Original Article

MEDICINE PALLIATIVE CARE

DOI: 10.47582/jompac.1405773

J Med Palliat Care. 2024;5(1):57-64

The assessment of serum uric acid-to-HDL cholesterol ratio as a new predictor of mortality in ST-elevation myocardial infarction: a cross-sectional study

Ömer Genç[1], Abdullah Yıldırım[2], Eyüp Özkan[1]

Department of Cardiology, Başakşehir Çam and Sakura City Hospital, İstanbul, Turkiye
Department of Cardiology, Adana City Hospital, Adana, Turkiye

Cite this article as: Genç Ö, Yıldırım A, Özkan E. The assessment of serum uric acid-to-HDL cholesterol ratio as a new predictor of mortality in ST-elevation myocardial infarction: a cross-sectional study. *J Med Palliat Care*. 2024;5(1):57-64.

Received: 16.12.2023 ◆ **Accepted**: 19.02.2024 ◆ **Published**: 29.02.2024

ABSTRACT

Aims: The objective of this study was to evaluate the predictive efficacy of the Uric Acid-to-High Density Lipoprotein Cholesterol Ratio (UHR) as a novel inflammatory and metabolic marker for mortality in patients with ST-segment Elevation Myocardial Infarction (STEMI).

Methods: This retrospective, single-center, cross-sectional, observational study enrolled 1361 patients diagnosed with STEMI undergoing primary percutaneous coronary intervention (PPCI) from March 2021, to January 2022. The participants were categorized into two groups: those experiencing in-hospital mortality (n=100) and those without in-hospital mortality (n=1265).

Results: In-hospital mortality occurred in 100 patients (7.3%). UHR was notably higher in the mortality group compared to the non-mortality group (23.6±14.9% vs. 15.3±6.9%, p<0.001). Logistic regression analysis identified several independent determinants of in-hospital mortality among STEMI patients, including age (odds ratio [OR]=1.050, p<0.001), the presence of DM (OR=2.077, p=0.016), serum glucose (OR=1.004, p=0.002), hemoglobin (OR=0.855, p=0.020), White blood cell count (OR=1.064, p=0.025), UHR (OR=1.683, p<0.001), and SYNTAX score (OR=1.099, p<0.001). Receiver operating characteristic curve analysis revealed that UHR>13.9% determined in-hospital mortality in STEMI patients who underwent PPCI with 81.1% sensitivity and 49.2% specificity (AUC=0.668, p=0.001)

Conclusion: The UHR exhibited a significant and independent positive correlation with in-hospital mortality among STEMI patients undergoing PPCI.

Keywords: Uric acid, HDL-C, uric acid-to-high density lipoprotein cholesterol ratio, STEMI, SYNTAX score, in-hospital mortality

INTRODUCTION

Despite the advancements in coronary reperfusion strategies, ST-segment elevation myocardial infarction (STEMI) remains the leading cause of death worldwide.[1,2] This critical cardiovascular event necessitates prompt intervention due to its time-sensitive nature. STEMI is characterized by the combination of chest pain or equivalent symptoms and the presence of ST-segment elevation on the electrocardiogram (ECG) or newly confirmed left bundle branch block and elevated cardiac troponin levels.[3] Recognizing the urgency, primary percutaneous coronary intervention (PPCI) has become the main treatment strategy of choice for patients with STEMI.[4,5]

Research has increasingly highlighted the combined impact of elevated uric acid (UA) levels and low high-density lipoprotein cholesterol (HDL-C) on the cardiovascular system, revealing synergistic deleterious effects.[6-9] Recognizing the need for a comprehensive biomarker that captures the interplay between these two factors, the UA-to-HDL-C ratio (UHR) emerged. Developed from two inflammatory parameters, UHR has garnered attention as a novel biomarker of metabolic dysfunction and has been extensively investigated for various comorbid conditions.[10-14] Its unique profile positions UHR as a promising inflammatory and metabolic biomarker, demonstrating potential in predicting cardiac outcomes.[12,15]

Despite extensive investigations into the impact of numerous risk factors on mortality in patients with STEMI, a leading global cause of death, the efficacy of UHR, a novel proinflammatory marker, in predicting mortality within this patient cohort remains an understudied aspect in the existing literature. Thus, the present study aims to address this gap by evaluating the predictive capacity of UHR in assessing mortality risk among individuals with STEMI undergoing PPCI.

Corresponding Author: Ömer GENÇ, dr.genc@hotmail.com

This work is licensed under a Creative Commons Attribution 4.0 International License.

METHODS

Ethics

The study protocol received approval from the Adana City Training and Research Hospital Clinical Researches Ethics Committee (Date: 20.07.2023, Decision No: 2720). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Design and Population Sample

The retrospective, single-center, cross-sectional, observational study included a sample of 1365 patients. These individuals were hospitalized between March 2021 and January 2022 due to STEMI and underwent PPCI. The study was conducted at the Adana City Training and Research Hospital Coronary Intensive Care Unit.

