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Segment Yükselmeli Miyokart Enfarktüsü ile Başvuran Stent Trombozlu Hastalarda Non-HDL Kolesterol/HDL Kolesterol Oranının Prognostik ve Prediktif Değeri

Prognostic and Predictive Value of the Non-HDL Cholesterol/HDL Cholesterol Ratio in Patients with Stent Thrombosis Presenting with ST-Segment Elevation Myocardial Infarction

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ÖZ

Giriş: Non-HDL kolesterol ile HDL kolesterol oranı (non-HDL-C/HDL-C oranı; NHHR) ile stent trombozu (ST) zemininde gelişen ST-segment elevasyonlu miyokart enfarktüsü (STEMI) arasındaki ilişki yeterince araştırılmamıştır. Bu çalışmada, NHHR'nin bu hasta grubundaki prognostik değerini değerlendirmeyi amaçladık.

Yöntem: Ocak 2020 ile Nisan 2024 tarihleri arasında ST'ye bağlı STEMI nedeniyle primer PTCA uygulanan toplam 168 hasta retrospektif olarak incelendi. Birincil sonlanım noktası, 1 yıllık tüm nedenlere bağlı mortaliteydi. Hastalar NHHR değerlerine göre <3 ve ≥3 olmak üzere iki gruba ayrıldı.

Bulgular: Bir yıllık takip süresince 35 hasta (%20,8) hayatını kaybetti. Mortalite grubundaki hastalar, sağ kalanlara kıyasla daha yaşlıydı (64 vs. 58 yaş, p = 0,049), daha sık kadın cinsiyetteydi (%48,6 vs. %30,1, p = 0,040) ve hiperkolesterolemi, kalp yetersizliği ve başvuru Killip ≥2 oranları daha yüksekti (tüm p < 0,05). Bu hastalarda LVEF daha düşüktü (yüzde 41 vs. 45, p = 0,009) ve başvuru glukoz, kreatinin, CRP değerleri anlamlı olarak yüksekti. Ayrıca, HDL kolesterol ve hemoglobin düzeyleri mortalite grubunda daha düşüktü. NHHR ≥3 olan hastaların oranı mortalite grubunda anlamlı olarak daha fazlaydı (%71,4 vs. %51,1, p = 0,032) ve bu hastalarda 1 yıllık mortalite oranı daha yüksekti (%26,8 vs. %13,3, p = 0,032). Çok değişkenli Cox regresyon analizinde, NHHR ≥3 değeri 1 yıllık mortalite için bağımsız bir öngördürücü olarak saptandı (HR = 2,751; %95 GA: 1,048–7,221; p = 0,040).

Sonuç: ST'ye bağlı STEMI ile başvuran hastalarda 1 yıllık mortalite halen yüksektir. Non-HDL-C/HDL-C oranının yüksek oluşu, bu hasta grubunda artmış mortalite ile bağımsız olarak ilişkili bulunmuştur. NHHR, bu yüksek riskli hasta grubunda kolay erişilebilir, basit ve değerli bir risk belirteci olabilir.

Anahtar Kelimeler: stent trombozu, ST elevasyonlu miyokart enfarktüsü, non-HDL/HDL kolesterol oranı, mortalite

ABSTRACT

Objective: The relationship between the non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (non-HDL-C/HDL-C ratio, NHHR) and stent thrombosis (ST) in the setting of ST-elevation myocardial infarction (STEMI) has been understudied. We aimed to evaluate the prognostic value of NHHR in patients presenting with STEMI due to ST.

Method: This retrospective study included 168 patients who underwent primary PCI for ST-related STEMI between January 2020 and April 2024. The primary outcome was 1-year all-cause mortality. Patients were stratified according to NHHR: <3 or ≥3.

Results: During 1-year follow-up, 35 patients (20.8%) died. Compared with survivors, non-survivors were older (64 vs. 58 years, p = 0.049), more often female (48.6% vs. 30.1%, p = 0.040), and had higher rates of hypercholesterolemia, heart failure, and Killip class ≥2 (all p < 0.05). They also had lower LVEF (41% vs. 45%, p = 0.009), and higher admission levels of creatinine, CRP, and glucose. HDL-C and hemoglobin were significantly lower in non-survivors. An NHHR ≥3 was more frequent in the mortality group (71.4% vs. 51.1%, p = 0.032), and associated with higher 1-year mortality (26.8% vs. 13.3%, p = 0.032). In multivariate Cox regression, NHHR ≥3 remained an independent predictor of 1-year mortality (HR = 2.751; 95% CI, 1.048–7.221; p = 0.040).

