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Research Article

NUTRITIONAL STATUS AND CARDIOVASCULAR RISK: THE ROLE OF THE CONTROLLING NUTRITIONAL STATUS (CONUT) SCORE IN PATIENTS WITH ACUTE CORONARY SYNDROME TREATED WITH TICAGRELOR

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ABSTRACT

Objective: This study aimed to evaluate the prognostic value of the Controlling Nutritional Status (CONUT) score for predicting 1-year major adverse cardiovascular events (MACE) in patients hospitalized for acute coronary syndrome (ACS) and treated with the potent $P2Y_{12}$ inhibitor ticagrelor.

Materials and Methods: A total of 817 consecutive ACS patients admitted between September 2024 and July 2025 were prospectively enrolled. Patients with advanced malignancy, active infection, end-stage liver disease, or receiving other $P2Y_{12}$ inhibitors were excluded. The CONUT score was calculated from serum albumin, total cholesterol, and lymphocyte count. The primary endpoint was 1-year MACE, defined as a composite of cardiovascular death, myocardial infarction, stroke, or unplanned revascularization.

Results: MACE occurred in 72 patients (8.8%). Patients with MACE had higher age, hypertension, diabetes, and chronic kidney disease rates, and lower left ventricle ejection fraction (LVEF) and albumin levels ($p<0.05$). In the multivariate model, only lower LVEF (aOR 0.97; $p = 0.030$) and higher CONUT score (aOR 1.15; $p = 0.049$) remained independent predictors of MACE.

Conclusion: The CONUT score independently predicts 1-year MACE in ACS patients receiving contemporary ticagrelor therapy. Incorporating nutritional assessment into routine risk stratification may guide intensified secondary prevention strategies.

Keywords: Acute coronary syndrome, CONUT score, nutritional status, major adverse cardiovascular events, prognosis

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INTRODUCTION

Even with recent progress in therapy, acute coronary syndrome (ACS) still accounts for a significant proportion of cardiovascular disease burden and mortality internationally. (1). Traditional risk scores (e.g., GRACE, TIMI), although based on clinical and biochemical parameters, may not fully capture nutritional status. Recently, the importance of malnutrition in influencing cardiovascular events and long-term outcomes has been receiving increasing attention (2,3). Malnutrition can accelerate atherosclerosis and worsen clinical outcomes by affecting inflammation, immune function, and endothelial health (4,5).

However, despite increasing data on the impact of malnutrition on ACS prognosis, nutritional indices have still received relatively limited investigation in the contemporary ACS treatment era. One of the main reasons for this is that advanced antithrombotic therapies, early revascularization strategies, and guideline-based secondary prevention practices primarily focus on hemodynamic and ischemic risk markers. Furthermore, many key clinical trials in the ACS field have not systematically incorporated nutritional assessment into risk models. Therefore, malnutrition is often overlooked, particularly in patients who are clinically stable or treated with potent P2Y12 inhibitors such as ticagrelor. Furthermore, pathophysiological mechanisms related to nutritional status, such as inflammation, immune dysregulation, and metabolic imbalance, can negatively impact plaque instability, microvascular dysfunction, and myocardial healing, even under optimal pharmacoinvasive therapy.

Studies addressing the impact of various biomarkers reflecting nutritional status on ACS prognosis have increased in recent years. Nutritional indices reflecting inflammatory activity and immune system dysfunction are associated with an increased risk of mortality and major adverse cardiovascular events (MACE) following ACS (6,7). Moreover, in patients with acute coronary syndrome undergoing percutaneous coronary intervention, high-sensitivity C-reactive protein (hs-CRP) and the Nutritional Risk Index (NRI) — as markers of inflammation and nutritional status — were shown to be independently and jointly associated with adverse long-term cardiovascular outcomes (8). These findings suggest that nutritional status is a determinant not only of overall clinical well-being but also of mechanisms underlying cardiovascular processes, including inflammation, the immune response, and myocardial recovery. The impact of nutritional status on clinical outcomes has been linked to inflammation, thrombosis, and myocardial recovery, particularly in patients with ACS. Therefore, assessing nutritional status with simple and accessible scores may be valuable in clinical practice. Therefore, our study aimed to evaluate the contribution of nutritional status to clinical outcomes under modern antiplatelet therapy.