The hospitalization files of the patients were meticulously reviewed in a retrospective fashion. Comprehensive patient information, including demographic and clinical characteristics such as age, gender, hypertension (HTN), diabetes mellitus (DM), current smoking status, history of coronary artery disease (CAD), and history of cerebrovascular event/transient ischemic attack (CVE/TIA), was obtained from the National Chronic Disease and Drug Use Information System (accessible at https://medeczane.sgk.gov.tr/doktor/login.jsp).

The study inclusion criteria comprised individuals aged between 18 and 85 who presented to the hospital with STEMI within 12 hours of the onset of related symptoms and underwent PPCI. Exclusion criteria included chronic obstructive pulmonary disease (COPD), estimated glomerular filtration rate <30 ml/min/1.73 m² or chronic renal failure requiring hemodialysis, liver transplantation or decompensated cirrhosis, history of moderate-to-severe heart valve disease (insufficiency or stenosis), secondary hyperlipidemia, gout, systemic infection, thyroid disease history, active cancer, and current use of immunosuppressive or steroid therapy for any reason (Figure 1).

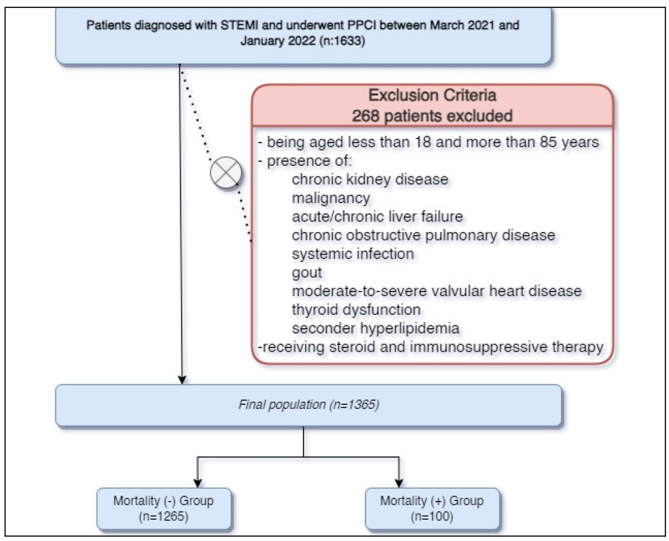

Figure 1. Flowchart of study inclusion and exclusion criteria

Data Collection and Definitions

ST-segment elevation myocardial infarction was diagnosed based on the presence of new ST-elevation at the J point in two consecutive leads on routine ECG, adhering to specific criteria: ≥0.25 mV in men <40 years old, ≥0.2 mV in men ≥40 years old, ≥0.15 mV in leads V2-V3 in women, and/or ≥0.1 mV in other leads. The diagnosis also considered the presence of a new left bundle branch block or prolonged chest pain lasting ≥30 minutes, under current clinical guidelines for positive cardiac markers.[3]

All patients received acetylsalicylic acid along with loading and maintenance doses of purinergic receptor type Y subtype 12 (P2Y12) inhibitors, adhering to the current treatment guidelines.[3] The initiation of beta-blocker and angiotensin-converting enzyme inhibitor (ACEI)/angiotensin receptor blocker (ARB) treatment was contingent upon the absence of contraindications, as determined during coronary angiography conducted during hospitalization.

Routine blood samples were collected from all patients at the emergency triage unit, serving as the initial medical point of contact before PPCI. These samples underwent laboratory analyses using an automated chemistry analyzer (Roche Diagnostic Modular Systems, Tokyo, Japan). The laboratory parameters, including plasma glucose, serum creatinine, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), HDL-C, triglyceride (TG), UA, complete blood count, electrolytes, brain natriuretic peptide (BNP), high-sensitive troponin I (hs-TnI), and high-sensitivity C-reactive protein (Hs-Crp), were obtained for each patient through the hospital information system. The UHR was calculated by dividing UA by HDL-C.

Coronary Angiography, Percutaneous Coronary Intervention, and SYNTAX Score

Angiographic imaging of the left and right coronary systems was conducted in multiple orthogonal projections using the Judkins technique, administered from the femoral route for all patients. Coronary intervention procedures were performed using 6 French (Fr) or 7 Fr guiding catheters. All patients received intracoronary administration of 100 U/kg unfractionated heparin. Decision-making during the procedure, including intracoronary nitrate administration, pre-dilatation before stent implantation, selection of stent size and type, implantation technique, and post-dilatation, was at the discretion of the operator. Epicardial coronary arteries with at least 70% luminal stenosis were deemed indicative of critical coronary lesions. Further analysis was conducted on coronary lesions with at least 50% diameter and a lumen diameter exceeding 1.5 mm. The SYNTAX (Synergy Between Percutaneous Coronary Intervention (PCI) With Taxus and Coronary Artery Bypass Surgery (CABG)) scores were independently calculated by two cardiologists, blinded to patient survival data. The calculations were performed using www.syntaxscore.com (version 2.10) as described previously.[16]

The study group was categorized into two distinct groups: patients who experienced in-hospital mortality (mortality (+) group, n=100) and those who did not experience in-hospital mortality (mortality (-) group, n=1265).

