ORIGINAL ARTICLE

Comparison of RCHA2DS2-VASc score and CHA2DS2-VASc score prediction of no-reflow phenomenon in patients with ST-segment elevation myocardial infarction

ST segment yükselmeli miyokart enfarktüsü olan hastalarda "no reflow" öngörmede RCHA2DS2-VASc skoru ve CHA2DS2-VASc skorlarının karşılaştırılması

Çağrı Zorlu, M.D., Cemal Köseoğlu, M.D.

Department of Cardiology, Tokat State Hospital, Tokat, Turkey

ABSTRACT

Objective: No-reflow is a phenomenon that can arise due to factors such as distal embolization, microvascular occlusion, or prolonged myocardial ischemia and damage. It occurs in about 5% to 10% of patients after primary percutaneous coronary intervention. The CHA2DS2-VASc score can be easily calculated in daily practice and the components of this score are similar to common risk factors for no-reflow. Chronic renal disease generates a hypercoagulable state, which is associated with increased risk of no-reflow in cases of ST-segment elevation myocardial infarction (STEMI). A modified CHA2DS2-VASc score has been developed to include patients with renal dysfunction. The aim of this study was to investigate the prognostic significance of this scoring system, the RCHA2DS2-VASc score, in patients with no-reflow.

Methods: A total of 75 patients with no-reflow and 1138 patients without no-reflow after STEMI were retrospectively enrolled in this study. The CHA2DS2-VASc and RCHA2DS2-VASc scores of the two groups were compared.

Results: The median CHA2DS2-VASc score and the median RCHA2DS2-VASc score were significantly higher in the no-reflow group (p<.001, for both). There was a statistically significant difference between the groups in all of the components of the CHA2DS2-VASc score. An RCHA2DS2-VASc score of ≥2 was a predictor of no-reflow with a sensitivity of 83% and specificity of 62%.

Conclusion: The RCHA2DS2-VASc score is a simple, inexpensive, and easily accessible score to predict no-reflow.

ÖZET

Amaç: "No reflow" distal embolizasyon, mikrovasküler oklüzyon, uzamış miyokardiyal iskemi ve hasardan oluşan bir fenomendir. Primer perkütan koroner girişim sonrası hastaların yaklaşık %5 ila %10'u oranında ortaya çıkar. CHA2DS2-VASc skoru günlük uygulamada kolayca uygulanabilir ve bu skorun komponentleri ile no reflow'un risk faktörleri benzerdir. Kronik böbrek hastalığı hiperkoagülopati riskini getirir ve ST segment yükselmeli miyokart enfarktüsünde (STYME) artmış no reflow riski ile ilişkilidir. Böbrek disfonksiyonu olan hastalarda modifiye CHA2DS2-VASc skorlaması geliştirilmiştir. Bu çalışmanın amacı, no-reflow gelişen hastalarda CHA2DS2-VASc skorlama sisteminin prognostik önemini araştırmaktır.

Yöntemler: Bu çalışmaya, geriye dönük olarak, STYME olup no reflow gelişen 75 hasta ile no reflow gelişmeyen 1138 hasta alındı. İki grup arasında CHA2DS2-VASc skoru ve RCHA2DS2-VASc skoru karşılaştırıldı.

Bulgular: Medyan CHA2DS2-VASc skoru ve medyan RCHA2DS2-VASc skoru no reflow gelişen gruptan istatistiksel olarak anlamlı olup yüksek bulundu (her ikisi için de, p<.001). Ayrıca, CHA2DS2-VASc skorunun tüm bileşenleri iki grup arasında istatistiksel olarak anlamlı derecede farklıydı. RCHA2DS2-Vasc ≥2 puanı, %83 sensivite ve %62 spesifite ile no reflow öngördürücüsü olarak kullanılabilir.

Sonuç: RCHA2DS2-VASc skoru no reflow gelişmini öngörmede basit, ucuz ve kolay erişilebilir bir skordur.