As a straightforward index that incorporates serum albumin, total cholesterol, and lymphocyte levels, the Controlling Nutritional Status (CONUT) score serves as a readily accessible measure of nutritional condition. (9). Evidence from previous studies indicates that elevated CONUT scores are associated with higher risks of MACE, overall mortality, and repeated hospital admissions among patients with coronary artery disease (10,11). Investigations into ACS populations suggest that the CONUT score meaningfully enhances risk stratification for MACE, particularly in patients of advanced age or those with comorbidities. (12,13). However, studies investigating the independent prognostic value of the CONUT score in current ACS cohorts under a homogeneous treatment protocol (e.g., use of a potent P2Y12 inhibitor) are limited.

This study aimed to evaluate the predictive value of the CONUT score for 1-year MACE in consecutive patients hospitalized for ACS and treated with the potent P2Y12 inhibitor ticagrelor. Our objective was to investigate whether nutritional status provides additional prognostic information for risk stratification when current standards of care are applied.

MATERIALS AND METHODS

This investigation, conducted as a prospective observational study, comprised consecutive individuals presenting with ACS between September 2024 and July 2025. ACS subtypes were defined based on the 2023 European Society of Cardiology (ESC) guidelines (1). Accordingly, ST-segment elevation myocardial infarction (STEMI) was defined as persistent ST elevation in 2 or more contiguous leads and/or new left bundle branch block, with cardiac biomarker positivity. Non-ST-elevation myocardial infarction (NSTEMI) was defined as cases with elevated cardiac biomarkers without ST elevation. Unstable angina (UA) was defined as cases with ischemia symptoms but without cardiac biomarker elevation and persistent ST elevation. All eligible patients were initiated on ticagrelor-based dual antiplatelet therapy. Primary percutaneous coronary intervention (PCI) was performed for STEMI patients, and early

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Table 1. Association Between Comorbidities and 1-Year MACE

Variable	MACE=0 (n=745)	MACE=1 (n=72)	Total (n = 817)	p
Age(years)	60.0 [52.0–68.0]	65.5 [52.8–75.2]	61.0 [52.0–69.0]	0.019
Gender (Male)	596/745 (80.0%)	51/72 (70.8%)	647 (79.2%)	0.093
CKD	91/745 (12.2%)	25/72 (34.7%)	116 (14.2%)	<0.001
DM	247/745 (33.2%)	40/72 (55.6%)	287 (35.1%)	<0.001
HT	417/745 (56.0%)	53/72 (73.6%)	470 (57.5%)	0.006
COPD	19/745 (2.6%)	6/72 (8.3%)	25 (3.1%)	0.018
Smoking	382/745 (51.3%)	32/72 (44.4%)	414 (50.7%)	0.325
CVA	5/745 (0.7%)	1/72 (1.4%)	6 (0.7%)	1.0
LVEF(%)	45.00 [40.00-55.00]	42.50 [35.00-50.00]	45.0 [39.0–55.0]	0.006

Abbreviations: CKD: Chronic kidney disease, DM: Diabetes mellitus, HT: Hypertension, COPD: Chronic obstructive pulmonary disease, CVA: Cerebrovascular accident, LVEF: Left ventricular ejection fraction, MACE: Major adverse cardiovascular events

invasive strategies were used for NSTEMI and UA patients. Medical treatment was preferred when PCI was contraindicated or the patient refused intervention.

