ORIGINAL ARTICLE

CHA_2DS_2-VASc score as a predictor of no-reflow phenomenon after saphenous vein graft percutaneous coronary intervention in patients with non–ST-segment elevation acute coronary syndromes

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KEY WORDS
CHA_2DS_2-VASc score, no-reflow phenomenon, percutaneous coronary intervention, saphenous vein graft disease

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ABSTRACT

BACKGROUND Percutaneous coronary intervention (PCI) of saphenous vein grafts (SVGs) is associated with an increased risk of complications, particularly no-reflow phenomenon and distal embolization due to low patency rates. The CHA_2DS_2-VASc score is a clinical risk stratification tool used to predict thromboembolism events especially in patients with nonvalvular atrial fibrillation.

AIMS The aim of this study was to investigate the relationship between the CHA_2DS_2-VASc score and no-reflow phenomenon after SVG PCI in patients with non–ST-segment elevation acute coronary syndromes (NSTE-ACS).

METHODS In this study, we included 268 patients diagnosed with NSTE-ACS who underwent PCI for SVG disease in our tertiary cardiovascular center. Patients were divided into 2 groups: group 1 without no-reflow phenomenon (n = 190) and group 2 with no-reflow phenomenon (n = 78) following the intervention, and then compared based on CHA_2DS_2-VASc scores.

RESULTS The CHA_2DS_2-VASc score (P <0.001) was significantly higher in group 2, even though no significant difference regarding atrial fibrillation was observed between the study groups. The CHA_2DS_2-VASc score (P <0.001), degenerated saphenous vein graft (P = 0.006), and intraluminal thrombus (P <0.001) were found to be independent predictors of no-reflow phenomenon. Receiver operating characteristics analysis showed that a CHA_2DS_2-VASc score of 4 predicted no-reflow phenomenon with 67.9% sensitivity and 69.3% specificity.

CONCLUSIONS Our findings suggest that the CHA_2DS_2-VASc score can be an independent predictor of no-reflow phenomenon in patients undergoing SVG interventions. As a simple and easy-to-calculate score, it might be a useful assessment tool to predict no-reflow phenomenon before SVG interventions in patients with NSTE-ACS.

INTRODUCTION Coronary artery bypass grafting (CABG) is a revascularization method that is widely used in multivessel and / or left main coronary artery disease. Due to good availability, saphenous vein grafts (SVGs) are the most commonly employed conduits in CABG. However, SVG patency rates decrease to 50% after 10 years, which is caused by degenerative and / or occlusive disease.[1] Loss of the vasa vasorum at harvesting, high inflammatory

WHAT'S NEW?

Interventions to saphenous vein grafts (SVGs) are strenuous for interventional cardiologists, because they carry a high risk of complications such as no-reflow phenomenon observed in these conduits. So far, no prominent predictors of this cardiovascular event have been determined. The CHA_2DS_2-VASc score is used in routine clinical practice to predict thromboembolic events in patients with atrial fibrillation. Based on the fact that no-reflow phenomenon is characterized by thromboembolic pathophysiology, we investigated the relationship between the CHA_2DS_2-VASc score and no-reflow phenomenon after percutaneous intervention in SVGs in patients with non–ST-segment elevation acute coronary syndromes. The analysis showed that the CHA_2DS_2-VASc score was significantly higher in the presence of no-reflow phenomenon. Therefore, we concluded that the CHA_2DS_2-VASc score might be used as a simple tool to predict this event in patients with non–ST-segment elevation acute coronary syndromes who undergo SVG interventions.

mediator levels, and exposure to arterial pressures accelerate SVG occlusion.[2] There are 2 options of SVG disease treatment: reoperation or percutaneous coronary intervention (PCI). Since reoperation is associated with high mortality rates, PCI is the preferred treatment of choice in SVG diseases. Saphenous vein graft PCIs represent about 5% to 10% of all PCIs, but distal embolization and slow reflow or no-reflow phenomenon are more common than in native coronary artery interventions.[3] A 10-fold increased risk of in-hospital mortality has been reported for SVG PCIs.[4] The procedure is related to high rates of acute (no-reflow phenomenon and periprocedural myocardial infarction) and long-term (restenosis and reocclusion) complications.[5,6] Percutaneous treatment of SVG lesions is challenging for interventional cardiologists because of high rates of complications.[1] No-reflow phenomenon, which occurs in around 15% of SVG PCIs, is a serious issue for interventional cardiologists, as there is no specific treatment for this condition. It is also associated with high rates of early and late major adverse cardiac events and mortality.[7-9] No-reflow mechanisms have not been fully elucidated yet, but they are thought to be caused by microembolism and microvascular dysfunction due to thrombosis and spasm.[10]