Conclusion: Among patients presenting with STEMI due to ST, an elevated non-HDL-C/HDL-C ratio was independently associated with increased 1-year all-cause mortality. NHHR may serve as a simple, accessible biomarker for risk stratification in this high-risk population.

Keywords: stent thrombosis, ST-elevation myocardial infarction, non-HDL/HDL cholesterol ratio, mortality

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INTRODUCTION

Stent thrombosis (STH) represents a significant complication of percutaneous coronary intervention (PCI), often presenting as acute myocardial infarction (MI) or acute coronary syndrome (ACS) and is linked to considerable morbidity and mortality (1). The incidence of STH in the first-year post-implantation varies between 0.5% and 1.0% (2). Previous clinical studies indicated mortality rates near 30% (3). However, recent data, frequently confirmed by autopsy-confirmed events, demonstrate improved survival rates under 10% in current practice (3). STH occurs more frequently in patients with ST-elevation myocardial infarction (STEMI) who undergo primary PCI (2). The sudden occlusion of the stented artery presents a considerable risk for recurrent MI and mortality. Approximately 20% of patients who experience an initial episode of STH may have a recurrence within two years (4). Drug-eluting stents (DES) markedly decrease restenosis rates in comparison to bare-metal stents; however, early-generation DES exhibited a heightened risk of STH (5). Data regarding the predictors and frequency of STH with new-generation DESs remain limited.

Non-high-density lipoprotein cholesterol (non-HDL-C) is defined as total cholesterol minus HDL-C and includes all atherogenic lipoproteins (6). Recent findings indicate that non-HDL-C serves as an independent and significant predictor of cardiovascular risk, suggesting its inclusion as a secondary treatment target in high-risk groups (7). The non-HDL-C to HDL-C ratio has been gaining attention for its significant correlation with metabolic syndrome and its enhanced efficacy compared to traditional lipid measures in evaluating atherosclerotic burden (8, 9). Nevertheless, there is an absence of data regarding its association with STH. This study investigates the association between the non-HDL-C/HDL-C ratio and STH, evaluating its potential as a prognostic and predictive biomarker in patients with STH who present with STEMI.

MATERIALS AND METHODS

Patients and Study Design

This retrospective study included 2,922 patients who were admitted to our cardiology department with STEMI between January 2020 and April 2024. Among these, 181 patients (6.2%) were diagnosed with STH. Patients were excluded if they had missing data regarding lipid profiles or follow-up, had received thrombolytic therapy, or had end-stage malignancy. The study included the remaining 168 patients who fulfilled the inclusion criteria of receiving primary PCI for stent thrombosis and presented with STEMI. The study was approved by the local institutional ethics committee.

Clinical Evaluation

The diagnosis of STEMI was made by the Fourth Universal Definition of Myocardial Infarction (10). Clinical and laboratory data were retrieved from hospital records, and patients were compared based on 1-year all-cause mortality. The clinical evaluation included baseline demographic and clinical characteristics such as age, sex, presence of hypertension, diabetes mellitus, and hypercholesterolemia; concomitant comorbidities including history of stroke; prior MI, PCI, and coronary artery bypass grafting (CABG); STEMI subgroup classification; Killip class at admission; left ventricular ejection fraction (LVEF);and discharge medications. Laboratory parameters assessed at admission included serum glucose, hemoglobin, white blood cell (WBC) count, platelet count, serum creatinine, and estimated glomerular filtration rate (eGFR). Inflammatory and metabolic markers such as C-reactive protein (CRP), hemoglobin A1c, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), non-HDL-C, and the non-HDL-C/HDL-C ratio were also recorded.