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Correspondence: Dr. Cemal Köseoğlu. Tokat Devlet Hastanesi, Kardiyoloji Kliniği, Tokat, Turkey.
Tel: +90 356 - 214 54 00 e-mail: drcemalkoseoglu@hotmail.com

No-reflow is a phenomenon that may occur due to complex and multifactorial mechanisms, such as distal embolization of thrombotic materials, microvascular occlusion and cellular edema, and prolonged myocardial ischemia and damage.[1] This condition contributes to complications and short- and long-term morbidity and mortality in patients with acute ST-segment elevation myocardial infarction (STEMI).[2] It occurs in about 5% to 10% of patients after primary percutaneous coronary intervention (PCI).[3] Currently, there is no generally accepted risk assessment scale for the prediction of this complication.

The CHA2DS2-VASc (congestive heart failure, hypertension [HT], age ≥75 years [doubled], diabetes mellitus [DM], prior stroke or transient ischemic attack [doubled], vascular disease, age 65–74 years, and sex category [female]) score can be calculated easily and is used in daily practice to predict thromboembolic risk in atrial fibrillation (AF) patients. The components of this score are associated with atherosclerosis, vascular spasm, and microvascular dysfunction, which are similar to common risk factors of no-reflow.[4] Moreover, it has been shown to be a predictor of adverse outcomes after acute coronary syndromes.[5] Recently, Ipek et al.[6] reported that the CHA2DS2-VASc score was a predictor of no-reflow in patients with STEMI.

Impaired renal function has been shown to be a predictor of stroke and systemic embolism in AF patients without valvular disease.[7] In addition, a relationship between renal dysfunction and no-reflow has been demonstrated in the literature.[8,9] Barra et al.[10] found that a modified CHA2DS2-VASc score that included an element evaluating renal sufficiency, the RCHA2DS2-VASc score, was a predictor of ischemic stroke and myocardial infarction.

The aim of this study was to compare the ability of the CHA2DS2-VASc score and the modified score that includes renal function to predict the no-reflow phenomenon in patients with STEMI with the goal of helping clinicians identify those at risk for this condition.

METHODS

Study population

This retrospective, analytical cross-sectional study examined the data of 1388 consecutive patients from December 2017 to December 2019 who were admitted to a single cardiovascular center with a diagnosis of acute STEMI and underwent primary PCI. Acute STEMI was diagnosed when patients had symptoms of acute myocardial infarction and new ST-segment elevation in at least 2 contiguous leads of ≥0.2 mV in men or ≥0.15 mV in women in leads V2 to V3 and/or of ≥1 mm (0.1 mV) in other contiguous leads, or new left bundle branch block, later confirmed by increases in creatine kinase and CK-myocardial band isoenzyme and/or troponin.[11] Patients with a venous graft as the infarct-related artery (n=22), no intervention due to normal coronary anatomy (n=51), noncritical stenosis (n=34), inappropriate coronary anatomy for stenting or emergency surgery (n=49), and only percutaneous transluminal coronary angioplasty (n=19) were excluded. Ultimately, 1213 patients were enrolled in the study (Fig. 1). Bedside 12-lead electrocardi-

Abbreviations:

AF	*Atrial fibrillation*
CAD	*Coronary artery disease*
CHA2DS2-VASc	*Congestive heart failure, hypertension, age ≥75 years (doubled), diabetes mellitus, prior stroke or transient ischemic attack (doubled), and vascular disease, age 65-74 years, and sex category (female)*
CI	*Confidence interval*
CKD-EPI	*Chronic Kidney Disease Epidemiology*
GFR	*Glomerular filtration rate*
OR	*Odds ratio*
PCI	*Percutaneous coronary intervention*
RCHA2DS2-VASc	*Renal failure addition to CHA2DS2-VASc*
ROC	*Receiver operating characteristic*
STEMI	*ST-segment elevation myocardial infarction*
TIMI	*Thrombolysis in Myocardial Infarction*