The study included patients aged 18 years and older who were diagnosed with ACS and initiated on ticagrelor during hospitalization. Patients with advanced malignancy, cachexia, active infection, end-stage liver disease, contraindications to ticagrelor, a history of clopidogrel or prasugrel use, and those with missing nutritional parameters were excluded (Figure 1). Left ventricular ejection fraction (LVEF) was measured using transthoracic echocardiography using the biplane Simpson method. Diabetes mellitus (DM) was defined by medical history, use of antidiabetic therapy, or fasting glucose ⩾126 mg/dL or HbA1c ⩾6.5% at presentation. Hypertension (HT) was defined by medical history, use of antihypertensive treatment, or positive blood pressure measurements (⩾140/90 mmHg) at admission. Chronic kidney disease (CKD) was defined as an eGFR below 60 mL/min/1.73 m². Chronic obstructive pulmonary disease was defined by prior diagnosis or regular use of fbronchodilator/inhaled corticosteroid therapy. A history of prior stroke or transient ischemic attack is defined as cerebrovascular disease. Thrombolysis In Myocardial Infarction (TIMI) flow scores were recorded before and after PCI. No-reflow was defined as inadequate myocardial perfusion despite successful epicardial vessel recanalization. According to the TIMI flow grade, post-PCI TIMI 0–2 flow in the infarct-related artery was considered indicative of the no-reflow phenomenon. Inflammatory markers, including white blood cell count and hs-CRP level, were evaluated when available.

Table 2. Association Between Complications and 1-Year MACE

Variable	MACE=0 (n=745)	MACE=1 (n=72)	Total (n = 817)	p
No-reflow	51/745 (6.8%)	11/72 (15.3%)	62 (7.6%)	0.019
Segmental wall motion abnormalities	566/654 (86.5%)	63/66 (95.5%)	629/720 (87.4%)	0.060
Contrast-induced nephropathy	14/745 (1.9%)	3/72 (4.2%)	17 (2.1%)	0.386

Abbreviations: MACE: Major adverse cardiovascular events

Nutritional status was calculated using the CONUT score, which is based on albumin (g/dL), total cholesterol (mg/dL), and lymphocyte (×10³/μL) levels (9). A score of 0–1 represents normal nutritional status, 2–4 represents mild malnutrition, 5–8 represents moderate malnutrition, and 9–12 represents severe malnutrition. The primary endpoint of the study was the 1-year occurrence of MACE, which included cardiovascular death, myocardial infarction, stroke, and unplanned revascularization. Statin therapy, nutritional support, or dietitian recommendations at discharge were recorded, if any; however, the lack of systematic recording of nutritional therapy information was considered a limitation of the study. Ethical approval for the study was granted by the local committee (reference 1775; September 18, 2024), and the work was undertaken in alignment with the Declaration of Helsinki.

Statistical Analysis

SPSS version 22 was used for data processing. Owing to the lack of normal distribution, the distribution of continuous variables was summarized by medians and IQRs, and categorical variables were represented as numerical counts and proportions. For comparisons of the MACE and non-MACE groups, the Mann–Whitney U test was applied to continuous data. In contrast, categorical data were analyzed with chi-square or Fisher's exact test. To explore predictors of MACE, univariate logistic regression was applied. The multivariate regression model

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Table 3. Association Between Laboratory Parameters and 1-Year MACE

