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Clinical Significance of the Naples Prognostic Score in Patients with Coronary Artery Ectasia: A Novel Biomarker of Inflammation and Nutrition

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ABSTRACT

Coronary artery ectasia (CAE) is an abnormal dilatation of coronary arteries associated with ischemic complications and thrombotic events. Systemic inflammation and impaired nutritional status are believed to play a pivotal role in its pathogenesis. The Naples Prognostic Score (NPS), a composite marker of inflammation and nutrition, has been associated with adverse cardiovascular outcomes. This study aimed to evaluate the relationship between NPS and the presence of CAE.

We retrospectively analyzed 139 patients who underwent coronary angiography between May 2023 and February 2026. Patients were classified into CAE (n=26) and control (n=113) groups. NPS was calculated based on serum albumin, total cholesterol, neutrophil-to-lymphocyte ratio (NLR), and lymphocyte-to-monocyte ratio (LMR). Univariable and multiple logistic regression analyses were performed to identify predictors of CAE. Receiver operating characteristic (ROC) analysis assessed the discriminatory ability of NPS.

Patients with CAE exhibited significantly higher NPS values compared to controls (2.15 ± 1.4 vs. 1.42 ± 0.9; p=0.025). Multiple analysis demonstrated that NPS was an independent predictor of CAE (OR: 2.018, 95% CI: 1.301–3.129, p=0.002). ROC analysis revealed good discriminatory performance for NPS (AUC: 0.759, p<0.001).

Higher NPS values are independently associated with the presence of CAE. NPS may serve as a simple, accessible biomarker reflecting systemic inflammation and malnutrition, providing additional risk stratification in patients undergoing coronary evaluation.

Keywords: Coronary artery ectasia; Naples Prognostic Score; Systemic inflammation; Nutritional status; Coronary angiography; Biomarkers

Introduction

Coronary artery ectasia (CAE) is characterized by an abnormal dilation of coronary arteries, defined as an enlargement of at least 1.5 times wider than the diameter of the healthy arterial segments. The prevalence of CAE among patients undergoing coronary angiography has been reported to range between 0.15% and 5.3% (1-5). While CAE is often diagnosed incidentally, it is not merely a morphological variant but a pathology capable of leading to potentially serious clinical outcomes (6).

CAE can lead to slowed coronary blood flow, potentially resulting in myocardial ischemia, angina, thrombus formation, and rarely, myocardial infarction (7). Furthermore, turbulent blood flow within ectatic segments increases the risk of thromboembolic complications. Consequently, there is growing recognition that CAE represents not only an anatomical anomaly but also a clinically significant condition (8).

Inflammation is among the most implicated mechanisms in the pathogenesis of CAE. Histopathological studies have revealed elastic lamina degeneration, destruction within the medial layer, and inflammatory cell infiltration in ectatic vessels. Additionally, other mechanisms such as oxidative stress, vascular remodeling, matrix

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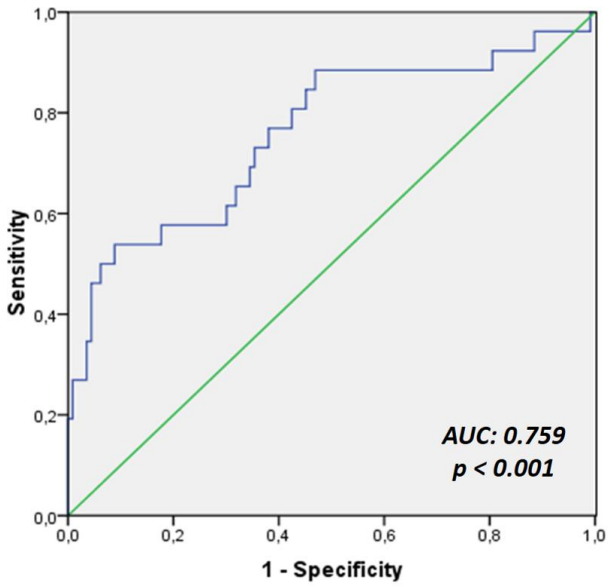

Fig. 1. ROC curve analysis demonstrated the performance of NPS in predicting CAE

metalloproteinase activity, and endothelial dysfunction may also play roles in the development of CAE (8-10).