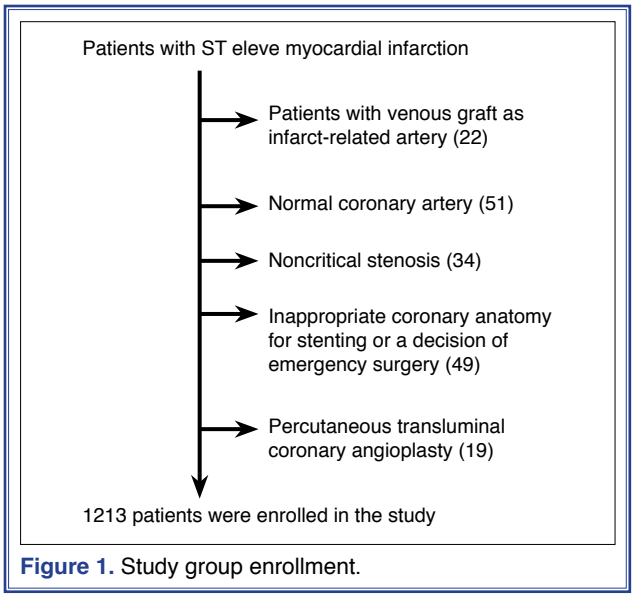

Figure 1. Study group enrollment.

ography and routine blood tests were performed, as well as bedside echocardiography. The study protocol was approved by the local ethics committee (date: 26/12/2019, no: 19-KAEK-261).

Coronary angiography and primary PCI

All of the patients underwent coronary angiography using a standard technique. Prior to the procedure, 300 mg of acetylsalicylic acid was administered, followed by 180 mg of ticagrelor. Patients not treated with enoxaparin were given 1 mg/kg intravenous heparin immediately after the decision to perform a coronary intervention. For those with an initial enoxaparin dose of 1 mg/kg, a 0.3 mg/kg booster of enoxaparin was administered intravenously within 8 hours of the first dose. Stenting of the infarct-related artery was successfully completed immediately after the coronary angiography in all cases. Thrombus aspiration was applied in patients with a high thrombus burden at the operator's discretion.

A tirofiban infusion (0.15 mg/kg/min) was given to selected patients with no contraindications or tendency for bleeding. The Thrombolysis in Myocardial Infarction (TIMI) flow grade was evaluated by 2 blinded cardiologists. The cine image film rate was 30 frames per second. Analysis of the cineangiograms was performed using an Axiom system (Siemens AG, Munich, Germany).

Definitions

The study population was divided into 2 groups according to the final angiographic TIMI flow rate after the primary PCI. The control group comprised those with a TIMI flow rate of >2 and the no-reflow group was defined by a TIMI flow rate of ≤2 despite mechanical reopening of the infarct-related artery in patients without dissection of the coronary artery.[12]

The CHA2DS2-VASc score was calculated for each patient using the data available in the patient files recorded during hospitalization. Patients were given 1 point for congestive heart failure (signs/symptoms of heart failure and/or ejection fraction of ≤40%), hypertension (taking anti-hypertensive medicine or systolic/diastolic blood pressure of ≥140/90 mmHg), diabetes mellitus (defined as a fasting blood glucose level of >126 mg/dL, blood glucose level of ≥200 mg/dL, or use of anti-diabetic drugs), a history of vascular disease (peripheral artery disease, defined as stenosis of at least 50% in non-coronary artery circulation), age 65–74 years, female sex, and 2 points for an age of 75 years or older and a previous stroke or transient ischemic attack.[13] Data on race were also collected to determine the estimated glomerular filtration rate (GFR) using the Chronic Kidney Disease Epidemiology (CKD-EPI) creatinine equation.[14] One additional point was added for renal failure, which was defined as a calculated GFR of <60 mL/min/1.73m^2 using the CDK-EPI equation:

Gender	Creatinine concentration	Formula
Woman	≤0.7	$GFR = 114 \times (Cr/0.7)^{-0.329} \times (0.993)^{age}$
	>0.7	$GFR = 114 \times (Cr/0.7)^{-1.209} \times (0.993)^{age}$
Man	≤0.9	$GFR = 141 \times (Cr/0.9)^{-0.411} \times (0.993)^{age}$
	>0.9	$GFR = 141 \times (Cr/0.9)^{-0.209} \times (0.993)^{age}$

Statistical analysis

Quantitative variables were described using the median and interquartile range, and qualitative variables were defined by frequency and percentage. The Kolmogorov-Smirnov test was used to evaluate the normality of the distribution of continuous variables. Categorical and continuous variables were analyzed using a chi-squared test or an independent sample t-test, respectively. Multivariate logistic regression analysis was performed to determine independent predictors. Variables with a significant p value that could be a predictor of no-reflow were entered into multivariate analysis.

The results of univariate and multivariate regression analyses were presented as the odds ratio (OR) with a 95% confidence interval (CI). Nonparametric receiver operating characteristic (ROC) curve analysis was used to calculate the cutoff value of the CHA2DS2-VASc and RCHA2DS2-VASc scores with the highest sensitivity and specificity. A p value of <0.5 was accepted as statistically significant. The statistical analyses were performed using PASW Statistics for Windows, Version 18.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

A total of 1213 patients (219 female [18.6%], median age: 60.3 years [16]) were included in this study. The control group comprised 1138 patients and 75 patients were designated as the no-reflow group. Demographic, clinical and angiographic data of the

Table 1. Demographic, clinical and angiographic features of the patients

Variables	Control (n=1138)	No-reflow (n=75)	*p* value
Age, years, median, IQR	60 [16]	65 [16]	<0.001
Female gender, n (%)	209 (18.3)	21 (28)	0.018
Smoker, n (%)	366 (31.1)	23 (29.5)	0.891
Diabetes mellitus, n (%)	365 (32)	32 (42.6)	0.023
Hypertension, n (%)	390 (34.2)	31 (41.3)	0.014
Systolic BP, mmHg, median, IQR	133.4 [24.2]	125.3 [30.4]	0.178
Diastolic BP, mmHg, median, IQR	81.8 [13.1]	74.8 [17]	0.058
Hyperlipidemia, n (%)	406 (36.6)	32 (43.2)	0.017
History of stroke/TIA, n (%)	21 (1.8)	6 (8)	<0.001
Vascular disease, n (%)	159 (13.8)	19 (25.3)	0.001
Previous MI, n (%)	137 (12.8)	11 (16.1)	0.432
Peripheral arterial disease, n (%)	24 (2.2)	9 (12.7)	<0.001
Previous bypass surgery, n (%)	23 (2.3)	5 (6.5)	0.005
LV ejection fraction, %, n (%)	38.1 (7.8)	33.9 (8.2)	<0.001
CHA2DS2-VASc score, median (IQR)	1 [2]	2 [3]	<0.001
RCHA2DS2-VASc score, median (IQR)	1 [2]	2 [4]	<0.001
Chronic renal failure, n (%)	102 (9.2)	17 (22.7)	<0.001
Serum creatinine, mg/dL, median (IQR)	1.1 [0.29]	1.18 [0.64]	<0.001
GFR, mL/min/1.73 m^2, median (IQR)	64.9 [14.6]	43.2 [6.5]	<0.001
GFR, (≥90 mL/min), n (%)	689 (60.5)	34 (45.3)	
GFR, (60–89 mL/min), n (%)	347 (30.4)	24 (32)	
GFR, (30–59 mL/min), n (%)	98 (8.6)	13 (17.3)	
GFR, (15–29 mL/min), n (%)	3 (0.2)	4 (5.3)	
GFR, (≤15 mL/min), n (%)	0	0	
MI type, n (%)			0.001
Anterior	480 (42.4)	45 (58.3)	
Nonanterior	599 (53.4)	30 (37.5)	
Initial TIMI flow rate, n (%)			<0.001
TIMI=0	766 (77.6)	71 (89.2)	
TIMI ≥1 (1,2,3)	372 (42.6)	4 (3.8)	
Anemia, n (%)	148 (13)	19 (25.3)	0.032
Drug-eluting stent, n (%)	812 (71.4)	54 (72)	0.714
Stenting without PTCA, n (%)	461 (40.5)	30 (40)	0.78
Stent length, mm, median (IQR)	22 [11]	26 [14]	<0.001
Stent diameter, mm, median (IQR)	3 [0.75]	2.75 [0.50]	<0.001
Tirofiban infusion, n (%)	536 (48.4)	51 (67.3)	<0.001
Thrombus aspiration, n (%)	61 (5.9)	7 (9.2)	0.187
Time to PCI, min, median (IQR)	158.8 [128.2]	179.6 [122.1]	0.175
In-hospital mortality, n (%)	33 (3.2)	14 (18.8)	<0.001

