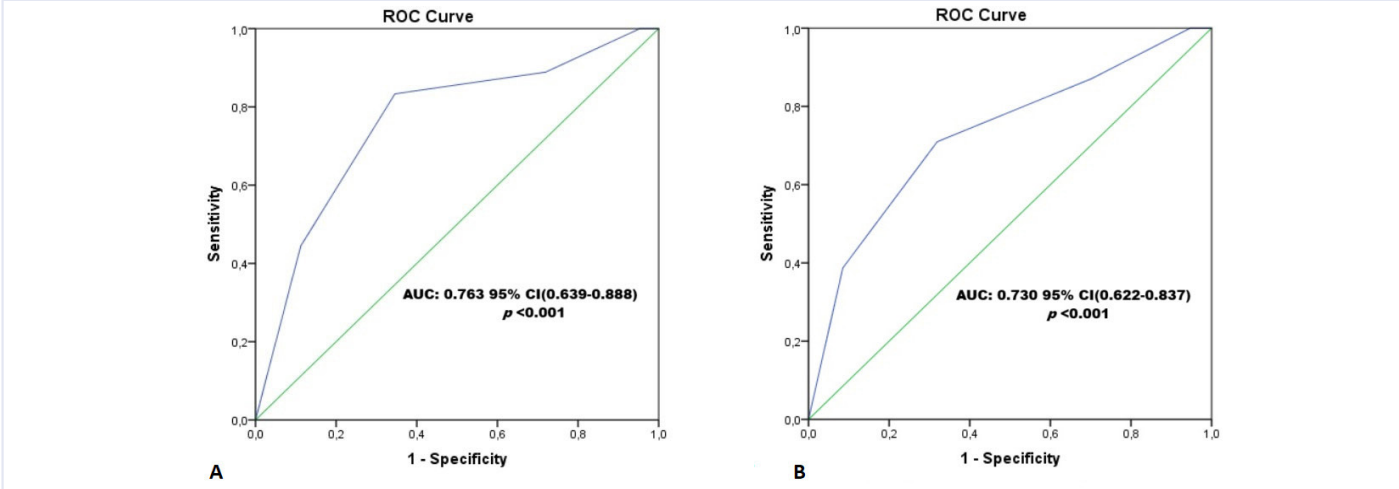

Figure 2. A. The ROC curve analysis of the Naples score, which predicts long-term mortality based on all causes, shows a cut-off value of 2.5, sensitivity of 83%, and specificity of 66%. **B.** The ROC curve analysis of the Naples score predicting MACE development, cut-off value 2.5, sensitivity 71%, and specificity 69%.

ROC: Receiver operating characteristic; AUC: Area under the curve; CI: Confidence interval; MACE: Major adverse cardiac events.