Statistical Analysis

The statistical analyses for the collected data were conducted using the SPSS 22.0 (Statistical Product and Service Solutions for Windows, Version 22.0, IBM Corp., Armonk, NY, U.S., 2013) software package. Homogeneously distributed variables were presented as mean±standard deviation and compared using the independent sample t-test. Non-normally distributed variables were expressed as median and interquartile range (IQR) and compared using the Mann-Whitney U test. Categorical variables were presented as frequency and percentage and compared using Pearson's chi-squared test or Fisher's exact test. The odds ratio (OR) and 95% confidence interval (CI) were calculated for each independent variable.

Multivariable binary logistic regression models were employed to identify independent predictors of in-hospital mortality. Variables exhibiting significant probability (p) values in the univariable analysis underwent further analysis within the scope of the multivariable analysis. Internal correlation analysis (multicollinearity) was conducted by testing whether variance inflation factor <3, condition index <15, and variance proportions <0.6 were achieved using numerical expressions for all parameters.

The results of both univariable and multivariable regression analyses were presented as OR with 95% CI values. Receiver operating characteristics (ROC) curve analysis was utilized to determine the optimal cut-off values of UHR in predicting in-hospital mortality. A significance level of $p<0.05$ was considered statistically significant.

RESULTS

Clinical, Demographic, and Procedural Characteristics of the Study Group

The study included 1365 patients, with a mean age of 60.9±12.2 years, of whom 955 (70.0%) were male. In-hospital mortality occurred in 100 (7.3%) patients. The mortality (+) group was significantly older, with a higher proportion of patients with DM. Various biomarkers and laboratory values at admission, including blood glucose, UA, creatinine, hs-Crp, BNP, hs-TnI levels, as well as platelet and white blood cell counts, were significantly higher in the mortality (+) group. TC, LDL-C, and hemoglobin values were significantly higher in the mortality (-) group. No significant difference was observed between the groups regarding the rate of ST-elevation MI type, implanted stent type, associated lesion, and whether pre-dilatation or post-dilatation was performed. However, the mortality (-) group exhibited significantly higher total stent length and SYNTAX score, while the stent diameter was significantly narrower. Table 1 displays the distribution of demographic, clinical, and laboratory characteristics by the mortality groups, while Table 2 illustrates the distribution of angiographic and procedural characteristics by the mortality groups.

Table 1. Demographic and clinical characteristics

Variables	All (n=1365)	Alive (n=1265)	Deceased (n=100)	p*
Age (years)	60.9±12.2	60.3±11.9	68.9±13.1	<0.001
Male gender (n [%])	955 (70.0)	892 (70.5)	63 (63.0)	0.115
CVE/TIA (n [%])	60 (4.4)	54 (4.3)	6 (6.0)	0.416
Hypertension (n [%])	648 (47.5)	592 (46.8)	56 (56.0)	0.076
DM (n [%])	307 (22.5)	260 (20.6)	47 (47.0)	<0.001
Previus CAD (n [%])	106 (7.8)	101 (8.0)	5 (5.0)	0.283
Current smokers (n [%])	427 (31.3)	387 (30.6)	40 (40.0)	0.052
Laboratory parameters				
Glucose (mg/dl)	138 (110-200)	136 (109-193)	184 (136-324)	<0.001
Sodium (mmol/L)	134.6±6.2	134.7±6.2	134.2±6.2	0.508
Potassium (mEq/L)	4.2±1.3	4.2±1.3	4.4±0.8	0.247
TC (mg/dl)	188.2±48.6	189.6±48.1	167.9±51.5	<0.001
LDL-C (mg/dl)	126.7±42.7	167.9±51.5	127.7±42.1	0.003
HDL-C (mg/dl)	38.4±11.4	38.5±11.2	37.4±14.0	0.470
TG (mg/dl)	126 (79-199)	129 (82-201)	90 (66-138)	0.001
UA (mg/dl)	5.5±1.7	5.4±1.6	7.2±3.0	<0.001
Creatinine (mg/dl)	0.9±0.6	0.9±0.6	1.4±1.0	<0.001
Hemoglobin (g/dl)	13.4±2.0	13.5±1.9	12.3±2.5	<0.001
Platelet count (10^3/ul)	263.1±83.8	261.3±76.8	285.5±143.2	0.009
WBC (10^3/ul)	12.2±4.3	11.9±4.0	15.0±6.7	<0.001
Hs-Crp (mg/L)	0.5 (0.2-1.2)	0.4 (0.2-1.0)	1.1 (0.2-4.6)	0.007
BNP (pg/ml)	95 (32-271)	80 (29-231)	677 (162-2879)	<0.001
hs-TnI (ng/ml)	2.5 (0.4-12.0)	2.4 (0.4-10.9)	5.9 (0.7-28.7)	0.001
UHR (%)	15.7±7.8	15.3±6.9	23.6±14.9	<0.001

