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ORIGINAL ARTICLE

The association of PRECISE-DAPT score with development of contrast-induced nephropathy in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention

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Abstract

Given that parameters used in the calculation of the PRECISE-DAPT risk score are important contributors to contrast-induced nephropathy (CIN) development, we hypothesized that the PRECISE-DAPT risk score would show good accuracy for predicting CIN in patients with ST-elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI). Therefore, in this study, we aimed to determine the predictive value of the admission PRECISE-DAPT score for the occurrence of CIN in patients with STEMI treated with primary PCI. After evaluation regarding with exclusion criteria, 1280 patients were, respectively, enrolled in the study. The primary end-point was the incidence of CIN. The PRECISE-DAPT score of CIN group was higher than the non-CIN group [31 (24–41) vs. 14 (9–23), $p < 0.001$, respectively]. In multivariate logistic regression analysis, PRECISE-DAPT score was independently associated with the development of CIN [odds ratio (OR) 1.090, 95% confidence interval (CI) 1.066–1.114, $p < 0.001$]. A receiver-operating characteristic (ROC) analysis was drawn to show the best cut-off value of the PRECISE-DAPT score to predict CIN was ≥ 21 with 81.3% sensitivity and 72.7% specificity [area under curve (AUC): 0.834; 95% CI 0.812–0.854; $p = 0.017$]. The PRECISE-DAPT score may be a significant independent predictor of CIN in patients with STEMI treated with primary PCI. Therefore, follow-up of patients with higher PRECISE-DAPT score should be performed more cautiously, and it should be noted that the development of CIN risk of these patients group is high.

Keywords PRECISE-DAPT score · ST-elevation myocardial infarction · Contrast-induced nephropathy · Primary percutaneous coronary intervention

Introduction

ST-elevation myocardial infarction (STEMI) is associated with a significant morbidity and mortality in patients with ischemic heart diseases. According to current guidelines, the standard of care treatment for STEMI is a diagnostic angiography followed by percutaneous coronary intervention (PCI), which is aimed to restore coronary blood flow immediately [1]. However, even though successful revascularization, primary PCI is related with a higher incidence of contrast-induced nephropathy (CIN), a complication that is associated with increased in-hospital, short and long-term mortality, when compared to elective procedures [2]. CIN is defined as the elevation of serum creatinine levels by 0.5 mg/dL or 25% that occurs within 72 h after the

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intravascular administration of contrast media, with excluding other causes [3]. Multiple risk factors have been showed to be related with CIN including the type and dosage of contrast media, concomitant nephrotoxic medication, inflammation, diabetes mellitus, renal insufficiency, congestive heart failure (CHF), increasing age, low hemoglobin levels, white blood cell count, and female gender [3–6]. A crucial step to minimize the risk of developing CIN is to identify patients at risk and then initiate appropriate prophylactic measures [7].

The PRECISE-DAPT score has been recently developed to decide the optimal duration of dual anti-platelet treatment in patients after PCI [8]. The PRECISE-DAPT score includes age, hemoglobin level, white blood cell count, creatinine clearance rate, and prior history of bleeding. Given that age, hemoglobin level, white blood cell count, and creatinine clearance rate are all important contributors to CIN development, we hypothesized that the PRECISE-DAPT risk score would show a good accuracy for predicting CIN in patients with STEMI undergoing primary PCI. Nonetheless, the suitability of the PRECISE-DAPT risk score for predicting CIN in patients with STEMI remains unknown. Therefore, in this study, we aimed to evaluate the association of admission PRECISE-DAPT score for the occurrence of CIN in patients with STEMI treated with primary PCI.