Venous blood samples were obtained at the time of hospital admission following a fasting period of 10 to 12 hours. Serum concentrations of fasting lipid parameters, including TC, TG, LDL-C, and HDL-C, along with other biochemical and hematological markers, were measured using standard laboratory techniques. eGFR was calculated using the Levey-modified Modification of Diet in Renal Disease (MDRD) formula as follows: (186.3 x serum Cr [mg/dL]-1.154) x (age [years]-0.203) x (0.742 if female) (11). The non-HDL-C to HDL-C ratio was calculated using the following formula: non-HDL-C/HDL-C ratio = (TC − HDL-C) / HDL-C (12).

Data Analysis

The study population was divided into two groups based on non-HDL-C/HDL-C ratio values: patients with a ratio < 3 (n = 75) and those with a ratio ≥ 3 (n = 93). The primary clinical endpoints were in-hospital and follow-up outcomes, including in-hospital mortality, Killip class at admission, clinically significant bleeding, acute kidney injury (AKI), medication requiring hypotension, stroke, repeat PCI, and 1-year all-cause mortality. In-hospital events were assessed through a review of the institutional electronic medical records, while 1-year mortality data were obtained from the national death notification system.

Statistical Analysis

The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Continuous variables were expressed as mean ± standard deviation and compared between groups using the independent samples t-test or the Mann–Whitney U test, as appropriate. Categorical variables were presented as counts and percentages, and comparisons between groups were performed using the chi-square test or Fisher's exact test, where applicable. A two-tailed p-value of <0.05 was considered statistically significant.

To evaluate the impact of the non-HDL-C/HDL-C ratio on 1-year all-cause mortality, a Cox proportional hazards regression model was constructed. Covariates included in the model were age, sex, admission serum creatinine, LVEF, and Killip class ≥2 at admission, using a forward stepwise method. Variables with a p-value <0.10 in univariate analysis or those deemed clinically relevant were entered into the multivariate model. The predictive performance of the non-HDL-C/HDL-C ratio for 1-year mortality was assessed using receiver operating characteristic (ROC) curve analysis. Statistical analyses were performed using SPSS software version 20.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Of the 168 patients admitted with STEMI and diagnosed with stent STH, 35 patients (20.8%) died during the 1-year follow-up period. The baseline clinical and demographic characteristics of the study patients are summarized in Table 1.Compared with survivors, patients in the mortality

group were older (64 vs. 58 years, p = 0.049), more likely to be female (48.6% vs. 30.1%, p = 0.040), and had a higher prevalence of hypercholesterolemia (31.4% vs. 26.3%, p = 0.030) and heart failure (25.7% vs. 11.3%, p = 0.030). Additionally, non-survivors had lower LVEF (41% vs. 45%, p = 0.009) and were more likely to present with a higher Killip class on admission (p < 0.001).

The biochemical and hematological parameters of the study patients are summarized in Table 2. Compared with survivors, patients in the mortality group had significantly higher admission glucose levels (152 vs. 139 mg/dL, p = 0.025), admission creatinine (1.59 vs. 1.02 mg/dL, p < 0.001), peak creatinine (2.48 vs. 1.26 mg/dL, p < 0.001), and admission CRP levels (21.8 vs. 5.4 mg/L, p < 0.001). In contrast, non-survivors had significantly lower HDL cholesterol (37 vs. 40 mg/dL, p = 0.047) and hemoglobin levels (12.5 vs. 13.4 g/dL, p = 0.043).Furthermore, the proportion of patients with a non-HDL-C/HDL-C ratio ≥ 3 was significantly higher in the mortality group compared with the survivor group (71.4% vs. 51.1%, p = 0.032).

Table 3 summarizes in-hospital and follow-up outcomes according to the non-HDL-C/HDL-C ratio. There were no statistically significant differences between the groups concerning in-hospital outcomes, including in-hospital mortality, admission Killip class, clinically important bleeding, acute kidney injury, stroke, repeat PCI, or hypotension requiring medication. However, the 1-year all-cause mortality rate was significantly higher among patients with a non-HDL-C/HDL-C ratio ≥3 compared with those with a ratio <3 (26.8% vs. 13.3%, p = 0.032) (Figure 1).