Biomarkers combining assessments of inflammation and nutritional status have shown promise in diagnosing and prognosticating cardiovascular diseases. The Naples Prognostic Score (NPS) is a novel composite scoring system evaluating systemic inflammation and nutritional status (11). NPS incorporates serum albumin, total cholesterol, neutrophil-to-lymphocyte ratio (NLR), and lymphocyte-to-monocyte ratio (LMR), assigning a scoring range from 0 to 4 based on these parameters. Initially proposed for predicting prognosis in oncological diseases, NPS has recently been associated with cardiovascular diseases such as coronary artery disease, acute myocardial infarction, and chronic kidney disease (12-14).

A higher NPS, reflecting both impaired nutritional status and heightened systemic inflammation, has recently been recognized as a potential biomarker associated with the development and progression of various vascular diseases. However, to the best of our knowledge, the present study is the first in the medical literature to specifically investigate the association between the Naples Prognostic Score and coronary artery ectasia. By exploring this relationship, we aim to evaluate whether NPS could serve as a novel, accessible biomarker for predicting the presence of CAE in clinical practice

Materials and Methods

Study population: Patients with chronic coronary syndrome who were referred for invasive coronary angiography based on coronary computed tomography angiography findings between May 2023 and February 2026 were retrospectively screened for eligibility. CAE was detected in 37 patients. The study excluded patients with malignancies (n = 3), individuals with chronic liver disease (n = 1), and patients with end-stage kidney disease (n = 7). In addition, patients who were already receiving lipid-lowering therapy at the time of index evaluation were excluded. After applying the exclusion criteria, the final study population consisted of 139 patients (CAE group: 26 and control group: 113). Patients were subsequently divided into two groups; the CAE group and the control group. Baseline demographic information and laboratory results were recorded from the electronic medical record system of the hospital. The NPS consists of four components: total cholesterol, serum albumin, lymphocyte-to-monocyte ratio (LMR), and neutrophil-to-lymphocyte ratio (NLR) (11). Patients were classified into three categories based on their NPS: Group 1 included patients with a score of 0, Group 2 included those with scores of 1 or 2, and Group 3 included patients with scores of 3 or 4 (Table 1).

CAE was defined as coronary artery dilatation involving more than one-third of the vessel length, with the diameter of the dilated segment measuring more than 1.5 times that of an adjacent normal segment (1).

Ethical Approval: The present study was conducted in full compliance with the ethical principles outlined in the Declaration of Helsinki and its later amendments. The study protocol was reviewed and approved by a local ethics committee (approval date: 14 May 2026; protocol number: 2026-91). Given the retrospective case-control design of the study, the requirement for obtaining written informed consent from the participants was waived.

Statistical Analysis: Data analysis was conducted using SPSS software version 22.0 (SPSS Inc., Chicago, IL, USA) and R software (R Core Team, 2020) via RStudio (RStudio Team, 2020). The Kolmogorov-Smirnov test was used to assess the normality of continuous variables. Normally distributed variables were presented as mean ± standard deviation, while non-normally distributed variables were expressed as median (interquartile range). Categorical variables were summarized as

Table 1: Naples Prognostic Score Variables

Variables	Cut-off value	Points	NPS group
Serum albumin (mg/dL)	≥ 4	0	
	< 4	1	
Total cholesterol (mg/dL)	> 180	0	Group 1: 0 point
	≤ 180	1	Group 2: 1 or 2 points
NLR	≤ 2.96	0	Group 3: 3 or 4 points
	> 2.96	1	
LMR	> 4.44	0	
	≤ 4.44	1	

NPS: Naples prognostic score, NLR: Neutrophil-to-lymphocyte ratio, LMR: Lymphocyte-to-monocyte ratio

frequencies and percentages. Group comparisons were performed using the Independent Samples t-test for normally distributed continuous variables, the Mann–Whitney U test for non-normally distributed variables, and the Chi-square or Fisher's exact test for categorical variables, as appropriate. Correlation analyses were performed using Pearson's correlation coefficient for normally distributed variables and Spearman's rank correlation coefficient for non-normally distributed variables. A p-value < 0.05 was considered statistically significant.