Materials and Methods

Patient population

The present retrospective study included 1280 patients who had been diagnosed with STEMI and undergone primary PCI within 12 h after the onset of symptoms between January 2014 and June 2017. Patients with severe infection, cardiogenic shock, who were pregnant or breastfeeding, allergic to contrast media, and had been exposed to contrast media and nephrotoxic medications (e.g.; aminoglycosides and non-steroidal anti-inflammatory drugs) within the previous 7 days were excluded from the study. In addition, patients who admitted to hospital with the onset of chest pain after 12 h, undergoing chronic peritoneal dialysis or hemodialysis treatment, and treatment with any thrombolytic agents were also excluded from the study. Besides that, a total of 6 patients were excluded from the study because of insufficient data form the hospital electronic database. Baseline demographic characteristics and related clinical information were obtained for each patient from the hospital electronic database. The PRECISE-DAPT score was calculated for each patient using web calculator. The study protocol was approved by the Ethics and Research Committee of our hospital and it was performed according to the Principles of Declaration of Helsinki. The need for written informed consent was waived due to the retrospective study design.

Primary percutaneous coronary intervention

All patients underwent PCI via femoral artery using non-ionic, iso-osmolarity, contrast medium; iodixanol (320 mg iodine/mL; 290 mOsm/kg of water; Vispaque, GE Healthcare Inc., USA). Unless contraindicated, all patients were treated with 300 mg acetylsalicylic acid, along with a loading dose of 600 mg clopidogrel. The amount of intravenous hydration and the infusion rate in patients with or without severe left-ventricular dysfunction or overt heart failure were left at the discretion of the interventional cardiologist. However, in accordance of hospital protocol in our institution, intravenous hydration rate for the prevention of CIN is 1 mL/kg/h with normal saline over 12 h in patients without heart failure. For patients with heart failure, the intravenous hydration rate is 0.5 mL/kg/h with normal saline over 12 h. Standard intravenous bolus unfractionated heparin (70–100 U/kg) and additional doses as needed were given to achieve activating clotting time of >250 s before the coronary intervention. The GpIIb-IIIa inhibitor [Tirofiban (Aggrastat) 12.5 mg/50 mL; DSM Pharmaceuticals, Greenville, North Carolina] was also left to the operator's judgment per institutional protocol. Before the discharge from hospital, all patients were prescribed aspirin, clopidogrel, statins, angiotensin-converting enzyme inhibitor or angiotensin receptor blocker, and beta-blockers according to their blood pressure and heart.

Analysis of blood samples and echocardiography

The blood samples [baseline and peak creatinine, estimated glomerular filtration rate (eGFR), white blood cell count, platelet count, C-reactive protein (CRP), and hemoglobin levels] were collected upon admission to emergency department and analyzed using the standard biochemical techniques with the Beckman Coulter LH 780 device (Beckman Coulter Ireland Inc. Mervue, Galway, Ireland). After fasting for ≥ 8 h, lipid profiles were measured within the first 24 h. In addition, cardiac biomarker levels including troponin I and creatine kinase–myocardial band (CK–MB) were measured on admission and their peak values were obtained for analysis. eGFR was calculated using the Modification of Diet in Renal Diseases study equation [9]. Echocardiographic assessment of the left-ventricular ejection fraction was performed within 24 h after primary PCI using a Vivid 7 system (GE Vingmed Ultrasound AS, Horten, Norway) to study patients by an expert on cardiovascular imaging. Left-ventricular ejection fraction was calculated using the Simpson method.

Definitions

CIN was described as the absolute ≥ 0.5 mg/dL or relative > 25% increase in serum creatinine from baseline on emergency department admission to 72 h after exposure of contrast medium [3]. We calculated the change in serum creatinine and confirmed the occurrence of CIN according to the definition of CIN. STEMI was defined as: 1—at least two contiguous leads with ST-segment elevation ≥ 2.5 mm in men < 40 years, ≥ 2 mm in men > 40 years, or ≥ 1.5 mm in women in leads V2–V3 and/or ≥ 1 mm in the other leads [in the absence of left-ventricular (LV) hypertrophy or left bundle branch block LBBB)]; 2—prolonged (> 30 min) typical chest pain at rest; 3—increased serum biomarker of myocardial damage [10]. Hypertension was described as undergoing anti-hypertensive treatment or a systolic pressure > 140 mmHg, and/or a diastolic pressure > 90 mm Hg on at least 2 separate measurements during hospitalization [11]. Diabetes mellitus was described as taking oral antidiabetics or insulin, or follow-up fasting blood glucose levels fulfilling the American Diabetes Association's criteria [12]. Hyperlipidemia was defined as taking lipid-lowering medication upon presentation [13]. Anemia was defined as a baseline hemoglobin value below 13 g/dL for men and 12 g/dL for women [14]. Stroke was defined as a neurologic deficit lasting < 24 h as TIA or if longer as stroke. In addition, all patients were evaluated according to Killip exam classification.