The CHA_2DS_2-VASc score is a clinical risk stratification scale to predict thromboembolic events.[11] Its use is recommended in patients with nonvalvular atrial fibrillation in whom oral anticoagulant therapy is necessary. Recent studies have shown that the CHA_2DS_2-VASc score could predict adverse clinical outcomes in patients with stable coronary artery disease or acute coronary syndrome (ACS) regardless of the presence of atrial fibrillation.[12] The components of this score include similar, common risk factors such as atherosclerosis, vascular spasm, and microvascular dysfunction with no-reflow phenomenon.[13] The presence of similar risk factors and pathophysiological mechanisms of no-reflow phenomenon suggests that the CHA_2DS_2-VASc score could be used to predict its development. Therefore, we investigated the relationship between the CHA_2DS_2-VASc score and no-reflow phenomenon after PCI of SVGs in patients with non–ST-segment elevation ACS (NSTE-ACS).

METHODS We retrospectively analyzed the data of 386 consecutive patients who underwent PCI for SVG disease accompanied by ACS in our tertiary cardiovascular center between February 2016 and January 2019. The culprit lesion in NSTE-ACS involved the SVG in all patients in our study; therefore, PCIs were performed during the index procedure in the whole study group. We did not include patients treated with stent implantation for stable coronary artery disease. Acute coronary syndromes were diagnosed according to the European Society of Cardiology guidelines.[14] Patients with cardiogenic shock (n = 21), stent restenosis and thrombosis (n = 20), those who underwent PCI for ST-segment elevation MI (STEMI; n = 35), and those who underwent percutaneous transluminal balloon angioplasty only (n = 42) were excluded from the study. Laboratory, clinical, and demographic data were obtained from hospital records. The local ethics committee approved the study. Patient written informed consent was waived, as the study had a retrospective design.

Coronary angiography was performed under standard medical treatment for ACS. Each patient received a loading dose of P2Y12 inhibitor (including clopidogrel 600 mg as the main agent as well as prasugrel 60 mg and ticagrelor 180 mg in a minority of patients) combined with atorvastatin (80 mg), unfractionated heparin (UFH), and acetylsalicylic acid (300 mg) in antiplatelet-naive patients. In patients who had already been on antiplatelets, the maintenance dose was continued and the loading dose of absent antiplatelet treatment was administered. If the glycoprotein IIb/IIIa receptor antagonist use was planned, we administered a 50- to 70-unit/kg intravenous bolus of UFH to achieve the therapeutic activated clotting time. Otherwise, we considered a 70- to 100-unit/kg bolus dose of UFH to achieve the therapeutic activated clotting time.

Coronary no-reflow phenomenon was defined as a Thrombolysis In Myocardial Infarction (TIMI) flow grade below 3 without clear evidence of dissection, stenosis, or vasospasm.[15] The TIMI flow grades were defined as follows: grade 0—no antegrade flow beyond the lesion; grade 1—a poor distal antegrade flow leading to incomplete filling of the artery; grade 2—slow antegrade flow despite the complete opacification of the entire coronary bed;

and grade 3—the opacification of the entire coronary bed with normal speed.[16] Twenty-five angiograms were randomly selected and evaluated at various time periods by another investigator in order to assess the intra- and interobserver reproducibility of TIMI flow measurements. Cronbach α coefficients for the intra- and interobserver reproducibility of TIMI flow measurements were found to be 0.94 and 0.92, respectively. Patients were classified into 2 groups based on the postintervention TIMI flow grade: group 1 included patients with TIMI flow grade 3, and group 2, those with TIMI flow grades 0 to 2. A degenerated SVG was defined as luminal irregularities or ectasia involving more than 50% of the total graft length. An angiographic thrombus was defined as a separate intraluminal filling defect with defined boundaries substantially separated from the adjacent wall.[17] The CHA_2DS-VASc risk score was calculated by summing the points assigned to each of the risk factors, which included congestive heart failure (CHF) (1 point), hypertension (1 point), age of 75 years and older (2 points), diabetes (1 point), previous stroke, transient ischemic attack or thromboembolism (2 points), vascular disease (history of myocardial infarction [MI], peripheral arterial disease [PAD], or complex aortic plaques) (1 point), age between 65 and 74 years (1 point), and female sex (1