Cox regression analysis identified the non-HDL-C/HDL-C ratio (hazard ratio [HR] = 2.751; 95% confidence interval [CI], 1.048–7.221; p = 0.040), age (HR = 1.066; 95% CI, 1.012–1.123; p = 0.016), admission creatinine (HR = 2.115; 95% CI, 1.123–3.984; p = 0.020), and Killip class ≥2 at presentation (HR = 8.041; 95% CI, 5.033–13.141; p < 0.001) as independent predictors of 1-year all-cause mortality (Table 4).

The diagnostic performance of the non-HDL-C/HDL-C ratio in predicting 1-year mortality is illustrated in the ROC curve shown in Figure 2. The area under the curve (AUC) was 0.692 (95% CI, 0.640–0.744; p = 0.047), indicating modest discriminative ability.

Table 1. Baseline Clinical and Demographic Characteristics of Study Group According to 1-Year All-Cause Mortality. [a]

Variables	Total n=168	Deceased n= 35	Survivors n= 133	p
Age, years	59±9	64±10	58±9	**0.049**
Sex, female %	33.9	48.6	30.1	**0.040**
DM, %	26.8	28.6	26.3	0.780
Hypertension, %	39.3	48.6	36.8	0.206
Hypercholesterolemia, %	27.4	31.4	26.3	**0.030**
Previous MI, %	10.9	13.4	7.3	0.546
Previous CABG, %	4.2	2.9	4.5	0.140
COPD, %	61.5	16.7	16.2	0.868
Prior Heart Failure, %	14.3	25.7	11.3	**0.030**
LVEF, %	44±10	41±11	45±9	**0.009**
Hospital presentation, %				**0.046**
Anterior STEMI	49.4	71.4	45.2	
Inferior STEMI	42.5	21.4	46.6	
Lateral STEMI	11.5	7.1	12.3	
Posterior STEMI	9.2	14..3	8.2	
Killip class ≥2, %	21.2	60.0	9.8	**<0.001**
Discharge medication, %				
Clopidogrel	42.4	42.9	42.3	0.953
Ticagrelor	47.3	17.1	55.4	**<0.001**
Prasugrel	1.2	0.0	1.5	0.460

[a] Data are presented as mean ± standard deviation or n (%). Statistically significant p values shown in boldface.
Abbreviations: DM: diabetes mellitus, MI: myocardial infarction, PCI: Percutaneous coronary intervention, CABG: coronary artery bypass grafting, COPD: chronic obstructive pulmonary disease, LVEF: left ventricular ejection fraction, STEMI: ST-segment Elevation Myocardial Infarction.

Table 2. Biochemical and Hematological Parameters of Study Group According to 1-Year All-Cause Mortality. [a]

Variables	Total n= 168	Deceased n= 35	Survivors n= 133	p
Glucose, mg/dL	141±94	152±99	139±93	**0.025**
Hgb, g/dL	13.2±1.8	12.5±2.1	13.4±1.6	**0.043**
WBC, $\times 10^9$ /L	11.3±4.0	12.1±5.1	11.1±3.6	0.340
PLT count, $\times 10^9$ /L	262±82	254±107	264±74	0.570
Admission Cr, mg/dl	1.16±0.8	1.59±1.5	1.02±0.4	**<0.001**
Maximum Cr, mg/dl	1.51±1.2	2.48±2.1	1.26±0.7	**<0.001**
Admission CRP	6.41±2.7	21.8±6.0	5.4±2.3	**<0.001**
HbA1c	6.1±0.6	6.2±0.8	6.1±0.5	0.241
ALT	21±31	22±38	21±29	0.868
AST	31±66	39±113	28±53	0.166
Total cholesterol, mg/dL	170±39	174±41	169±39	0.806
LDL cholesterol, mg/dL	107±39	110±36	107±40	0.643
HDL cholesterol, mg/dL	40±10	37±10	40±10	**0.047**
Triglyceride, mg/dL	125±59	132±56	124±60	0.373
Non-HDL cholesterol, mg/dL	131±39	137±42	129±38	0.413
Non-HDL/HDL-C ratio	3.51±1.5	4.10±2.0	3.36±1.3	0.064
Non-HDL/HDL-C ratio ≥3, %	55.4	71.4	51.1	**0.032**

[a] Data are presented as mean ± standard deviation or n (%). Statistically significant p values shown in boldface.
Abbreviations: Hgb: hemoglobin, WBC: white blood cell, PLT: platelet, eGFR: glomerular filtration rate, Cr: creatinine, CRP: C reactive protein, HbA1c: hemoglobin A1c, ALT: Alanine transaminase; AST: Aspartate aminotransferase, LDL: low density lipoprotein, HDL: high density lipoprotein, AIP: atherogenic index of plasma.