Variable	MACE=0 Median [IQR]	MACE=1 Median [IQR]	Total (N = 817) Median [IQR]	p
CONUT score	1[0-2]	2[1-3]	1 [0–2]	0.002
Albumin(mg/dl)	3.65 [3.34-3.88]	3.45 [3.15-3.75]	3.62 [3.30–3.86]	0.006
WBC(x10³ /μL)	9.30 [8.00-11.00]	9.60 [7.88-11.15]	9.32 [7.98–11.05]	0.592
Platelets(x10³ /μL)	258.00 [219.00-306.00]	244.00 [208.25-308.75]	256.00 [218.00–306.00]	0.681
LDL-C(mg/dl)	126.70 [103.00-155.75]	112.70 [84.60-144.70]	124.00 [100.00–154.00]	0.055
HbA1c (%)	6.35 [5.90-8.10]	6.90 [6.10-8.60]	6.40 [5.90–8.20]	0.115
hs-CRP(mg/dl)	31.60 [13.78-69.12]	38.70 [18.75-69.62]	32.40 [14.50–69.30]	0.120
Total cholesterol(mg/dl)	200.00 [168.00-234.00]	191.50 [160.00-224.00]	199.00 [166.00–232.00]	0.134
AST (U/L)	43.0 [28.0–69.0]	37.0 [24.0–61.5]	42.0 [27.0–67.0]	0.190
ALT (U/L)	31.0 [22.0–49.0]	29.5 [21.2–44.0]	30.8 [21.5–48.0]	0.472
Sodium (mmol/L)	139 [137–142]	139 [136–141]	139 [137–142]	0.650
Potasyum (mmol/L)	4.2 [3.9–4.4]	4.2 [3.8–4.5]	4.2 [3.9–4.4]	0.720

Abbreviations: WBC: White blood cell, LDL: Low-density lipoprotein cholesterol, HbA1c: Hemoglobin A1c, hs-CRP: High-sensitivity C-reactive protein, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, MACE: Major adverse cardiovascular events

was constructed using variables that reached a p-value of less than 0.10 in univariate analysis, combined with established clinical predictors (age, sex, LVEF, and CONUT). To avoid multicollinearity, CKD and DM were excluded from the adjusted model, as both conditions are strongly associated with nutritional status and directly influence key components of the CONUT score, particularly serum albumin and total cholesterol levels. Including these comorbidities together with the CONUT score could obscure the independent prognostic value of nutritional status; therefore, they were excluded to allow a clearer interpretation of the association between CONUT score and clinical outcomes. We expressed the findings as odds ratios (OR) together with their 95% confidence intervals (CI). A p-value of less than 0.05, based on a two-sided test, was considered statistically significant. ROC curve analysis was performed to assess the predictive performance of the CONUT score, and AUC values were reported. Kaplan–Meier survival analysis was considered; however, due to limited event numbers and insufficient statistical power, this analysis was not conducted. A post-hoc power analysis for the ROC curve was performed using MedCalc (MedCalc Software, Ostend, Belgium). For an AUC of 0.585 versus the null hypothesis value of 0.5, with 72 cases and 745 controls and a two-sided α of 0.05, the achieved statistical power was approximately 0.68.

Table 4: Univariate Logistic Regression Analysis for 1-Year MACE

Variable	OR	95% CI	p
CKD	3.823	[2.24-6.51]	0.0000
DM	2.520	[1.55-4.11]	0.0002
CONUT score	1.239	[1.09-1.41]	0.0011
LVEF	0.964	[0.94-0.99]	0.0032
HT	2.194	[1.27-3.78]	0.0046
Albumin	0.436	[0.25-0.78]	0.0047
Age	1.026	[1.01-1.05]	0.0103
Gender(male)	0.607	[0.35-1.04]	0.0696
Total cholesterol	0.996	[0.99-1.00]	0.1308
HbA1c	1.082	[0.95-1.23]	0.2269
Smoking	0.760	[0.47-1.24]	0.2694
hs-CRP	1.001	[1.00-1.01]	0.5223
WBC	0.986	[0.93-1.05]	0.6504
Platelets	0.999	[1.00-1.00]	0.7548

Abbreviations: CKD: Chronic kidney disease, DM: Diabetes mellitus, HT: Hypertension, LVEF: Left ventricular ejection fraction, MACE: Major adverse cardiovascular events, HbA1c: Hemoglobin A1c, hs-CRP: High-sensitivity C-reactive protein