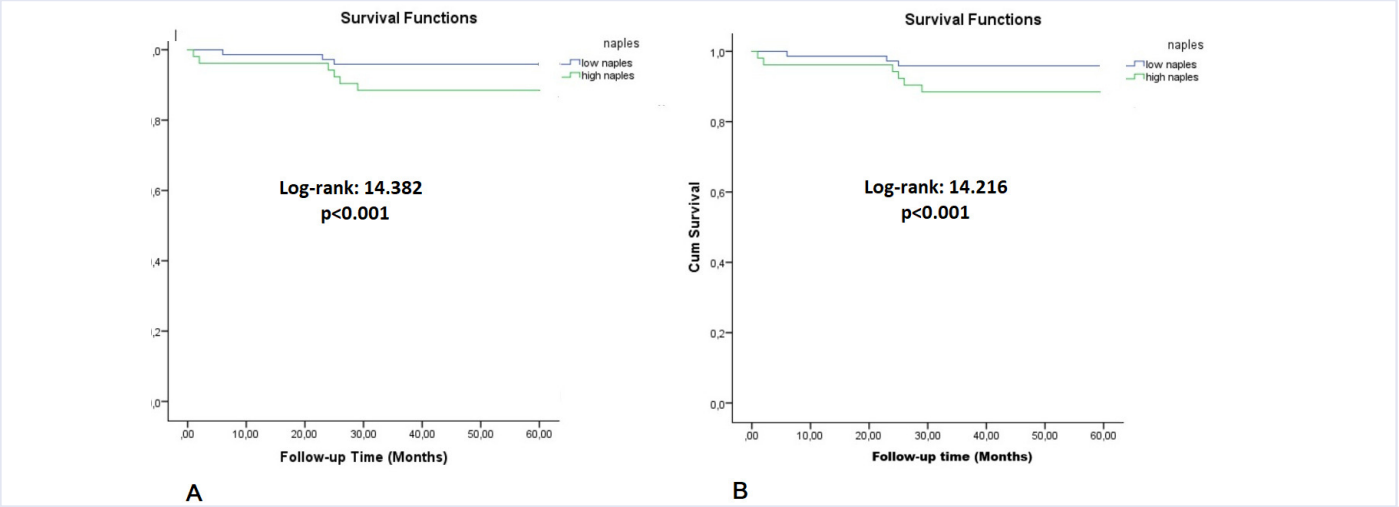

Figure 3. A. Kaplan-Meier survival curves comparing long-term mortality from all causes between groups with low and high Naples scores (log-rank test: 14.382, p<0.001) **B.** Kaplan-Meier survival curves showing MACE development between groups with low and high Naples scores (log-rank test: 14.216, p<0.001).

MACE: Major adverse cardiac events.

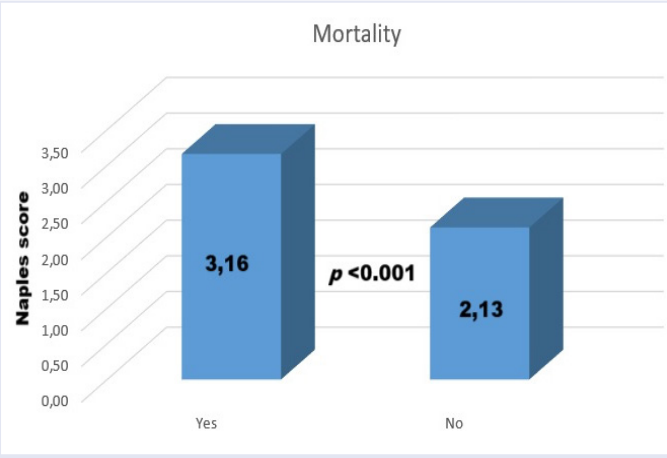

Figure 4. Naples score/mortality relationship: Bar chart showing average Naples scores in those with and without mortality.

It also contributes to poor survival rates by reflecting both systemic inflammation and malnutrition.[28] Indeed, a study by Bicciré et al.[29] demonstrated that low serum albumin values were connected to elevated death in patients with STEMI. Although hypercholesterolemia is a recognized major risk factor for CAD,[30] hypocholesterolemia—one of the components of the NPS—has been shown to be linked to elevated all-cause mortality in patients with CAD.[31] Although the underlying mechanism of this paradox remains unclear, several studies have showed an inverse correlation between TC values and mortality.[32,33]

Composed of lymphocyte count, serum albumin concentration, and TC values, the controlling nutritional status (CONUT) score has proven to be an effective means of determining nutritional status in hospitalized populations. The geriatric nutritional risk index (GNRI) is a practical and widely used scoring system for analyzing nutritional status and forecasting the risk of morbidity and mortality in older adults individuals. Both scores include the albumin parameter and are biomarkers specifically developed to assess nutritional status. Malnutrition, as evaluated by indices such as CONUT and GNRI, has been shown to be linked to higher rates of cardiovascular events and overall mortality.[34,35]

Validated risk assessment scores such as TIMI, GRACE, and CRUSADE have been developed to guide the handling of patients suffering from NSTEMI and the selection of optimal treatment strategies. While the TIMI and GRACE scores primarily emphasize myocardial ischemic injury and mortality, the CRUSADE score focuses on bleeding risk assessment. These prognostic tools have been validated in different populations and are widely recognized as valuable instruments for clinical practice. They provide essential guidance in estimating prognosis and shaping treatment strategies for patients presenting with NSTEMI.[36,37] Nonetheless, these scoring systems have limitations, as they do not incorporate critical factors such as inflammation and nutritional status, both of which substantially affect cardiovascular outcomes. As a composite marker of both inflammation and malnutrition, the NPS may offer a prognostic advantage over isolated biomarkers of either condition in patients with NSTEMI. This positions the NPS as a potentially more comprehensive indicator of long-term outcomes in this patient population.