Table 3. In-Hospital and Follow-Up Outcomes of Study Group According to Non-HDL/HDL-C Ratio [A]

Variables	Non-HDL/HDL-C ratio < 3 n: 75	Non-HDL/HDL-C ratio ≥ 3 n: 93	p
In-hospital outcomes			
Mortality, %	8.0	10.7	0.486
Killip class ≥2, %	13.7	22.6	0.810
Bleeding, %	5.3	8.7	0.403
Acute kidney injury, %	37.9	36.7	0.897
Hypotension (medication required)	10.7	21.5	0.061
Stroke	0.2	0.1	0.191
Repeat PCI	0.1	0.2	0.441
Follow-up outcomes			
1-year mortality, %	13.3	26.8	**0.032**

[a] Data are presented as n (%). Statistically significant p values shown in boldface.

Table 4. Cox Regression Analysis to Detect Independent Predictors of 1-Year All-Cause Mortality

Variables	Multivariate		
	HR	(95% CI)	p
Non-HDL/HDL-C ratio	2.751	1.048-7.221	**0.040**
Sex, female	0.780	0.306-1.986	0.620
Age	1.066	1.012-1123	**0.016**
Admission Cr	2.115	1.123-3.984	**0.020**
LVEF	0.998	0.951-1.047	0.931
Killip ≥ 2	8.041	5.033-13.141	**<0.001**

HR: hazard ratio: CI: confidence interval HDL: high density lipoprotein; Cr: creatinine.

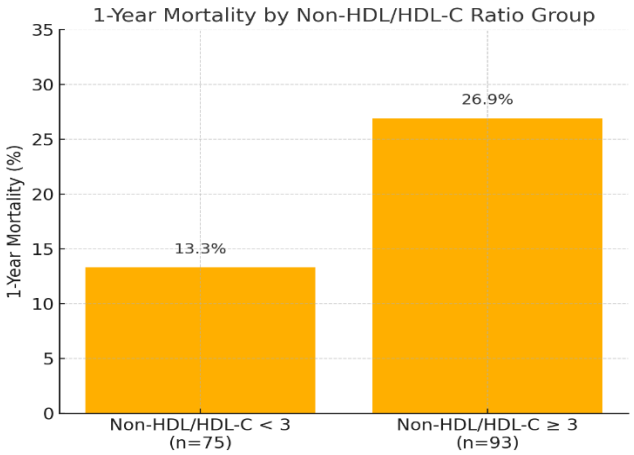

Figure 1. Comparison of 1-year all-cause mortality rates according to non-HDL-C/HDL-C ratio groups in patients presenting with STEMI due to stent thrombosis.

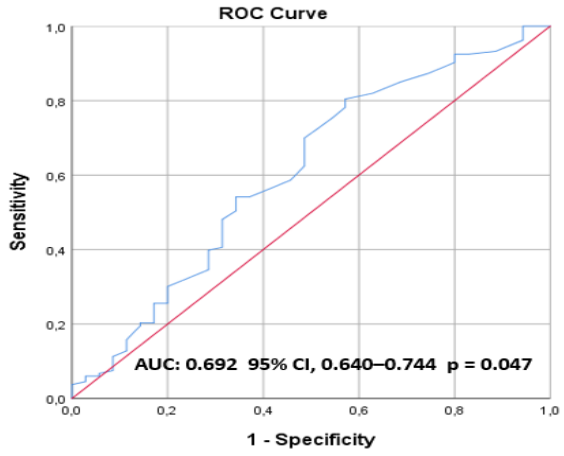

Figure 2. Receiver operating characteristic (ROC) curve evaluating the predictive value of the non-HDL-C/HDL-C ratio for 1-year all-cause mortality in patients presenting with STEMI due to stent thrombosis.