Values are n (%), median (interquartile range [IQR]), or mean± standard deviation. P value was calculated using an independent samples t-test or the Mann-Whitney U-test for continuous variables and a chi-squared test or the Fisher's exact test for categorical variables, as appropriate. Abbreviations: BNP, brain natriuretic peptide; CAD, coronary artery disease; CVE/ TIA, cerebrovascular event/ transient ischemic attack; DM, Diabetes mellitus; HDL-C, High-density lipoprotein cholesterol; hs-TnI, high-sensitive troponin I; Hs-Crp, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; UA, uric acid; TG, triglycerides; UHR, uric acid to High-density lipoprotein cholesterol ratio; WBC, White blood cell count. *p value <0.05 was considered significant.

Table 2. Angiographic and procedural features

Variable	All (n=1365)	Alive (n=1265)	Deceased (n = 100)	p*
Culprit vessel, (n [%])				0.190
LMCA	6 (0.4)	5 (0.4)	1 (1.0)	
LAD	648 (47.5)	592 (46.8)	56 (56.0)	
LCx	441 (32.3)	417 (33.0)	24 (24.0)	
RCA	270 (19.8)	251 (19.8)	19 (19.0)	
ST elevation type				0.059
Anterior MI (n [%])	654 (47.9)	597 (47.2)	57 (57.0)	
Inferior MI (n [%])	711 (52.1)	668 (52.8)	43 (43.0)	
Type of stent				0.053
DES (n [%])	852 (62.4)	799 (63.2)	53 (53.0)	
BMS (n [%])	513 (37.6)	466 (36.8)	47 (47.0)	
Stent length (mm)	28.5±16.2	28.2±16.2	32.2±16.1	0.018
Stent diameter (mm)	2.9±0.4	2.9±0.4	2.8±0.3	0.028
Pre-dilatation (n [%])	1206 (88.4)	1112 (87.4)	94 (94.0)	0.067
Post-dilatation (n [%])	1186 (86.9)	1096 (86.6)	90 (90.0)	0.338
SYNTAX Score	14.8±8.2	14.2±7.7	22.9±10.1	<0.001

Values are n (%), mean± standard deviation. P value was calculated using an independent samples t-test and chi-squared test or the Fisher's exact test for categorical variables, as appropriate. Abbreviations: BMS, bare-metal stent; DES, drug-eluting stent; LMCA, left main coronary artery; LAD, left anterior descending artery; LCx, left circumflex artery; MI, Myocardial Infarction; RCA, right coronary artery; SYNTAX, Synergy Between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery; *p<0.05 was considered significant.

The Relationship between UHR and In-hospital Mortality

The serum UA level was significantly higher in the mortality (+) group compared to the mortality (-) group (5.4±1.6 vs. 7.2±3.0, p<0.001). Conversely, there was no significant difference between the two groups in terms of HDL-C level (38.5±11.2 vs. 37.4±14.0, p=0.470). However, UHR exhibited a significant increase in the mortality (+) group compared to the mortality (-) group (23.6±14.9% vs. 15.3±6.9%, p<0.001), indicating a substantial positive correlation between UHR and mortality among STEMI patients.

Regression and Sensitivity Analyses

Logistic regression analysis identified several independent determinants of in-hospital mortality in STEMI patients who underwent PPCI. The determinants included age (OR=1.050, 95% CI 1.027-1.173, p<0.001), the presence of DM (OR=2.077, 95% CI 1.147-3.758, p=0.016), serum glucose (OR=1.004, 95% CI 1.001-1.006, p=0.002), hemoglobin (OR=0.855, 95% CI 0.749-0.975, p=0.020), WBC (OR=1.064, 95% CI 1.008-1.123, p=0.025), UHR (OR=1.683, 95% CI 1.261-2.246, p<0.001), and SYNTAX score (OR=1.099, 95% CI 1.069-1.130, p<0.001) (Table 3). Additionally, based on the Youden index, ROC analysis revealed that a UHR>13.9% determined in-hospital mortality in STEMI patients who underwent PPCI with 81.1% sensitivity and 49.2% specificity (AUC=0.668, 95% CI 0.566-0.771, p=0.001) (Figure 2).