TABLE 1 **Preprocedural demographic, clinical, and laboratory data of the study patients**

Characteristics	Normal reflow (n = 190)	No reflow (n = 78)	*P* value
Demographic data			
Age, y, mean (SD)	62.6 (8.7)	66.2 (9.5)	0.003
Male sex	160 (84.2)	58 (74.4)	0.06
Clinical presentation			
Unstable angina pectoris	54 (28.4)	14 (17.9)	0.07
NSTEMI	136 (71.6)	64 (82.1)	0.07
Age ≥75 y	14 (7.4)	21 (26.9)	<0.001
Age between 65–74 y	70 (36.8)	23 (29.5)	0.25
Hypertension	145 (76.3)	66 (84.6)	0.13
Diabetes	73 (38.4)	42 (53.8)	0.02
Dyslipidemia	101 (53.2)	44 (56.4)	0.63
Current smoking status	42 (22.1)	21 (26.9)	0.4
PAD	38 (20)	22 (28.2)	0.13
History of stroke / TIA	9 (4.7)	17 (22.1)	<0.001
Previous MI	86 (45.3)	48 (61.5)	0.02
Previous PCI	67 (35.3)	37 (38.8)	0.06
COPD	25 (13.2)	5 (6.4)	0.11
Congestive HF	34 (17.9)	43 (55.1)	<0.001
EF, %, mean (SD)	51.8 (9.1)	44.7 (10.8)	<0.001
AF	15 (7.9)	12 (15.4)	0.06
CKD	42 (22.1)	24 (30.8)	0.14
CHA_2DS_2-VASc score, median (IQR)	3 (2–4)	5 (3–6)	<0.001
Serum creatinine, mg/dl, median (IQR)	1.11 (0.8–1.1)	1.15 (0.9–1.22)	0.05
Total cholesterol, mg/dl, mean (SD)	193 (57)	183 (53)	0.23
HDL cholesterol, mg/dl, mean (SD)	39.4 (8.9)	39.1 (11)	0.88
LDL cholesterol, mg/dl, mean (SD)	113 (45)	110 (46)	0.67
Triglycerides, mg/dl, median (IQR)	164 (114–243)	146 (108–245)	0.28

Data are presented as number (percentage) unless otherwise indicated.

Abbreviations: AF, atrial fibrillation; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; EF, ejection fraction; HDL, high-density lipoprotein; HF, heart failure; LDL, low-density lipoprotein; NSTEMI, non–ST-segment elevation myocardial infarction; MI, myocardial infarction; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; TIA, transient ischemic attack

point).[11] Congestive heart failure referred to heart failure with low ejection fraction or a previous diagnosis of heart failure. Hypertension was defined as the use of antihypertensive medication or blood pressure consistently above 140/90 mm Hg. Diabetes was defined as a previous diagnosis of diabetes or use of insulin or oral hypoglycemic agents at the time of admission. Stroke and transient ischemic attack were assessed based on patient history. A stenosis exceeding 50% in noncoronary arteries was defined as PAD.

Statistical analysis Statistical analyses were performed using the 2012 Statistical Package for Social Sciences software for Windows, version 24.0 (IBM SPSS Statistics, IBM Corp., Armonk, New York, United States). For categorical variables, data were expressed as number (percentage) and the Pearson χ^2 and Fisher exact tests were performed. Once the Kolmogorov–Smirnov test was performed to evaluate data distribution, data were expressed as median (interquartile range) for variables without normal distribution and mean (SD) for variables with normal distribution. The *t* test was used to compare quantitative variables with normal distribution, and the Mann–Whitney test was applied to quantitative variables without normal distribution. Univariate and multivariate logistic regression analyses were used to determine independent predictors of no-reflow phenomenon. Receiver operating characteristic analysis was conducted to determine the optimal value of the CHA_2DS_2-VASc score to indicate no-reflow phenomenon in terms of both sensitivity and specificity. A *P* value less than 0.05 was considered significant.