RESULTS

A total of 817 patients were evaluated, and 72 of them (8.8%) experienced MACE within one year. Individuals who developed MACE were older (65.5 [52.8–75.2] vs. 60.0 [52.0–68.0] years; p=0.019) and showed a higher prevalence of HT (73.6% vs. 56.0%; p=0.006), DM (55.6% vs. 33.2%; p<0.001), and CKD (34.7% vs. 12.2%; p<0.001) compared with those without MACE (Table 1). LVEF was significantly reduced in the MACE group (42.5 [35.0–50.0] vs. 45.0 [40.0–55.0]; p=0.006) (Table 1). As demonstrated, the incidence of the no-reflow phenomenon was significantly higher in patients with MACE, while other complications did not show a statistically significant association with outcomes (Table 2). In laboratory assessments, serum albumin levels were lower in patients with MACE (3.45 [3.15–3.75] vs. 3.65 [3.34–3.88]; p = 0.0056), whereas AST, ALT, sodium, and potassium levels showed no significant differences between the groups (Table 3).

Table 5: Multivariatüe Logistic Regression Analysis for 1-Year MACE

Variable	Adjusted OR	95% CI	p
LVEF	0.97	[0.94–1.00]	0.030
CONUT score	1.15	[1.01–1.33]	0.049
HT	1.66	[0.91–3.03]	0.101
Age	1.01	[0.99–1.03]	0.424
Gender(male)	0.87	[0.47–1.62]	0.665

Abbreviations: HT: Hypertension, LVEF: Left ventricular ejection fraction,

Univariate logistic regression analysis revealed that CKD (OR 3.82, 95% CI 2.24–6.51; p<0.001), DM (OR 2.52, 95% CI 1.55–4.11; p=0.0002), HT (OR 2.19, 95% CI 1.27–3.78; p=0.0046), lower LVEF (OR 0.96, 95% CI 0.94–0.99; p=0.0032), higher CONUT score (OR 1.24, 95% CI 1.09–1.41;

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p=0.0011), reduced albumin (OR 0.44, 95% CI 0.25–0.78; p=0.0047), and older age (OR 1.03, 95% CI 1.01–1.05; p=0.010) were all associated with MACE (Table 4).

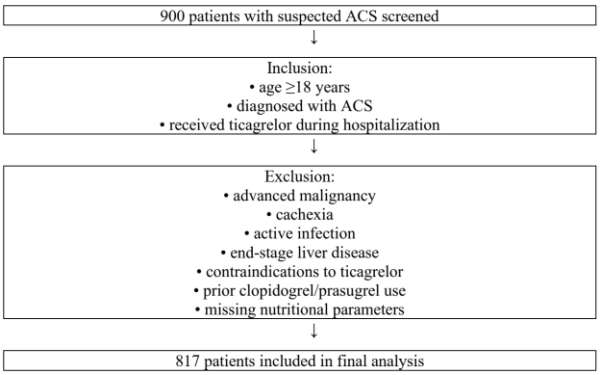

Figure 1. Study flow diagram

In the multivariate model, which excluded DM and CKD to avoid collinearity, both lower LVEF (aOR 0.97, 95% CI 0.94–1.00; p = 0.030) and elevated CONUT score (aOR 1.15, 95% CI 1.01–1.33; p = 0.049) remained independent predictors of 1-year MACE. Neither HT, age, nor sex showed an independent relationship with adverse outcomes (Table 5).

The ROC analysis demonstrated a significant association between the CONUT score and 1-year MACE, with an area under the curve (AUC) of 0.585 (95% CI: 0.510–0.640; p= 0.041) (Figure 2).