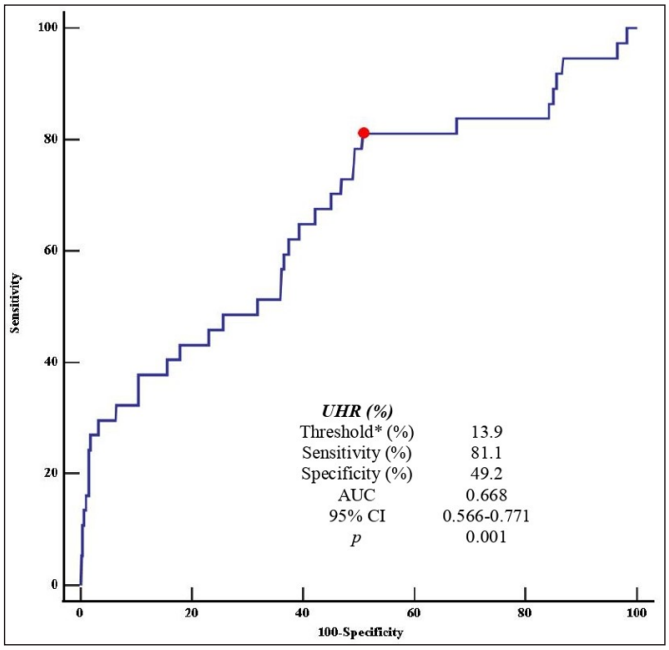

Figure 2. Receiver operating characteristic curve analysis of UHR for determining in-hospital mortality. *The cut-off was determined by the Youden Index. Abbreviations: AUC, Area under the curve; CI, Confidence Interval; UHR: uric acid-to-High-density lipoprotein cholesterol ratio.

Table 3. Univariable and multivariable analyses of predictors of in-hospital mortality on logistic regression analysis

Variable	Univariable			Multivariable+		
	OR	95% CI	p*	OR	95% CI	p*
Age (years)	1.058	1.040-1.076	<0.001	1.050	1.027-1.173	<0.001
DM (n [%])	3.428	2.262-5.195	<0.001	2.077	1.1.47-3.758	0.016
Glucose (mg/dl)	1.005	1.003-1.006	<0.001	1.004	1.001-1.006	0.002
TC (mg/dl)	0.992	0.988-0.996	<0.001			
TG (mg/dl)	0.998	0.996-1.000	0.088			
Creatinine (mg/dl)	1.667	1.367-2.034	<0.001			
Hemoglobin (g/dl)	0.719	0.649-0.796	<0.001	0.855	0.749-0.975	0.020
Platelet count (10^3/ul)	1.002	1.000-1.004	0.020			
WBC (10^3/ul)	1.122	1.080-1.166	<0.001	1.064	1.008-1.123	0.025
Hs-Crp (mg/L)	1.004	0.995-1.012	0.388			
BNP (pg/ml)	0.999	0.998-1.001	0.004			
hs-TnI (ng/ml)	1.015	1.008-1.023	<0.001			
UHR (%)	1.697	1.389-2.073	<0.001	1.683	1.261-2.246	<0.001
Stent diameter (mm)	0.542	0.313-0.939	0.029			
Stent length (mm)	1.013	1.002-1.024	0.020			
SYNTAX score	1.116	1.091-1.143	<0.001	1.099	1.069-1.130	<0.001

Abbreviations: BNP, brain natriuretic peptide; DM, Diabetes mellitus; Hs-Crp, high-sensitivity C- reactive protein; hs-TnI, high-sensitive troponin I; SYNTAX, Synergy Between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery; TC, total cholesterol; TG, triglycerides; UHR, uric acid to High-density lipoprotein cholesterol ratio; WBC, White blood cell count. +Model performance parameters: Hosmer-Lemeshow test p=0.532; Omnibus tests of model coefficients p<0.001; Nagelkareke R square=0.364, -2 log-likelihood=479.9; *p value<0.05 was considered significant.

DISCUSSION

The primary findings of this study can be summarized as follows: 1) In-hospital mortality occurred in 100 (7.3%) patients; 2) The mortality (+) group exhibited a significantly higher mean age and a greater prevalence of DM; 3) Age, the presence of DM, serum glucose, hemoglobin, WBC, UHR, and SYNTAX score emerged as independent predictors of mortality in patients with STEMI.

ST-segment elevation myocardial infarction stands as a predominant global cause of mortality. Numerous studies conducted across European countries have scrutinized in-hospital mortality rates among patients with STEMI. Notably, the Euro Heart Survey of Acute Coronary Syndromes and GRACE (Global Registry of Acute Coronary Events) registry[17] reported a 7.0% mortality rate, the Swiss national registry AMIS Plus (Acute Myocardial Infarction and Unstable Angina in Switzerland)[18] documented a 7.6% mortality rate, the RO-STEMI registry (ROmanian ST-Elevation Myocardial Infarction registry)[19] reported a 7.1% mortality rate, and MINAP (Myocardial Infarction National Audit Project)[20] recorded a 10.6% mortality rate. Consistent with these findings, the in-hospital mortality rate among STEMI patients in this study was determined to be 7.3%. A recent meta-analysis highlighted higher in-hospital mortality rates for STEMI in low- and middle-income countries compared to high-income countries.[21] Consequently, while the in-hospital mortality data presented in this study, which focuses on STEMI patients, may not cover the entire country, it might offer insights into the observed trends in European countries, particularly those with geographical and political affiliations to Turkiye.