BP: Blood pressure; CHA2DS2-VASc: Congestive heart failure, hypertension, age ≥75 years (doubled), diabetes mellitus, prior stroke or transient ischemic attack (doubled), and vascular disease, age 65-74 years, and sex category (female); GFR: Glomerular filtration rate; IQR: Interquartile range; LV: Left ventricular; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; PTCA: Percutaneous transluminal coronary angioplasty; RCHA2DS2-VASc: Renal failure addition to CHA2DS2-VASc; TIA: Transient ischemic attack; TIMI: Thrombolysis in Myocardial Infarction.

Table 2. Univariate and multivariate regression analysis of predictors of no-reflow in the study population

Variables	Unadjusted OR (95% CI)	p value (95% CI)	Adjusted OR	*p* value
CHA_2DS_2-VASc score, 1-SD increase	1.72 (1.47–2.05)	<0.001	1.56 (1.31–1.84)	<0.001
R2CHA2DS2-VASc score, 1-SD increase	2.82 (1.93–3.41)	<0.001	3.26 (2.81–4.31)	<0.001
GFR 1-SD increase	1.84 (1.21–2.55)	<0.001	2.26 (2.03–3.21)	<0.001
Anemia	2.19 (1.36–3.52)	0.001	1.35 (0.66–2.67)	0.39
Stent length, 1-SD increase	1.45 (1.21–1.70)	<0.001	1.42 (1.19–1.70)	<0.001
Stent diameter, 1-SD increase	0.56 (0.44–0.71)	<0.001	0.65 (0.51–0.83)	<0.001
MI type	1.94 (1.26–2.79)	0.001	1.71 (1.16–2.58)	0.10

CHA2DS2-VASc: Congestive heart failure, hypertension, age ≥75 years (doubled), diabetes mellitus, prior stroke or transient ischemic attack (doubled), and vascular disease, age 65-74 years, and sex category (female); CI: Confidence interval; GFR: Glomerular filtration rate; MI: Myocardial infarction; OR: Odds ratio; RCHA2DS2-VASc: Renal failure addition to CHA2DS2-VASc.