Multiple binary logistic regression analysis was conducted to identify independent predictors of CAE, with results reported as odds ratios (ORs) and 95% confidence intervals (CIs). Receiver operating characteristic (ROC) curve analysis was used to evaluate the predictive performance of the NPS for the presence of CAE. The area under the curve (AUC) was calculated to assess the discriminatory ability.

Results

The study comprised 139 patients, with an average age of 62.6 ± 9.4 years, of whom 31.7% were female. Patients were categorized into two groups: the CAE group (n: 26) and the control group (n: 113). Table 2 provides an overview of the demographic and clinical features of both groups. No significant differences were observed in terms of age, gender, hypertension, diabetes mellitus, dyslipidemia, smoking, chronic obstructive pulmonary disease, and the left ventricular ejection fraction between the groups. Fasting blood glucose, creatinine, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglyceride, and hemoglobin levels did not differ significantly between the groups. Albumin levels were significantly lower in CAE group [3.9 (3-4.3)] compared to the control group [4.2 (3.3-5.2)] (p = 0.005). C-reactive protein (CRP) levels were notably elevated in the CAE group [2.1 (0.1-17.5)] compared to the control group [1.6 (0.1-6.8)] (p = 0.027). Additionally, the NPS was significantly higher in the CAE group (2.15 ± 1.4) versus the control group (1.42 ± 0.9) (p = 0.025), with substantial differences observed in NPS group distribution (p < 0.001) (Table 2).

Univariable logistic regression analysis identified the NPS as a significant predictor of CAE (OR: 1.862, 95% CI: 1.242–2.793, p = 0.003). Although CRP (p = 0.062), smoking (p = 0.089), and gender (p = 0.199) showed potential associations in univariable analysis, they did not reach statistical significance and were not included in the final multiple model. In the multiple logistic regression analysis, NPS remained an independent predictor of CAE (OR: 2.018, 95% CI: 1.301–3.129, p = 0.002) (Table 3). ROC curve analysis was used to assess the discriminatory performance of NPS in predicting CAE. The area under the curve (AUC) for NPS was 0.759 (95% CI: 0.643–0.875, p < 0.001), indicating good discriminatory ability (Figure 1). Although the inclusion of the NAPLES score increased the predictive performance of the model from an AUC of 0.680 to 0.759, this improvement did not reach statistical significance according to DeLong's test (Z = 1.35, p = 0.176, 95% CI for AUC difference: −0.035 to 0.193).

Discussion

To the best of our knowledge, this is among the first clinical studies to demonstrate that higher Naples Prognostic Score (NPS) values are associated with the presence of coronary artery ectasia (CAE). In this study, we investigated the association between the NPS - a composite marker of systemic inflammation and nutritional status - and the presence of CAE in patients

Table 2: Baseline Characteristics of The Patients According to The Coronary Artery Ectasia

		CAE group (n = 26)	Control group (n = 113)	Total (n = 139)	p value
Age, mean ± SD		62.3±8.9	62.7±9.6	62.6±9.4	0.819
Gender (Female), n (%)		11 (42.3)	33 (29.2)	44 (31.7)	0.195
Hypertension, n (%)		6 (23.1)	49 (43.4)	55 (39.6)	0.056
Diabetes mellitus, n (%)		5 (19.2)	16 (14.2)	21 (15.1)	0.546
Dyslipidemia, n (%)		1 (3.8)	17 (15)	18 (12.9)	0.195
Smoking, n (%)		5 (19.2)	42 (37.2)	47 (33.8)	0.081
Chronic obstructive pulmonary disease, n (%)		4 (15.4)	25 (22.1)	29 (20.9)	0.446
Left ventricular ejection fraction, mean ± SD		61.8±2.1	61.8±2.2	61.8±2.2	0.977
Fasting blood glucose, mg/dL, median (IQR)		104.5 (117)	99 (208)	99 (208)	0.527
Creatinine, mg/dL, mean ± SD		0.8±0.1	0.9±0.1	0.9±0.1	0.144
ALT, units/liter, median (IQR)		19 (31)	21 (8-8274)	21 (74)	0.661
AST, units/liter, median (IQR)		21.5 (21)	21 (192)	21 (192)	0.745
Total cholesterol, mg/dL, mean ± SD		205±55.5	189.7±47.6	192.5±49.4	0.155
LDL-C, mg/dL, mean ± SD		123.7±45	115±36.7	116.6±38.4	0.300
HDL-C, mg/dL, mean ± SD		48.8±11.5	45±11.6	45.7±11.6	0.141
Triglyceride, mg/dL, median (IQR)		102.5 (208)	126 (438)	124 (448)	0.395
Hemoglobin, g/dL, mean ± SD		14±1.5	13.9±1.4	13.9±1.5	0.622
CRP, mg/dL, median (IQR)		2.1 (17.4)	1.6 (6.7)	2 (17.4)	0.027
Albumin, g/dL, median (IQR)		3.9 (1.3)	4.2 (1.9)	4.1 (2.2)	0.005
Coronary artery calcium score, median (IQR)		111 (632)	90 (827)	97 (827)	0.305
NPS, mean ± SD		2.15±1.4	1.42±0.9	1.56±1	0.025
NPS group, n(%)	1	5 (19.2)	16 (14.2)	21 (15.1)	<0.001
	2	8 (30.8)	89 (78.8)	97 (69.8)	
	3	13 (50)	8 (7.1)	21 (15.1)	