RESULTS A total of 268 consecutive patients diagnosed with NSTE-ACS who underwent PCI of SVGs were included in our study. Baseline demographic, clinical, and laboratory characteristics of patients were demonstrated in TABLE 1. The median (SD) age was 62.6 (8.7) years in group 1, and 66.2 (9.5) years in group 2 ($P = 0.003$). The number of patients at the age of 75 years and older was larger in group 2 compared with group 1 (14 [7.4%] vs 21 [26.9%]; $P < 0.001$). Additionally, the number of patients with diabetes (73 [38.4%] vs 42 [53.8%]; $P = 0.02$), history of stroke (9 [4.7%] vs 17 [22.1%]; $P < 0.001$), previous MI (86 [45.3%] vs 48 [61.5%]; $P = 0.02$), and CHF (34 [17.9%] vs 43 [55.1%]; $P < 0.001$) were higher in group 2 than in group 1. Mean (SD) ejection fraction was lower in group 2 (51.8% [9.1%] vs 44.7% [10.8%]; $P < 0.001$), whereas the CHA_2DS_2-VASc score was higher in group 2 (3 [2–4] vs 5 [3–6]; $P < 0.001$). There was no significant difference in terms of sex, hypertension, dyslipidemia, smoking status, chronic obstructive pulmonary disease, atrial fibrillation, PAD, history of PCI, chronic kidney disease, oral anticoagulation as well as serum creatinine, total cholesterol, low- and high-density lipoprotein cholesterol, and triglyceride levels between the study groups.

Coronary angiography findings and procedural characteristics of the whole study group were demonstrated in TABLE 2. Preinterventional TIMI flow grade 0 (11 [5.8%] vs 14 [17.9%]; $P = 0.02$) and grade 3 (120 [63.2%] vs 24 [30.8%]; $P < 0.001$), degenerated SVG (42 [22.1%] vs 47 [60.3%]; $P < 0.001$), thrombus (30 [15.8%] vs 45 [58.4%]; $P < 0.001$), glycoprotein IIb / IIIa receptor antagonist use (27 [13.6%] vs 31 [44.9%]; $P < 0.001$), and predilatation rates (66 [34.7%] vs 38 [48.7%]; $P = 0.033$) were higher in group 2, while the number of patients implanted with drug-eluting stents (111 [58.4%] vs 33 [49.3%]; $P = 0.047$) was larger in group 1. Stent diameter (3.2 [0.52] vs 3.44 [0.57]; $P = 0.001$) was larger in group 2. The results of univariate and multivariate regression analyses for selected preprocedural and procedural variables in the prediction of no-reflow phenomenon are demonstrated in TABLE 3. The CHA_2DS_2-VASc score (odds ratio [OR], 1.631; 95% CI, 1.281–2.076; $P < 0.001$), degenerated SVG (OR, 2.719; 95% CI, 1.334–5.543; $P = 0.006$), thrombus (OR, 4.309; 95% CI, 2.118–8.766; $P < 0.001$), and an implanted drug-eluting stent (OR, 0.334; 95% CI, 0.16–0.693; $P = 0.003$) were found to be independent predictors of no-reflow phenomenon in multivariate logistic regression analysis. Additionally, the results of univariate and multivariate analyses regarding the predictive power of individual risk factors in the CHA_2DS_2-VASc score for no-reflow phenomenon are shown in TABLE 4. Congestive heart failure (OR, 4.99, 95% CI, 2.586–9.631; $P < 0.001$), age ≥75 years (OR, 2.637; 95% CI, 1.119–6.214; $P = 0.027$), vascular disease (OR, 2.604; 95% CI, 1.39–4.877; $P = 0.003$), and a history of transient ischemic attack / stroke (OR, 5.034; 95% CI, 1.953–12.975; $P = 0.001$]) were independent predictors of no-reflow phenomenon.