In-hospital outcomes

The primary end-point was CIN incidence after primary PCI. Besides that, we also evaluated in-hospital mortality according to CIN incidence in patients with STEMI treated with PCI.

Statistical analysis

SPSS version 22.0 (Inc, Chicago, IL) was used for statistical analysis. For appropriate distribution characteristics and normality, mean (± standard deviation) or median (0.25–0.75 percentile) was used to express continuous variables, and *t* test or Mann–Whitney *U* test was used for group comparisons. Categorical variables were reported as number (percentages) and compared to the Fisher's exact test or the Chi-square test. The Kaplan–Meier method was used to generate event-free survival curves, and the log-rank test was used to compare survival difference between CIN and non-CIN group. Univariate and multivariate logistic regression analyses identified risk factors for the development of CIN. The Receiver-Operating Curve (ROC) was utilized to derive the cut-off values of the PRECISE-DAPT, age, white blood cell count, hemoglobin, and the Mehran score for predicting CIN. The DeLong method was used to compare the PRECISE-DAPT and aforementioned parameters. A p value that is less than 0.05 indicates statistical significance.

Results

Our cohort was consisted of 1280 STEMI patients who undergone primary PCI. All patient baseline demographics as well as laboratory and angiographic characteristics are listed in Table 1. The mean age of the study population was 59.2 ± 12.4. 832 (65%) patients were female. The study population was divided into two groups: CIN and non-CIN groups. In the present study, CIN developed in 118 (9.2%) patients. A total of 8 (6.8%) patients were treated with hemodialysis or hemofiltration in the CIN group.

Comparison of baseline demographics, laboratory, and angiographic characteristics

Age, left-ventricular ejection fraction, left anterior descending (LAD) as the infarcted related artery, and anterior myocardial infarction were statistically different between groups ($p < 0.05$, for all), whereas Killip class > 1 on admission, gender, mean systolic blood pressure, and heart rate were similar between the groups ($p > 0.05$, for all). The frequency of diabetes mellitus, hypertension, CHF, cerebrovascular accident (CVA), history of bleeding, and anemia in the CIN group were significantly higher in patients with CIN than in those without CIN ($p < 0.05$, for all). In terms of laboratory findings, patients with CIN had lower levels of hemoglobin and eGFR, and higher admission blood glucose, CRP, baseline and peak creatinine, peak CK–MB, contrast volume/eGFR ratio, and troponin I ($p < 0.05$, for all). In terms of lipid profiles, only total cholesterol and triglycerides were different between two groups ($p = 0.035$ and $p = 0.004$, respectively). Multi-vessel involvement was higher in the CIN group compared with the non-CIN group ($p < 0.001$). The amount of contrast dye was similar in the CIN group compared with the non-CIN group ($p > 0.05$). The plasma osmolality in patients with CIN was 284 ± 9.1 mOsm/kg, while it was 281.7 ± 9.1 mOsm/kg for patients without CIN ($p = 0.006$).The PRECISE-DAPT and the Mehran score were significantly higher in patients with CIN compared to those without CIN (31 (24–41) vs. 14 (9–23), 12.6 ± 7.4 vs. 5.84 ± 3.8, $p < 0.001$ and $p < 0.001$, respectively). During the in-hospital course, a total of 67 (5.2%) major and minor bleeding events were occurred.