CAE: Coronary artery ectasia, ALT: Alanine transaminase, AST: Aspartate transaminase, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, CRP: C-reactive protein, NPS: Naples prognostic score, IQR: Interquartile range

Table 3: Univariable and Multiple Logistic Regression Analyses of Factors Associated with Coronary Artery Ectasia

Univariable	p value	OR	95% CI		Multiple	p value	OR	95% CI	
			Lower	Upper				Lower	Upper
Gender	0.199	1.778	0.739	4.275	Gender	-	-	-	-
Smoking	0.089	2.485	0.872	7.081	Smoking	-	-	-	-
CRP	0.062	1.316	0.986	1.756	CRP	-	-	-	-
Coronary artery calcium score	0.438	1.001	0.998	1.003	Coronary artery calcium score	-	-	-	-
NPS	0.003	1.862	1.242	2.793	NPS	0.002	2.018	1.301	3.129

OR: Odds ratio, NPS: Naples prognostic score

undergoing coronary angiography. Our findings demonstrated that a higher NPS was independently associated with the presence of CAE and that NPS had moderate discriminatory power for identifying patients with CAE.

CAE, historically considered a benign anatomical variant, is now recognized as a distinct pathological entity associated with myocardial ischemia, thrombotic complications, and adverse cardiovascular outcomes (1,7). The pathogenesis of CAE is multifactorial, with inflammation playing a central role alongside oxidative stress, extracellular matrix degradation, and vascular remodeling. Histopathological studies of ectatic coronary arteries consistently reveal features suggestive of chronic inflammation, including infiltration of lymphocytes, macrophages, and neutrophils into the vascular wall, destruction of the medial layer, fragmentation of elastic fibers, and increased expression of matrix metalloproteinases (MMPs). These enzymes degrade extracellular matrix proteins such as elastin and collagen, leading to weakening and dilatation of the vascular wall (15-18). Elevated levels of inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β), have also been detected in patients with CAE, further supporting an inflammatory-driven mechanism. Moreover, systemic markers of inflammation — such as CRP, erythrocyte sedimentation rate (ESR), and leukocyte-derived ratios like NLR — have been reported to be elevated in patients with CAE (19). These findings suggest that CAE may represent part of a broader inflammatory vasculopathy, sharing mechanistic overlaps with conditions like Kawasaki disease, Takayasu arteritis, and other vasculitic syndromes (20,21).

The NPS provides a comprehensive evaluation by simultaneously capturing markers of systemic inflammation and nutritional status. It is calculated by scoring four laboratory parameters — serum albumin, total cholesterol, neutrophil-to-lymphocyte ratio (NLR), and lymphocyte-to-monocyte ratio (LMR) — each assigned a value of 0 or 1, yielding a total score ranging from 0 to 4. Higher NPS values reflect a greater degree of systemic inflammation and poorer nutritional reserves. The prognostic value of NPS stems from its multi-component structure; while NLR and LMR reflect the activation of the innate and adaptive immune systems, serum albumin and total cholesterol levels serve as vital indicators of nutritional resilience and antioxidant capacity. Although initially introduced as a prognostic tool in oncology, the utility of the NPS has expanded into the cardiovascular field. Recent studies have demonstrated that elevated NPS levels are associated with increased mortality, adverse procedural outcomes, and a higher incidence of major cardiovascular events in populations with coronary artery disease, acute myocardial infarction, and chronic kidney disease (11-14). In patients with CAE, the synergism between chronic low-grade inflammation and malnutrition, as reflected by high NPS, may accelerate the degradation of the medial layer and facilitate progressive vessel dilatation.