Increased UA levels have been proposed as a risk factor for the development of DM and hypertension, while also serving as an independent biomarker for vascular complications and mortality.[22,23] In our study, the mortality (+) group showed significantly lower levels of TC, LDL-C, and TG, along with higher UA levels, compared to the mortality (-) group. Additionally, although not statistically significant, the mortality (+) group also exhibited lower levels of HDL-C compared to the mortality (-) group. Consistent with our study findings, elevated LDL-C and TG levels, along with low HDL-C levels, are widely recognized as risk factors for acute myocardial infarction (AMI). However, intriguingly, there exists a concept known as the 'lipid paradox' in AMI patients. Some studies suggest that low LDL-C and TG levels are associated with significantly higher in-hospital mortality rates in AMI patients.[24,25] Conversely, another study has demonstrated that low HDL-C levels are linked to significantly higher mortality rates among STEMI patients.[26] The findings from our study align with and contribute to the existing body of evidence surrounding this lipid paradox.

The combination of UA and HDL-C, represented by the UHR, has been proposed as a novel and more sensitive marker for various metabolic and inflammatory states. Previous studies have indicated that reduced levels of serum HDL-C and hyperuricemia might collectively worsen cardiovascular health by fostering insulin resistance and causing oxidative harm to endothelial cells.[27] For instance, Hu et al.[28] found that elevated serum UA levels alter how HDL-C affects carotid atherosclerosis. While a correlation between hyperuricemia and reduced HDL-C levels exists in cardiometabolic conditions, the impact of the interplay between UA and HDL-C on STEMI patients remains insufficiently explored. On the other hand, our study's results highlight the UHR as a reliable predictor of mortality in STEMI patients. In addition, one study reported elevated UHR values in patients with coronary fistula compared to control subjects.[29] Another study found that high UHR levels were associated with poor collateral circulation in patients with chronic total occlusion.[30] Similarly, UHR levels were reported to increase significantly in patients with hemodynamically significant coronary lesions.[31] Beyond coronary conditions, UHR has demonstrated associations with various non-coronary comorbidities. Studies have found significant correlations between UHR and adverse outcomes, including waist circumference, non-alcoholic fatty liver, body mass index, TG, fasting glucose, and glycated hemoglobin levels.[10,12] Taken together, the ease of obtaining a parameter such as UHR and its potential for predicting in-hospital mortality in individuals undergoing PPCI for STEMI highlight its clinical utility and the importance of its inclusion in risk assessment protocols.

Despite its initial development as a marker for the complexity of coronary lesions, the SYNTAX score has been extensively investigated for its implications for various complications and morbidities among patients with STEMI.[32-34] In alignment with existing literature, our study's findings affirm that a high SYNTAX score stands as an independent risk factor for in-hospital cardiovascular mortality in AMI patients.[35,36] Furthermore, parameters such as advanced age, high BNP levels, and an increased length of the implanted stent were also identified as contributors to poor outcomes, consistent with previous literature.[37-42] Moreover, anemia stands out as a prevalent and well-established factor contributing to short-term or in-hospital mortality among STEMI patients.[43] This association likely mirrors the link between anemia, comorbidities, and major adverse cardiovascular events. Similarly, our study reveals that higher hemoglobin levels are correlated with reduced in-hospital mortality. Additionally, impaired glucose regulation and uncontrolled DM emerge as significant predictors of mortality in STEMI cases.[44] Plausible mechanisms contributing to this observation among individuals with DM include advanced age, often accompanied by conditions such as dyslipidemia and elevated body mass index, endothelial dysfunction, and ultimately an increased prevalence of atherosclerosis. Our findings further emphasize an independent association between DM, elevated serum glucose levels, and in-hospital mortality in STEMI patients.

Limitations

Nevertheless, it's essential to acknowledge the primary limitation of our study, namely its retrospective, single-center design, which may introduce patient selection bias. Additionally, the determination of all-cause in-hospital mortality as the study's outcome poses another limitation. While our study successfully illustrates the association between UHR and short-term mortality in STEMI patients, we acknowledge the absence of comparative analysis between UHR and other inflammatory markers, as well as the lack of medium-to-long-term outcomes assessment in STEMI. Given the diverse causes of in-hospital mortality in the STEMI patient group, further investigations focusing on each etiological cause are warranted.

CONCLUSION

The UHR exhibited a significant positive correlation with in-hospital mortality in patients with STEMI undergoing PPCI. Serving as a novel biomarker, UHR, comprising serum UA and HDL-C, both easily and rapidly measurable through basic biochemical tests, holds promise for contributing to the prognostic classification of this patient group from the initial medical contact onward.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of Adana City Training and Research Hospital Clinical Researches Ethics Committee (Date: 20.07.2023, Decision No: 2720).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure
The authors declared that this study has received no financial support.