patients are provided in Table 1. Compared with the control group, patients in the no-reflow group were older (median age: 65 years [16] *vs.* 60 years [16]; p<0.001). The median CHA2DS2-VASc score was significantly higher in the no-reflow group compared with the control group (2 [3] *vs.* 1 [2]; p<.001). The median RCHA2DS2-VASc score was also significantly higher in the no-reflow group (2 [4] *vs.* 1 [2]; p<.001). Moreover, all of the components of the CHA2DS2-VASc score were statistically significantly different between the 2 groups: the left ventricular ejection fraction (%) was significantly lower in the no-reflow group (38.1% *vs.* 33.9%; p<0.001), while the rate of hypertension (41.3% *vs.* 34.2%; p<0.01), age 65–74 years (9.4% *vs.* 5.1%; p<0.001), diabetes mellitus (42.6% *vs.* 32%; p=0.02), previous stroke/transient ischemic attack (8% *vs.* 1.8%; p<0.001), vascular disease (25.3% *vs.* 13.8%; p=0.001), age ≥75 years (14.1% *vs.* 5.6%; p=0.002), and female sex (28% *vs.* 18.3%; p=0.01) were significantly higher in the no-reflow group. Patients with no-reflow had a significantly lower mean GFR (43.2 mL/min/1.73 m^2 *vs.* 64.9 mL/min/1.73 m^2). There was no significant difference between the 2 groups in the duration from symptom initiation to primary PCI (179.6 min [122.1] *vs.* 158.8 min [128.2]; p=0.15).

Angiographic findings revealed that a longer stent length (26 cm [14] *vs.* 22 cm [11]; p<0.001) and smaller stent diameter (2.75 mm [0.50] *vs.* 3 mm [0.75]; p<0.001) were associated with no-reflow.

Variables that had a significant p value in the descriptive analysis were entered into univariate and multivariate regression analysis to determine potential risk factors of no-reflow. Results of this analysis are illustrated in Table 2. Individual components of the RCHA2DS2-VASc score as a risk factor of no-reflow were not entered in this analysis to avoid multicollinearity. Ventricular arrhythmia, glycoprotein 2b/3a infusion, and intra-aortic balloon pump rates were also excluded from the analyses as potential consequences of no-reflow. The results of the multivariate logistic regression analysis showed that GFR was a significant independent predictor (OR: 2.26, 95% CI: 2.03–3.21; p<0.001), as well as the CHA2DS2-VASc score (OR: 1.56, 95% CI: 1.31–1.84; p<0.001) and the RCHA2DS2-VASc score (OR: 3.26, 95% CI: 2.81–4.31; p<0.001). The ROC analysis depicted in Figure

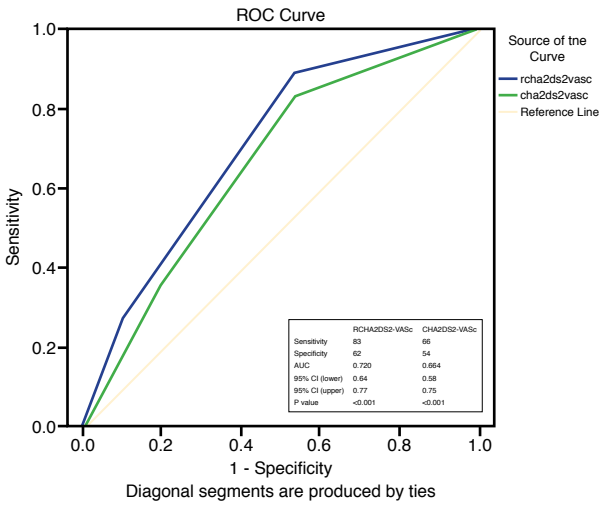

Figure 2. Receiver operating characteristic (ROC) analysis of a cut-off value for the CHA2DS2-VASc and RCHA2DS2-VASc scores to predict no-reflow. AUC: Area under the curve; CI: Confidence interval.

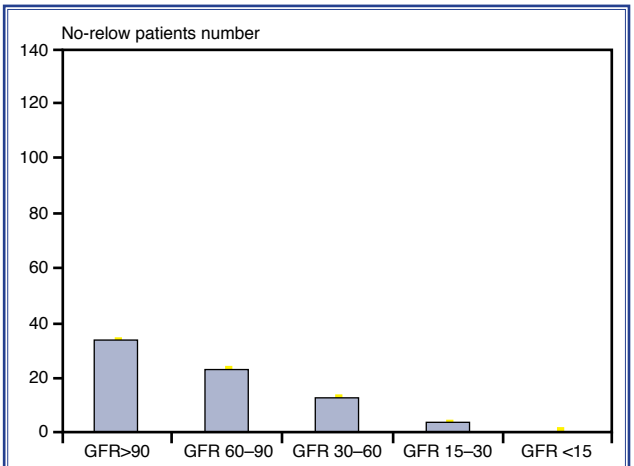

Figure 3. Glomerular filtration rate (GFR) in patients with no-reflow.