Receiver operating characteristics analysis was used to determine the optimal CHA_2DS_2-VASc score cutoff value to indicate no-reflow phenomenon (FIGURE 1). The highest combined sensitivity and specificity values crossed the curve at 4 (sensitivity of 67.9% and specificity of 69.3%). The area under the curve was 0.732 (95% CI, 0.658–0.805; $P < 0.001$). Additionally, the negative predictive value was 75.8%.

DISCUSSION Our study suggests the practicality of the CHA_2DS_2-VASc score in estimating no-reflow phenomenon in patients with NSTE-ACS

TABLE 2 Coronary angiographic findings and procedural characteristics of the study patients

Variable	Normal reflow (n = 190)	No reflow (n = 78)	*P* value
Time elapsed from surgery to angiography, y, mean (SD)	10.2 (4.9)	10.7 (4.7)	0.47
Narrowed saphenous vein graft to			
Left anterior descending artery	14 (7.4)	5 (6.4)	0.99
Diagonal artery	21 (11.1)	9 (11.5)	>0.99
Circumflex artery	82 (43.2)	37 (47.4)	0.95
Right coronary artery	73 (38.4)	27 (34.6)	0.95
Lesion site			
Osteal	24 (12.1)	14 (20.3)	0.12
Proximal	54 (27.1)	24 (34.8)	0.75
Middle	66 (33.2)	22 (31.9)	0.95
Distal	55 (27.6)	9 (13)	0.12
TIMI flow grade before the intervention			
0	11 (5.8)	14 (17.9)	0.02
1	10 (5.3)	8 (10.3)	0.52
2	49 (25.8)	32 (41)	0.1
3	120 (63.2)	24 (30.8)	<0.001
TIMI flow grade following the intervention			
0	0	8 (10.3)	<0.001
1	0	19 (24.4)	<0.001
2	0	51 (65.3)	<0.001
3	190 (100)	0	<0.001
Procedural data			
Degenerated saphenous vein graft	42 (22.1)	47 (60.3)	<0.001
Intraluminal thrombus	30 (15.8)	45 (58.4)	<0.001
Drug-eluting stent	111 (58.4)	33 (49.3)	0.047
Stent diameter, mm, mean (SD)	3.2 (0.52)	3.44 (0.57)	0.001
Stent length, mm, mean (SD)	23.5 (10.6)	26.6 (14.8)	0.11
Predilatation	66 (34.7)	38 (48.7)	0.03
Postdilatation	46 (23.1)	14 (20.3)	0.63
Oral anticoagulation	10 (5)	7 (10.1)	0.1
Glycoprotein IIb / IIIa inhibitor use	27 (13.6)	31 (44.9)	<0.001
Antiplatelets			
Acetylsalicylic acid	180 (95)	72 (92.3)	0.53
Clopidogrel	171 (89.5)	74 (94.9)	0.43
Ticagrelor	15 (7.9)	3 (3.8)	0.49
Prasugrel	4 (2.1)	1 (1.3)	0.88
Additional variables			
Distal protection device use	7 (3.7)	4 (5.1)	0.74
Thrombectomy	1 (0.5)	1 (1.3)	0.51

Data are presented as number (percentage) unless otherwise indicated.

Abbreviations: TIMI, Thrombolysis in Myocardial Infarction

TABLE 3 Univariate and multivariate regression analysis of selected preprocedural and procedural variables in predicting no-reflow phenomenon

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value
CHA_2DS_2-VASc	1.963 (1.589–2.425)	<0.001	1.631 (1.281–2.076)	<0.001
Dyslipidemia	1.14 (0.671–1.938)	0.63	–	–
Current smoking status	1.298 (0.708–2.381)	0.4	–	–
COPD	0.452 (0.166–1.227)	0.12	–	–
NSTEMI	1.815 (0.939–3.507)	0.08	–	–
Time elapsed from surgery to angiography	1.022 (0.968–1.078)	0.44	–	–
TIMI flow grade 0 before the intervention	1.418 (0.598–3.362)	0.43	–	–
Degenerated saphenous vein graft	5.343 (3.026–9.432)	<0.001	2.719 (1.334–5.543)	0.01
Thrombus	7.5 (4.125–13.637)	<0.001	4.309 (2.118–8.766)	<0.001
Drug-eluting stent	0.528 (0.304–0.91)	<0.001	0.334 (0.16–0.693)	0.003
Stent diameter	2.188 (1.352–3.54)	0.001	2.554 (0.998–7.669)	0.07
Stent length	1.02 (0.999–1.042)	0.06		
Predilatation	1.785 (1.045–3.048)	0.03	1.467 (0.721–2.984)	0.29
Postdilatation	0.953 (0.504–1.799)	0.88	–	–
Distal protection device use	1.413 (0.492–4.971)	0.59	–	–
AF	2.121 (0.943–4.769)	0.07	–	–
CKD	0.639 (0.354–1.152)	0.14	–	–