Author Contributions
All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Mathers CD, Boerma T, Ma Fat D. Global and regional causes of death. *Br Med Bull.* 2009;92(1):7-32.
2. Anderson JL, Morrow DA. Acute myocardial infarction. *NEJM.* 2017;376(21):2053-2064.
3. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J.* 2018;39(2):119-177.
4. Collet JP, Thiele H, Barbato E, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J.* 2021;42(14):1289-1367.
5. Zeymer U, Ludman P, Danchin N, et al. Reperfusion therapies and in-hospital outcomes for ST-elevation myocardial infarction in Europe: the ACVC-EAPCI EORP STEMI Registry of the European Society of Cardiology. *Eur Heart J.* 2021;42(44):4536-4549.
6. Chuang SY, Chen JH, Yeh WT, Wu CC, Pan WH. Hyperuricemia and increased risk of ischemic heart disease in a large Chinese cohort. *Int J Cardiol.* 2012;154(3):316-321.
7. Li L, Zhao M, Wang C, et al. Early onset of hyperuricemia is associated with increased cardiovascular disease and mortality risk. *Clin Res Cardiol.* 2021;110(7):1096-1105.
8. Goldbourt U, Yaari S, Medalie JH. Isolated low HDL cholesterol as a risk factor for coronary heart disease mortality: a 21-year follow-up of 8000 men. *Arterioscler Thromb.* 1997;17(1):107-113.
9. Yu S, Guo X, Li GX, Yang H, Zheng L, Sun Y. Lower or higher HDL-C levels are associated with cardiovascular events in the general population in rural China. *Lipids Health Dis.* 2020;19(1):1-6.
10. Kosekli MA, Kurtkulagii O, Kahveci G, et al. The association between serum uric acid to high density lipoprotein-cholesterol ratio and non-alcoholic fatty liver disease: the abund study. *Rev Assoc Med Bras.* 2021;67:549-554.
11. Zhou Y, Wei F, Fan Y. High serum uric acid and risk of nonalcoholic fatty liver disease: a systematic review and meta-analysis. *Clin Biochem.* 2016;49(7-8):636-642.
12. Kocak MZ, Aktas G, Erkus E, Sincer I, Atak B, Duman T. Serum uric acid to HDL-cholesterol ratio is a strong predictor of metabolic syndrome in type 2 diabetes mellitus. *Rev Assoc Med Bras.* 2019;65(1):9-15.
13. Zhang YN, Wang QQ, Chen YS, Shen C, Xu CF. Association between serum uric acid to hdl-cholesterol ratio and nonalcoholic fatty liver disease in lean chinese adults. *Int J Endocrinol.* 2020;2020:5953461.
14. Kurtkulagi O, Tel BMA, Kahveci G, et al. Hashimoto's thyroiditis is associated with elevated serum uric acid to high density lipoprotein-cholesterol ratio. *Rom J Intern Med.* 2021;59(4):403-408.
15. Aktas G, Kocak MZ, Bilgin S, Atak BM, Duman TT, Kurtkulagi O. Uric acid to HDL cholesterol ratio is a strong predictor of diabetic control in men with type 2 diabetes mellitus. *Aging Male.* 2020;23(5):1098-1102.
16. Sianos G, Morel MA, Kappetein AP, et al. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. *EuroIntervention.* 2005;1(2):219-227.
17. Granger CB, Goldberg RJ, Dabbous O, et al. Predictors of hospital mortality in the global registry of acute coronary events. *Arch Intern Med.* 2003;163(19):2345-2353.
18. Stolt Steiger V, Goy JJ, Stauffer JC, et al. Significant decrease in in-hospital mortality and major adverse cardiac events in Swiss STEMI patients between 2000 and December 2007. *Swiss Med Wkly.* 2009;139(31-32):453-457.
19. Cretu DE, Udroiu CA, Stoicescu CI, Tatu-Chitoiu G, Vinereanu D. Predictors of in-hospital mortality of ST-segment elevation myocardial infarction patients undergoing interventional treatment. An analysis of data from the RO-STEMI registry. *Maedica.* 2015;10(4):295-303.
20. Gale CP, Manda S, Batin PD, Weston CF, Birkhead JS, Hall AS. Predictors of in-hospital mortality for patients admitted with ST-elevation myocardial infarction: a real-world study using the Myocardial Infarction National Audit Project (MINAP) database. *Heart.* 2008;94(11):1407-1412.
21. Alves L, Ziegelmann PK, Ribeiro V, Polanczyk C. Hospital mortality from myocardial infarction in Latin America and the Caribbean: systematic review and meta-analysis. *Arq Bras Cardiol.* 2022;119(6):970-978.
22. Xu Y, Zhu J, Gao L, et al. Hyperuricemia as an independent predictor of vascular complications and mortality in type 2 diabetes patients: a meta-analysis. *PloS One.* 2013;8(10):e78206.
23. Mortada I. Hyperuricemia, type 2 diabetes mellitus, and hypertension: an emerging association. *Curr Hypertens Rep.* 2017;19(9):69.
24. Cheng KH, Chu CS, Lin TH, Lee KT, Sheu SH, Lai WT. Lipid paradox in acute myocardial infarction—the association with 30-day in-hospital mortality. *Crit Care Med.* 2015;43(6):1255-1264.
25. Reddy VS, Bui QT, Jacobs JR, Begelman SM, Miller DP, French WJ. Relationship between serum low-density lipoprotein cholesterol and in-hospital mortality following acute myocardial infarction (the lipid paradox). *Am J Cardiol.* 2015;115(5):557-562.
26. Ji MS, Jeong MH, Ahn YK, et al. Impact of low level of high-density lipoprotein-cholesterol sampled in overnight fasting state on the clinical outcomes in patients with acute myocardial infarction (difference between ST-segment and non-ST-segment-elevation myocardial infarction). *J Cardiol.* 2015;65(1):63-70.
27. Yang Y, Zhang J, Jia L, Su J, Ma M, Lin X. The interaction between uric acid and high-density lipoprotein cholesterol on the prognosis of patients with acute myocardial infarction. *Front Cardiovasc Med.* 2023;10:1226108.
28. Hu X, Liu J, Li W, et al. Elevated serum uric acid was associated with pre-inflammatory state and impacted the role of HDL-C on carotid atherosclerosis. *Nutr Metab Cardiovasc Dis.* 2022;32(7):1661-1669.
29. Mansiroglu AK, Cekici Y, Sincer I, Gunes Y. Serum uric acid and uric acid to HDL-cholesterol ratio in coronary artery fistulas. *Ann Med Res.* 2019;26(12):2771-2775.
30. Aydın C, Emlek N. The relationship between uric acid to high-density lipoprotein cholesterol ratio and collateral index in patients with chronic total occlusion. *Kardiologiia.* 2021;61(9):61-65.
31. Li F, Zhao D, Li Q, Lin X, Sun H, Fan Q. Uric acid to high-density lipoprotein cholesterol ratio is a novel marker to predict functionally significant coronary artery stenosis. *J Interv Cardiol.* 2022;2022:9057832.
32. Rencuzogullari I, Çağdaş M, Karakoyun S, et al. Association of Syntax Score II with contrast-induced nephropathy and hemodialysis requirement in patients with ST segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. *Korean Circ J.* 2018;48(1):59-70.