2 determined a cut-off value for the CHA2DS2-VASc and RCHA2DS2-VASc scores to predict no-reflow. Our results indicated that a CHA2DS2-VASc score of ≥2 could be used as a predictor of no-reflow in patients presenting with acute STEMI with a sensitivity of 66% and a specificity of 54%, with an area under the curve of 0.664 and a 95% CI of 0.58–0.75. An RCHA2DS2-VASc score of ≥2 could be used as a predictor of no-reflow in patients presenting with acute STEMI with a sensitivity of 83% and specificity of 62%, an area under the curve of 0.72 and a 95% CI of 0.64–0.77. The relationship between glomeruler filtration rate value and no-reflow is shown in Figure 3.

DISCUSSION

This study highlights that in patients with STEMI, renal failure is associated with an increased risk of no-reflow. Our findings demonstrated that the RCHA2DS2-VASc score served as a predictor of no-reflow after primary PCI in patients with STEMI. An RCHA2DS2-VASc score of ≥2 had a sensitivity of 83% and a specificity of 62%. Although both score systems are predictive, we found that the RCHA2DS2-VASc score had a greater sensitivity and specificity to predict no-reflow in patients with STEMI.

In most patients with acute STEMI, primary PCI is a priority method of revascularization. Yet despite achieving a patent epicardial coronary artery, a sudden reduction in myocardial blood flow after PCI, no-reflow phenomenon, can lead to adverse outcomes in these patients.[15] No-reflow is associated with a poorer prognosis in patients with STEMI. Although there are some treatment options for no-reflow, the success rate remains variable. This is due to the complexity of the pathophysiological mechanisms which were considered to be responsible.[16,17] Various elements of the no-reflow process have been examined. As confirmed by our findings, cardiogenic shock, a lesion length of >20 mm, and smaller stent diameter have been shown to be predictors of no-reflow.[18] Some authors have suggested postponing a stent strategy to prevent no-reflow after PCI.[18]

A fast and simple scoring system for no-reflow risk classification in STEMI patients who are candidates for primary PCI would help the physician to choose the best treatment strategy more easily. The underlying thromboembolic event mechanisms in no-reflow and AF are similar; therefore, the relationship between the CHA2DS2-VASc score, a thromboembolic risk marker for AF, and no-reflow has been examined in the literature.[6]

The hypercoagulable state of patients with chronic renal disease presents an increased risk of no-reflow in STEMI.[19] Ipek et al.[6] found that chronic renal failure was significantly higher in a no-reflow group; however, this study did not include a GFR parameter. We also found a significantly greater number of patients with chronic renal failure in the no-reflow group, in addition to an elevated GFR. Sensoy et al.[8] demonstrated that renal dysfunction at admission was an independent predictor of no-reflow in STEMI.

Since the CHA2DS2-VASc score is insufficient to fully indicate the risk of thromboembolism in patients with renal dysfunction, we used the modified RCHA2DS2-VASc score in this study.