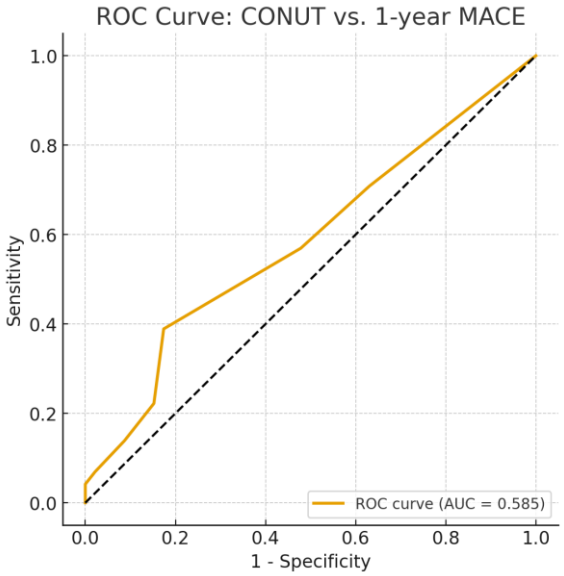

Figure 2. Receiver operating characteristic (ROC) curve showing the predictive value of the CONUT score for 1-year major adverse cardiovascular events (MACE).

DISCUSSION

Results revealed that, within this prospective observational cohort, a high CONUT score independently predicted the likelihood of 1-year MACE in patients with ACS treated with ticagrelor alone. Patients using clopidogrel or prasugrel were excluded from the study to ensure treatment homogeneity. The CONUT score remained significant even after removing DM and CKD variables from the multivariate model, a modeling strategy deliberately chosen to avoid multicollinearity, as diabetes mellitus and chronic kidney disease are closely linked to nutritional status and directly influence key components of the CONUT score, including serum albumin and total cholesterol levels. This approach allowed a clearer assessment of the independent prognostic value of nutritional status, although residual confounding related to these comorbidities cannot be entirely excluded. These findings demonstrate that nutritional status has prognostic significance independent of classical cardiovascular risk factors. Although the ROC analysis for the CONUT score was statistically significant, its discriminative performance was modest, suggesting that CONUT should be considered a complementary risk marker rather than a standalone prognostic tool. This finding is consistent with meta-analyses and cohort studies in the literature, which highlight the impact of malnutrition on cardiovascular prognosis (14,15). Previous studies have shown that the CONUT score predicts mortality and MACE in coronary artery disease and ACS populations (15-17). Deng et al. reported that the risk of MACE was significantly increased in patients with STEMI who underwent primary PCI with a CONUT score of ⩾5 (16). Zengin et al. also reported that a high CONUT score independently predicted long-term MACE and mortality in patients with STEMI who underwent primary PCI (17). Similarly, Yurdam and Kiş's demonstration that TIMI flow and MAPH scores correlate in STEMI patients undergoing primary PCI, and that low microvascular perfusion is associated with poorer clinical outcomes, highlights the prognostic importance of microcirculatory integrity (18). The adverse outcomes observed in patients with high CONUT scores in our study may also be explained by mechanisms such as inflammation, endothelial dysfunction, and impaired microvascular healing. These findings suggest that malnutrition may worsen cardiovascular prognosis by affecting

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microcirculation and may pose an increased risk even with potent antiplatelet therapy.

The pathophysiological mechanisms underlying the relationship between malnutrition and cardiovascular events are multifaceted. Impaired atherosclerotic plaque stability and increased thrombogenicity are also mechanisms that explain the association between CONUT and MACE. Upregulated pro-inflammatory mediators, such as TNF-α, IL-1β, and IL-6, may increase intraplaque cellular infiltration, leading to thinning of the fibrotic cap and expansion of the necrotic core, thereby increasing the risk of rupture (19). Furthermore, malnutrition impairs endothelial function and affects coronary microcirculation, increasing susceptibility to ischemia (20). Low serum albumin may indicate chronic inflammation and oxidative stress, in addition to malnutrition (21,22). It may negatively impact cardiovascular prognosis by decreasing plasma oncotic pressure and causing endothelial dysfunction (23). Lymphocyte count is a simple biomarker reflecting immune response capacity. Lymphopenia is associated with increased sympathetic activity and systemic inflammation, which impair T-cell and NK cell function, exacerbate plaque inflammation, and reinforce a prothrombotic environment. This contributes to poor prognosis by increasing thrombus burden and myocardial damage after plaque rupture (24). It has been reported that lymphopenia functions as an independent predictor of prognosis for both early mortality and the development of MACE. (25,26). Low total cholesterol levels may increase inflammatory responses by impairing cell membrane fluidity and immune regulation, contributing to the fragility of atherosclerotic plaques (27). Furthermore, low total cholesterol levels may impair membrane integrity and energy reserves, reducing healing capacity (28). The association of higher CONUT scores with higher adverse event rates in our study suggests that secondary prevention strategies, including nutritional optimization, may be clinically beneficial in this patient group.