DISCUSSION

In our study, 20.8% of patients presenting with STEMI and diagnosed with STH died during the 1-year follow-up period. When compared with survivors, patients who died were significantly older and more frequently presented with anterior STEMI. They also had a higher non-HDL-C/HDL-C ratio, elevated admission creatinine and CRP levels, and lower hemoglobin concentrations. Importantly, a high non-HDL-C/HDL-C ratio was identified as an independent predictor of 1-year all-cause mortality

The role of HDL-C in CAD is well established. Numerous studies have shown that low HDL-C levels are associated with increased risk of CAD (13). Similarly, elevated levels of LDL-C and TG are also recognized as significant contributors to CAD risk (14, 15). Currently, LDL-C remains the primary target in the management of hypercholesterolemia. In line with previous findings, our study demonstrated that patients with STH who died within 1 year of follow-up had lower HDL-C levels and a higher prevalence of hypercholesterolemia. Nevertheless, evidence indicates the individuals with low LDL-C and high HDL-C levels may furthermore have an increased risk for CAD (16). This result has led researchers to investigate alternate lipid-based indicators for improved prediction of cardiovascular risk (16). In this regard, non-HDL-C levels and the non-HDL-C/HDL-C ratio have been identified as significant indicators (8). Multiple studies have demonstrated that variations in these parameters correlate with atherosclerotic cardiovascular risk (8, 17). The association between elevated non-HDL-C/HDL-C ratios and negative cardiovascular outcomes is well established in the general population; however, information concerning patients with STH is limited (18). This study contributes to existing literature by indicating that a higher non-HDL-C/HDL-C ratio may correlate with elevated 1-year mortality in patients with STH.

STH represents a significant and potentially life-threatening complication related to coronary stent implantation (19). Previous research indicates that patients who suffer from STH are at a markedly increased risk for recurrent myocardial infarction and mortality (20). Kuramitsu et al. found that patients with STH had poorer long-term clinical outcomes than control patients. Second-generation DES have demonstrated a reduction in the incidence of STH compared to first-generation DES; however, STH remains a significant risk, even with the advancement of newer-generation stents (2). Identifying risk factors for STH following coronary stent implantation is essential. Recent studies have examined potential predictors to enable early identification and timely management of patients at high risk of STH (21, 22). Nevertheless, data on this particular topic is still limited. This study contributes to the subject by presenting 1-year follow-up data from a cohort of patients with documented STH. The non-HDL-C/HDL-C ratio was significantly elevated in patients who died during follow-up in comparison to survivors. Previous studies indicate an elevated non-HDL-C/HDL-C ratio to increased cardiovascular mortality in general populations (23). Our study is, to our knowledge, the first to demonstrate this association specifically in patients with stent thrombosis.

Systemic inflammation is significantly elevated in individuals diagnosed with CAD (24). Increased inflammatory status correlates with a higher disease burden, more extensive coronary involvement, and elevated

mortality rates in patients with CAD (25, 26). Coronary stent implantation is a significant treatment for obstructive CAD; however, stent thrombosis remains a considerable complication, even with advancements in stent technology and pharmacotherapy (3). Inflammation and hypercoagulability are acknowledged as significant factors in the pathogenesis of STH. Increased levels of inflammatory markers can facilitate thrombogenesis by activating coagulation pathways and impairing vascular endothelial cells (27). Prior research has established that increased systemic inflammation when coronary stenting serves as a risk factor for acute stent thrombosis (20, 28). Our study confirmed these findings, displaying that admission CRP levels were significantly elevated in patients who died during the 1-year follow-up period. The findings indicate that baseline CRP levels may serve as prognostic indicators for the severity of the condition and long-term outcomes in patients with stent thrombosis.

This study has several limitations, many of which are inherent to retrospective observational research. First, it was a single-center study, which may limit the generalizability of the findings. Second, the relatively small sample size restricted the statistical power to evaluate all clinical outcomes, and follow-up was limited to 1 year. The time interval between stent implantation and the occurrence of stent thrombosis was not available for all patients. Intravascular imaging techniques were not routinely utilized, potentially limiting insights into mechanical predictors of stent thrombosis, such as stent malapposition or underexpansion. Additionally, data regarding patients' adherence to prescribed medications, a significant factor that may affect long-term outcomes, were not available

CONCLUSION

In patients with STH presenting with STMEI, 1-year mortality remains high despite contemporary management strategies. Our findings highlight that advanced age, anterior infarct localization, impaired renal function, systemic inflammation, and anemia are associated with increased long-term mortality. Most notably, an elevated non-HDL-C/HDL-C ratio emerged as an independent predictor of 1-year all-cause mortality. These results suggest that the non-HDL-C/HDL-C ratio may serve as a valuable and readily accessible biomarker for risk stratification in this high-risk population.