In recent years, the NPS has been the subject of numerous studies as a prognostic indicator in cardiovascular diseases. Erdogan et al.[7] found that, in individuals with STEMI, the NPS was linked to both in-hospital and follow-up outcomes, concluding that it was an autonomous indicator of overall mortality and MACE. In line with these findings, Şaylık et al.[8] reported that the NPS may serve as a useful risk classification tool for evaluating long-term mortality in STEMI patients undergoing primary PCI. Birdal et al.[38] demonstrated that the NPS was markedly inversely related with EF and may aid in identifying high-risk patients with STEMI. These results indicate that the NPS could act as a valuable biomarker for risk classification and prognosis in STEMI patients. Furthermore, Aydın et al.[39] reported that the NPS is a promising separate marker of long-term mortality in individuals with heart failure. Moreover, studies on patients undergoing transcatheter aortic valve implantation for severe aortic stenosis have shown that the NPS is a valuable tool for predicting mortality and MACE.[40] These results suggest that the NPS may also be beneficial in patients with structural heart disease.

As evident from the literature, the prognostic value of the NPS has been showed in several cardiovascular diseases; however, its role in the context of NSTEMI remains particularly intriguing. In a study by Gitmez et al.,[10] the NPS was identified as a useful and autonomous indicator of one-year death in NSTEMI patients undergoing elective PCI. In this study, we sought to address the gap in the literature by examining the association of the NPS with both MACE and long-term all-cause mortality among NSTEMI patients. Our results indicate that the NPS could function as a practical risk marker for forecasting MACE and long-term mortality in this patient cohort.

The single-center and retrospective design of our study stands out as the main limitations. A further limitation of this study is the modest number of participants and clinical events, which may raise concerns regarding statistical power and model overfitting. This was due to the inclusion of consecutive patients within a specific time frame to minimize potential bias, along with the application of strict exclusion criteria to reduce the influence of confounding factors. Furthermore, the NPS was derived from laboratory data collected at hospital admission, without accounting for subsequent dynamic variations over time. Future wide-ranging, multi-institutional prospective research are needed to further clarify the predictive significance of the NPS in NSTEMI patients.

The NPS serves as an important and independent prognostic marker for both MACE and long-term overall mortality in NSTEMI patients. By integrating markers of both inflammation and malnutrition, the NPS enables a comprehensive approach to risk assessment. This makes it a more valuable tool compared to other biomarkers. Our findings support the potential utility of the NPS as an affordable and readily applicable tool to improve risk classification and support therapeutic management strategy in patients with NSTEMI.

Ethics

Ethics Committee Approval: The investigation was authorized by the Institutional Ethics Committee of Mardin Artuklu University (approval no: 2025/4-37, April 22, 2025), and the research was executed out in line with the principles of the Declaration of Helsinki.

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Concept: A.E., A.A., R.K., T.G., K.İ., M.Ö., M.Z.K.; Design: A.E., A.A., R.K., T.G., K.İ., M.Ö., M.Z.K.; Data Collection or Processing: A.E., A.A., R.K., T.G., K.İ., M.Ö., M.Z.K.; Analysis or Interpretation: A.E., A.A., R.K., T.G., K.İ., M.Ö., M.Z.K.; Literature Search: A.E., A.A., R.K., T.G., K.İ., M.Ö., M.Z.K.; Writing: A.E., A.A., R.K., T.G., K.İ., M.Ö., M.Z.K.

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

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