The CHA2DS2-VASc score is a set of risk factors for thromboembolism and stroke suggested by the present guidelines for use as a proven predictor of thromboembolic events in patients with AF.[20]

Stroke and transient ischemic attack may occur as a result of non-atherosclerotic vascular pathologies, as well as thromboembolism and atherosclerosis.[21] Abnormal vascular function may be a stroke mediator.[22] Microvascular dysfunction also plays a role in no-reflow. While the thrombus burden and embolism are important parts of the etiology of no-reflow, microvascular dysfunction and occlusion after primary PCI occur in half of the patients.[23] Most of the risk

factors discussed, such as hypertension, diabetes mellitus and female sex, are associated with microvascular dysfunction.[24] In our cohort, multivariate analysis revealed a significant relationship between no-reflow and female gender. Congestive heart failure,[6] hypertension and ischemic cardiomyopathy,[3] as well as age 65–74 years and age ≥756 were predictors of no-reflow in our study, as seen in previous research. Diabetes mellitus, also a component of the CHA2DS2-VASc score, has been shown to be associated with impaired microvascular perfusion after PCI because of the tendency for endothelial vasoconstriction and thrombosis.[25]

Our findings also revealed a significant positive correlation between the CHA2DS2VASc and R2CHADS2 scores. This is not a surprise, as the 2 scoring systems share many of the same components and give them similar weight. Therefore, this would appear to support the validity of using an R2CHADS2 score to estimate the risk of no-reflow. However, the addition of renal failure may not yet alter clinical decisions or outcomes since the CHA2DS2VASc has already been validated and is currently in use. Due to the high morbidity and mortality associated with thromboembolism, it is important to try to control modifiable risk factors such as renal function.

Although most of the risk factors used to calculate the CKD-EPI equation and to predict AF and stroke are the same, renal failure may be a separate independent risk factor for thromboembolism. Piccini et al.[7] added a GFR component using the Cockcroft–Gault formula to the CHADS2 score and created the R2CHADS2 score. In this study, we used the CKD-EPI equation instead of the

Cockcroft–Gault formula when calculating GFR because a previous study showed that the R2CHADS2, provided a significant improvement in the ability to predict mortality risk in older patients with AF.[26] Huang et al.[27] enrolled 3295 coronary artery disease (CAD) subjects and found that the predictability of the R2CHADS2 was comparable to the Global Acute Coronary Events Registry score. Compared with the CHADS2 score (C-statistic=0.61), the R2CHADS2 (C-statistic=0.66) score provided better discrimination for mortality ($p<0.05$). The results of this study showed that the RCHADS2 score could be used to predict composite events for patients with CAD, and that the area under the curve of R2CHADS2 was statistically greater than that of the CHADS2 score. Decreased renal function plays a critical role in the prognosis of cardiovascular results in patients with CAD. Several potential mechanisms may explain these findings. Patients with reduced renal function often have consequences such as anemia, excessive volume burden and oxidative stress that contribute to poor outcomes. In our study, the RCHA2DS2-VASc score (C-statistic=0.72) provided better discrimination for no-reflow than the CHA2DS2-VASc score (C-statistic=0.66) ($p=0.043$).

To our knowledge, although the CHA2DS2-VASc score has already been tested, the prognostic role of the RCHA2DS2-VASc score modification has not been investigated in no-reflow in patients with STEMI. The results of this study suggest that the RCHA2DS2-VASc score may predict risk for no-reflow for patients with STEMI better than the CHA2DS2-VASc score with reasonable efficacy.

Limitations

Our study has several limitations. This was an observational, retrospective, single-center study with a relatively small number of patients. Additional studies expanding on our preliminary results will be beneficial. For example, in the present study, patients without STEMI and those with unstable coronary artery disease were not included. Also, in our study, no-reflow phenomenon was defined by angiography, clinically diagnosed no-reflow was not addressed. Further studies are required to confirm the findings.

Conclusion

The RCHA2DS2-VASc is an easily calculated and efficient index that may be a powerful and independent predictor of no-reflow in STEMI patients. The authors suggest that it would be a useful adjunct to standard tests in the diagnosis of no-reflow.

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Ethical statement: This study was approved by the Tokat Gazi Osman Paşa University Faculty of Medicine, Clinic Research Ethics Committee (date: 26/12/2019, no: 19-KAEK-261).

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