Ethics Committee Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Ethics committee approval was obtained from the Clinical Research Ethics Committee.

Authors' contributions: Authors' Contributions. OSS, YD, and GC contributed to the acquisition of data, the analysis and interpretation of data, revising the manuscript critically for important intellectual content, and the final approval of the version to be published. ED, EK, SSY and MC contributed to the acquisition of data, the analysis and interpretation of data, the drafting of the article, and the final approval of the version to be published. BBK and KK made substantial contributions to conception and design, drafting the article, and final approval of the version to be published..

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REFERENCES

1. Palmerini T, Dangas G, Mehran R, Caixeta A, Généreux P, Fahy MP, et al. Predictors and implications of stent thrombosis in non-ST-segment elevation acute coronary syndromes: the ACUITY Trial. Circ Cardiovasc Interv. 2011;4(6):577-584.

2. Kuramitsu S, Ohya M, Shinozaki T, Otake H, Horie K, Kawamoto H, et al. Risk Factors and Long-Term Clinical Outcomes of Second-Generation Drug-Eluting Stent Thrombosis. Circ Cardiovasc Interv. 2019;12(6):e007822.

3. Klein LW, Nathan S, Maehara A, Messenger J, Mintz GS, Ali ZA, et al. SCAI Expert Consensus Statement on Management of In-Stent Restenosis and Stent Thrombosis. J Soc Cardiovasc Angiogr Interv. 2023;2(4):100971.

4. Kirtane AJ, Stone GW. How to minimize stent thrombosis. Circulation. 2011;124(11):1283-1287.

5. Gurbel PA, Bliden KP, Guyer K, Cho PW, Zaman KA, Kreutz RP, et al. Platelet reactivity in patients and recurrent events post-stenting: results of the PREPARE POST-STENTING Study. J Am Coll Cardiol. 2005;46(10):1820-1826.

6. Verbeek R, Hovingh GK, Boekholdt SM. Non-high-density lipoprotein cholesterol: current status as cardiovascular marker. Curr Opin Lipidol. 2015;26(6):502-510.

7. Carbayo Herencia JA, Simarro Rueda M, Palazón Bru A, Molina Escribano F, Ponce García I, Artigao Ródenas LM, et al. Evaluation of non-HDL cholesterol as a predictor of non-fatal cardiovascular events in a prospective population cohort. Clin Investig Arterioscler. 2018;30(2):64-71.

8. Zhu L, Lu Z, Zhu L, Ouyang X, Yang Y, He W, et al. Lipoprotein ratios are better than conventional lipid parameters in predicting coronary heart disease in Chinese Han people. Kardiol Pol. 2015;73(10):931-938.

9. Zhang N, Hu X, Zhang Q, Bai P, Cai M, Zeng TS, et al. Non-high-density lipoprotein cholesterol: High-density lipoprotein cholesterol ratio is an independent risk factor for diabetes mellitus: Results from a population-based cohort study. J Diabetes. 2018;10(9):708-714.

10. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction (2018). Circulation. 2018;138(20):e618-e51.

11.Levey AS, Coresh J, Greene T, Marsh J, Stevens LA, Kusek JW, et al. Expressing the Modification of Diet in Renal Disease Study equation for estimating glomerular filtration rate with standardized serum creatinine values. Clin Chem. 2007;53(4):766-772.

12.Cui Y, Choi M. Association between the non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (NHHR) and angina pectoris in US adults: a cross-sectional retrospective study based on NHANES 2009-2018. Lipids Health Dis. 2024;23(1):347.

13.Zvintzou E, Karampela DS, Vakka A, Xepapadaki E, Karavia EA, Hatziri A, et al. High density lipoprotein in atherosclerosis and coronary heart disease: Where do we stand today? Vascul Pharmacol. 2021;141:106928.