33. Alıcı G, Barman HA, Atıcı A, Tuğrul S, Genç Ö, Şahin İ. The impact of lesion complexity and the CHA 2 DS 2-VASc score on spontaneous reperfusion in patients with ST-segment elevation myocardial infarction. *Int J Clin Pract.* 2022;2022:8066780.
34. Cirakoglu OF, Aslan AO, Akyuz AR, et al. The value of syntax score to predict new-onset atrial fibrillation in patients with acute coronary syndrome. *Ann Noninvasive Electrocardiol.* 2019;24(4):e12622.
35. Kul S, Akgul O, Uyarel H, et al. High SYNTAX score predicts worse in-hospital clinical outcomes in patients undergoing primary angioplasty for acute myocardial infarction. *Coron Artery Dis.* 2012;23(8):542-548.
36. Alıcı G, Genç Ö, Urgun ÖD, et al. The value of PRECISE-DAPT score and lesion complexity for predicting all-cause mortality in patients with NSTEMI. *Egypt Heart J.* 2023;75(1):1-7.
37. Ali M, Lange S, Wittlinger T, Lehnert G, Rigopoulos A, Noutsias M. In-hospital mortality after acute STEMI in patients undergoing primary PCI. *Herz.* 2018;43(8):741-745.
38. Sanguanwong S, Srimahachota S, Tungsubutra W, Srichaiveth B, Kiatchoosakun S. Predictors of in-hospital mortality in Thai STEMI patients: results from TACSR. *J Med Assoc Thai.* 2007;90(Suppl 1):91-97.
39. Almeida I, Miranda H, Santos H, et al. Prognostic value of BNP in STEMI patients. *Eur Heart J Acute Cardiovasc Care.* 2021;10(Supplement_1):zuab020-093.
40. Akgul O, Uyarel H, Pusuroglu H, et al. High BNP level as risk factor for acute kidney injury and predictor of all-cause mortality in STEMI patients. *Herz.* 2014;39(4):507-514.
41. Caputo RP, Goel A, Pencina M, et al. Impact of drug eluting stent length on outcomes of percutaneous coronary intervention (from the EVENT registry). *Am J Cardiol.* 2012;110(3):350-355.
42. Konishi H, Miyauchi K, Dohi T, et al. Impact of stent length on clinical outcomes of first-generation and new-generation drug-eluting stents. *Cardiovasc Interv Ther.* 2016;31(2):114-121.
43. Moghaddam N, Wong GC, Cairns JA, et al. Association of anemia with outcomes among ST-segment–elevation myocardial infarction patients receiving primary percutaneous coronary intervention. *Circ Cardiovasc Interv.* 2018;11(12):e007175.
44. Rousan TA, Pappy RM, Chen AY, Roe MT, Saucedo JF. Impact of diabetes mellitus on clinical characteristics, management, and in-hospital outcomes in patients with acute myocardial infarction (from the NCDR). *Am J Cardiol.* 2014;114(8):1136-1144.