Abbreviations: OR, odds ratio; others, see TABLE 1

TABLE 4 Univariate and multivariate analysis of the predictive power of individual risk factors in CHA_2DS_2-VASc score regarding no-reflow phenomenon

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Congestive HF	5.637 (3.155–10.970)	<0.001	4.990 (2.586–9.631)	<0.001
Hypertension	1.707 (0.848–3.438)	0.13	–	–
Age ≥75 years	4.632 (2.211–9.701)	<0.001	2.637 (1.119–6.214)	0.03
Diabetes	1.78 (1.098–3.185)	0.02	1.232 (0.656–2.313)	0.52
History of stroke / TIA	5.698 (2.413–13.453)	<0.001	5.034 (1.953–12.975)	0.001
Vascular disease	2.456 (1.423–4.237)	0.001	2.604 (1.390–4.877)	0.003
Age of 65–74 years	0.717 (0.406–1.266)	0.25	–	–
Female sex	1.839 (0.969–3.49)	0.06	–	–

Abbreviations: see TABLES 1 and 3

undergoing SVG interventions. Also, a cutoff CHA_2DS_2-VASc value above 4 were found to predict no-reflow phenomenon in these patients. Our findings are similar to those of previous studies that evaluated the prediction of no-reflow phenomenon using the CHA_2DS_2-VASc score in patients with STEMI undergoing primary PCI.[18-20]

Saphenous vein grafts have a progressive closure rate, estimated at 12% to 20% at the end of the first year and approximately 50% by 10 years after the procedure.[21] Percutaneous coronary intervention is the preferred revascularization method in patients with narrowed or occluded SVGs and represents approximately 5% to 10% of all PCIs.[3] Atherosclerotic plaques in SVGs are more diffuse, friable, and prone to an extensive thrombotic burden in comparison to atherosclerotic plaques in native coronary arteries.[22] Therefore, SVG PCI is associated

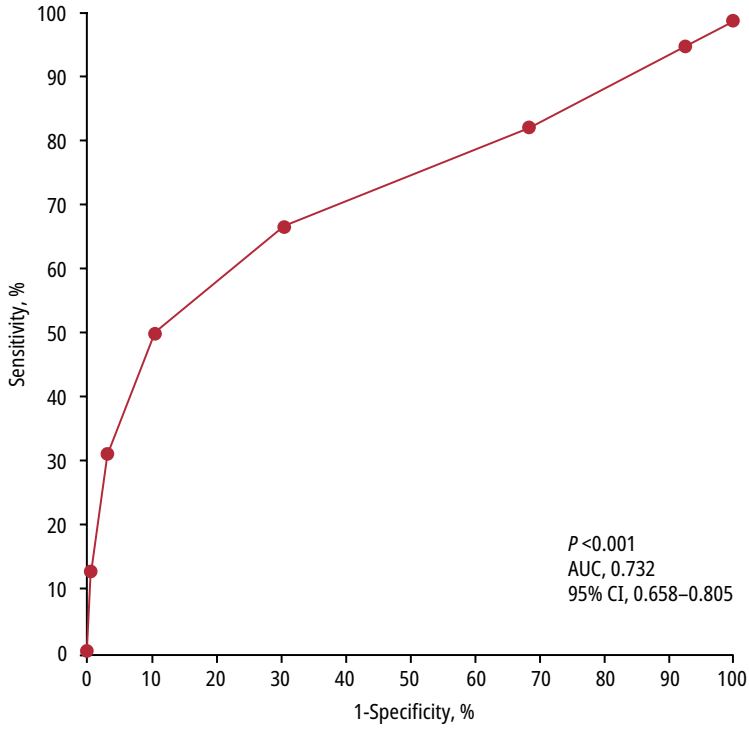

FIGURE 1 Receiver operating characteristics curve analysis showing the CHA_2DS_2-VASc score cutoff value of 4 that predicted no-reflow phenomenon with 67.9% sensitivity and 69.3% specificity
Abbreviations: AUC, area under the curve

with a high risk of major adverse cardiac events due to no-reflow phenomenon resulting from the distal embolization of atherosclerotic plaque and thrombotic debris within the graft.[4,21,23]