14.Barter P, Gotto AM, LaRosa JC, Maroni J, Szarek M, Grundy SM, et al. HDL cholesterol, very low levels of LDL cholesterol, and cardiovascular events. N Engl J Med. 2007;357(13):1301-1310.

15.Zhao X, Sun D, Xu RX, Guo YL, Zhu CG, Wu NQ, et al. Low-density lipoprotein-associated variables and the severity of coronary artery disease: an untreated Chinese cohort study. Biomarkers. 2018;23(7):647-53.

16.Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J. 2020;41(1):111-88.

17.Calling S, Johansson SE, Wolff M, Sundquist J, Sundquist K. Total cholesterol/HDL-C ratio versus non-HDL-C as predictors for ischemic heart disease: a 17-year follow-up study of women in southern Sweden. BMC Cardiovasc Disord. 2021;21(1):163.

18.Tajik B, Voutilainen A, Kauhanen J, Mazidi M, Lip GYH, Tuomainen TP, et al. Lipid profile, lipid ratios, apolipoproteins, and risk of cardiometabolic multimorbidity in men: The Kuopio Ischaemic Heart Disease Risk Factor Study. Lipids. 2022;57(2):141-149.

19.Gori T, Polimeni A, Indolfi C, Räber L, Adriaenssens T, Münzel T. Predictors of stent thrombosis and their implications for clinical practice. Nat Rev Cardiol. 2019;16(4):243-256.

20.Zheng PG, Chen P, Wang LJ, Zhang N. The association of the systemic immune-inflammation index and stent thrombosis in myocardial infarction patients after coronary stent implantation-a retrospectively study. J Thorac Dis. 2023;15(4):1726-1733.

21.Gupta PK, Balachander J. Predictor of in-stent restenosis in patients with drug-eluting stent (PRIDE)- a retrospective cohort study. Clin Investig Arterioscler. 2021;33(4):184-194.

22.de la Torre Hernández JM, Alfonso F, Gimeno F, Diarte JA, Lopez-Palop R, Pérez de Prado A, et al. Thrombosis of second-generation drug-eluting stents in real practice results from the multicenter Spanish registry ESTROFA-2 (Estudio Español Sobre Trombosis de Stents Farmacoactivos de Segunda Generacion-2). JACC Cardiovasc Interv. 2010;3(9):911-919.

23.Yu B, Li M, Yu Z, Zheng T, Feng X, Gao A, et al. The non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (NHHR) as a predictor of all-cause and cardiovascular mortality in US adults with diabetes or prediabetes: NHANES 1999-2018. BMC Med. 2024;22(1):317.

24.Pietilä KO, Harmoinen AP, Jokiniitty J, Pasternack AI. Serum C-reactive protein concentration in acute myocardial infarction and its relationship to mortality during 24 months of follow-up in patients under thrombolytic treatment. Eur Heart J. 1996;17(9):1345-1349.

25.Candemir M, Kiziltunç E, Nurkoç S, Şahinarslan A. Relationship Between Systemic Immune-Inflammation Index (SII) and the Severity of Stable Coronary Artery Disease. Angiology. 2021;72(6):575-581.

26.Bian X, He J, Zhang R, Yuan S, Dou K. The Combined Effect of Systemic Immune-Inflammation Index and Type 2 Diabetes Mellitus on the Prognosis of Patients Undergoing Percutaneous Coronary Intervention: A Large-Scale Cohort Study. J Inflamm Res. 2023;16:6415-6429.

27.Wang H, Liu Z, Shao J, Lin L, Jiang M, Wang L, et al. Immune and Inflammation in Acute Coronary Syndrome: Molecular Mechanisms and Therapeutic Implications. J Immunol Res. 2020;2020:4904217.

28.Akboga MK, Inanc IH, Sabanoglu C, Akdi A, Yakut I, Yuksekkaya B, et al. Systemic Immune-Inflammation Index and C-Reactive Protein/Albumin Ratio Could Predict Acute Stent Thrombosis and High SYNTAX Score in Acute Coronary Syndrome. Angiology. 2023;74(7):693-701.