From a mechanistic standpoint, a high NPS may reflect a chronic inflammatory state that promotes vascular injury, endothelial dysfunction, and extracellular matrix degradation — all key processes implicated in the formation of coronary ectasia. Hypoalbuminemia and hypocholesterolemia, components of NPS, may further exacerbate vascular vulnerability by impairing antioxidant defenses and reducing membrane stability.

In our study, patients with CAE exhibited significantly higher NPS values compared to those without ectasia. Multiple logistic regression confirmed that the NPS remained an independent

predictor of CAE after adjusting for other potential confounders. Moreover, receiver operating characteristic (ROC) curve analysis demonstrated that the NPS had a good discriminatory ability (AUC: 0.759) for identifying patients with CAE. Although the inclusion of NPS increased the model's AUC from 0.680 to 0.759, this improvement did not reach statistical significance ($p = 0.176$), likely due to the limited sample size. Nonetheless, NPS remained an independent predictor of CAE in multiple analysis, supporting its potential value in clinical risk assessment. Our results align with this growing body of evidence. We found significantly lower serum albumin and higher CRP levels in patients with CAE compared to controls, both of which are components of the systemic inflammatory response. More importantly, the NPS — which simultaneously captures inflammatory status (via NLR and LMR) and nutritional status (via albumin and cholesterol levels) — was independently associated with CAE presence, even after adjusting for other potential confounders. It is notable that while CRP alone was higher in CAE patients, it did not independently predict CAE in multiple analysis. This suggests that composite indices like the NPS, which integrate multiple biological pathways, may provide a more comprehensive reflection of the complex pathophysiological milieu underlying CAE than single biomarkers alone. The systemic inflammatory environment in CAE may contribute to endothelial dysfunction, promote a prothrombotic state, and facilitate ongoing vascular remodeling - all of which are likely important not only in the development of ectatic lesions but also in the associated ischemic and thrombotic clinical events. Furthermore, systemic inflammation may perpetuate a vicious cycle of vascular injury and maladaptive repair, leading to progressive vessel dilatation over time (22).

To the best of our knowledge, this is the first study to explore the relationship between NPS and CAE. Our findings extend previous observations that higher NPS levels are associated with poor outcomes in coronary artery disease, acute myocardial infarction, and chronic kidney disease, and suggest that systemic inflammation and impaired nutritional reserves may similarly contribute to the pathogenesis of CAE. However, several limitations should be acknowledged. The retrospective, single-center nature of the study may limit generalizability. The relatively small number of CAE cases could have affected the statistical power to detect other potential associations. Despite these limitations, our study provides a novel perspective on CAE and paves the way for future multicenter prospective studies involving larger cohorts and advanced imaging techniques like IVUS or OCT to further elucidate these mechanistic pathways. Additionally, although we applied standard angiographic definitions of CAE, the lack of intravascular imaging data may have limited our ability to characterize vessel wall pathology in detail. Furthermore, we were unable to perform external validation using an independent dataset, which limits the generalizability of our results. Although our findings demonstrate a significant association between NPS and CAE in this study, future studies involving larger, multicenter populations with external validation cohorts are warranted to confirm these observations. Lastly, although an association between NPS and CAE was demonstrated, causality cannot be established based on observational data alone. Future prospective studies with larger sample sizes and adjunctive imaging techniques, such as optical coherence tomography or intravascular ultrasound, are needed to further elucidate the mechanistic pathways linking systemic inflammation, malnutrition, and the development and progression of CAE.

In conclusion, our study suggests that a higher NPS is independently associated with the presence of CAE. NPS, by integrating systemic inflammation and nutritional status, may help identify patients with CAE. However, given the retrospective design and absence of outcome data, its role in risk stratification remains speculative and requires validation through prospective studies. Further large-scale studies are needed to confirm these findings and to elucidate the potential role of NPS in the risk stratification and management of CAE.

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