The CHA_2DS_2-VASc score is used to determine the risk of thromboembolism in patients with atrial fibrillation.[11] It has also been suggested that the CHA_2DS_2-VASc score can provide prognostic information regarding ischemic events and mortality in patients with coronary artery disease and in those undergoing PCI and CABG.[24-27] For this reason, the components of the CHA_2DS_2-VASc score may be associated with increased ischemic risk, thrombotic risk, atherosclerotic process, and microvascular dysfunction, all of which play a role in the mechanism and pathogenesis of no-reflow phenomenon.[19,28,29] Similar to our report, studies investigating the effect of the CHA_2DS_2-VASc score on predicting no-reflow phenomenon in patients with STEMI undergoing primary PCI have also found a relationship between them.[18-20] Various approaches and suggestions to prevent no-reflow phenomenon in SVG interventions were presented in clinical studies. These include thrombectomy, distal embolic protection device use, direct stenting, and glycoprotein IIb/IIIa inhibitor use.[30,31] Therefore, these additional therapies for the prevention of no-reflow phenomenon might be considered during SVG interventions. Detecting patients undergoing SVG intervention who are at high risk of developing no-reflow phenomenon by using this simple and quick scoring system may help to choose the best treatment strategy, eg, using additional protection devices and stenting of the narrowed or occluded native vessel to which the graft is attached.[32]

Several studies have shown that CHF and age ≥75 years were related to no-reflow phenomenon.[18,19,33] Consistently with previous reports, multivariate analysis in our study showed that CHF and age ≥75 years were independently associated with no-reflow phenomenon. Similar to another report, a history of cerebrovascular ischemic events was also found to be an independent predictor of no-reflow phenomenon.[19] One explanation of this finding may be the similarity between risk factors for microvascular dysfunction, which was suggested to be a mediator of no-reflow phenomenon and stroke.[28,34] However, we did not observe the previously known effects of female sex and hypertension on no-reflow phenomenon occurrence.[29,35] Furthermore, diabetes impairs normal endothelial function, leading to microvascular dysfunction.[36] Although the patients with no-reflow phenomenon had a significantly higher incidence of diabetes compared with those without no-reflow phenomenon, the relationship between diabetes and no-reflow phenomenon disappeared after multiple adjustments. The presence of a thrombus on angiography was found to be an independent predictor of no-reflow phenomenon.[37] Intravascular ultrasound studies showed that the deterioration of SVGs was associated with an increased risk of no-reflow phenomenon in SVG interventions.[38] Similar to these findings, our study also showed that thrombotic lesions and degenerated SVGs could independently predict no-reflow phenomenon before SVG interventions.

Antiplatelet use after CABG plays a vital role in adverse cardiovascular events and graft patency. DeStephan and Scheider[39] have already noted the significance of antiplatelet and anticoagulant therapy at follow-up in patients who underwent CABG surgery. Therefore, in our study, we also evaluated antiplatelet use and there was no significant difference observed between the 2 groups.

Study limitations Our study had several limitations. First, it was limited by the retrospective design. Second, the sample size was relatively small; hence, further prospective studies of larger cohorts are needed to confirm our results. Finally, the angiographic assessment of coronary lesions is less sensitive and less specific than intravascular ultrasound or optical coherence tomography evaluation.

Conclusions In conclusion, our findings suggest the CHA_2DS_2-VASc score is an independent predictor of no-reflow phenomenon in patients

with NSTE-ACS undergoing SVG interventions. The CHA_2DS_2-VASc score, calculation of which is simple and not time-consuming, can be a very useful risk assessment tool to stratify patients who are prone to no-reflow phenomenon before SVG interventions.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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