Additionally, other indices used in the literature to assess nutritional status, such as the Prognostic Nutritional Index (PNI) and the Geriatric Nutritional Risk Index (GNRI), are similarly associated with cardiovascular outcomes (29,30). Furthermore, the inclusion of lymphocyte count in the CONUT score may partially reflect the inflammatory burden. Therefore, direct comparisons between CONUT, PNI, GNRI, and inflammatory markers (e.g., NLR) will be significant in future studies to determine the most appropriate approach to cardiovascular risk stratification. A notable strength of this research is that it was carried out in a homogeneous population treated exclusively with ticagrelor. In the current literature, most studies examining the relationship between the CONUT score and ACS prognosis have included patients receiving different P2Y12 inhibitors. However, different antiplatelet agents may have distinct effects on platelet activity, inflammation levels, and myocardial healing. This makes it difficult to assess the prognostic contribution of nutritional markers independently of treatment effects. A key methodological strength of our study is the inclusion of consecutive ACS patients treated exclusively with ticagrelor. This eliminates treatment-related heterogeneity, allowing for a clearer understanding of the independent prognostic value of the CONUT score. Our findings demonstrate that malnutrition continues to affect clinical outcomes even under robust antiplatelet therapy, underscoring the importance of nutritional assessments in contemporary ACS management. Patients with high CONUT scores may benefit from individualized nutritional assessment and dietitian-guided nutritional support in addition to standard antiplatelet therapy.

This work is subject to certain limitations. The most important is its single-center design and modest sample size, which may limit the extent to which the results can be generalized. The fact that the entire patient population was treated solely with ticagrelor leaves it unclear whether the prognostic value of the CONUT score is similar across patient groups receiving different $P2Y_{12}$ inhibitors. Nutritional status was assessed at admission with a single measurement, and changes over time were not considered. Although the study was prospectively designed, its observational nature precludes causal inference regarding the relationship between the CONUT score and 1-year MACE. While excluding DM and CKD variables from the multivariate analysis was chosen to mitigate collinearity, the actual effects of these comorbidities on MACE may not have been fully differentiated.

Furthermore, since inflammatory biomarkers (CRP, IL-6, NLR, etc.) and other nutritional scores (PNI, GNRI) were not included in the study, direct comparisons between the CONUT score and these parameters were not possible. Information on the type of acute coronary syndrome and discharge medications was not recorded, which may limit subgroup analyses. However, all patients were discharged on guideline-directed optimal medical therapy, as per institutional practice.

CONCLUSION

The simplicity, low cost, and calculability of the CONUT score from routine laboratory parameters facilitate its

integration into clinical practice. Close monitoring, intensive secondary prevention strategies, and nutritional support may be considered in ACS patients with high CONUT scores. Future multicenter, randomized studies may determine whether models incorporating CONUT in combination with various inflammatory indices improve prognostic value.

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Authorship contributions
Concept: A.K.D., A.A.B. Design: A.K.D., Y.D. Data Collection or Processing: A.A.B., Y.D. Analysis or Interpretation: A.K.D., O.Ş. Literature Search: Y.D., O.Ş. Writing: A.K.D. Critical Review: O.Ş.

Data availibity statement
The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declaration of competing interest
The authors declare no competing interests.

Ethics
This study was approved by the Non-Interventional Ethics Committee of İzmir Bakırçay University (reference 1775; September 18, 2024).

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