Independent predictors of CIN

Independent predictors of CIN are shown in Table 2. Age, diabetes mellitus, hypertension, CVA, history of bleeding,

Table 1 Baseline characteristics, laboratory results, angiographic data of all study patients, and patients with and without CIN

	Total study population, $n=1280$	Non-CIN group $n=1162$	CIN group $n=118$	p value
Age, years	59.2 ± 12.4	58.3 ± 12	68.4 ± 11.9	< 0.001
Female gender, *n* (%)	832 (65)	763 (65.7)	69 (58.5)	0.119
On admission				
Systolic blood pressure, mm/Hg	131 ± 31	132 ± 32	130 ± 27	0.954
Heart rate, per minute	77 ± 16	77 ± 16	76 ± 18	0.332
Left-ventricular ejection fraction (%)	44 ± 16	44 ± 16	40 ± 16	< 0.001
Killip class > 1 on admission, *n* (%)	207 (16.2)	188 (16.2)	19 (16.1)	0.983
Anterior myocardial infarction, *n* (%)	572 (44.7)	503 (43.3)	69 (58.5)	0.002
Risk factors				
Diabetes mellitus, *n* (%)	266 (20.8)	224 (19.3)	42 (35.6)	< 0.001
Hypertension, *n* (%)	328 (25.6)	278 (23.9)	50 (42.4)	< 0.001
Hyperlipidemia, *n* (%)	246 (19.2)	230 (19.8)	16 (13.6)	0.102
CHF, *n* (%)	20 (1.6)	13 (1.1)	7 (5.9)	< 0.001
COPD, *n* (%)	12 (0.9)	10 (0.9)	2 (1.7)	0.370
CVA, *n* (%)	23 (1.8)	15 (1.3)	8 (6.8)	< 0.001
History of bleeding, *n* (%)	51 (4.0)	27 (2.3)	24 (20.3)	< 0.001
Smoking, *n* (%)	340 (26.6)	312 (26.9)	28 (23.7)	0.465
Anemia, *n* (%)	400 (31.3)	347 (29.9)	53 (44.9)	< 0.001
Laboratory results				
Hemoglobin, g/dL	13.38 ± 1.80	13.4 ± 1.75	12.7 ± 2.14	< 0.001
White blood cell count, cells/μL	12.39 ± 4.94	12.29 ± 4.70	13.38 ± 5.47	0.499
Platelet count, cells/μL	237.6 ± 72.19	237.52 ± 71.25	238.84 ± 81.16	0.789
Admission blood glucose, mg/dL	133 ± 113	131 ± 112	151 ± 126	< 0.001
CRP, mg/dL	2.15 ± 0.7	2.0 ± 0.66	6.35 ± 1.68	0.004
Baseline creatinine, mg/dL	0.91 ± 0.29	0.88 ± 0.26	1.18 ± 0.39	< 0.001
Peak creatinine, mg/dL	1.07 ± 0.57	0.98 ± 0.45	1.95 ± 0.84	< 0.001
eGFR, mL/m	96.46 ± 28.14	99.09 ± 27.21	70.60 ± 23.81	< 0.001
Peak creatine kinase–myocardial band, ng/mL	107 ± 53	102 ± 50	148 ± 62	0.003
Peak troponin I, ng/Dl	48.5 ± 17.2	45.9 ± 16.0	50.0 ± 32.8	0.010
Total cholesterol, mg/dL	177.1 ± 44.9	178 ± 44.6	168.2 ± 48.2	0.035
TG, mg/dL	131 ± 96	133 ± 96	114 ± 85	0.004
HDL, mg/dL	38.7 ± 11.0	38.6 ± 10.9	39.7 ± 12.2	0.631
LDL, mg/dL	108.5 ± 38.0	109.2 ± 37.7	100.6 ± 40.9	0.068
Contrast volume/eGFR ratio	2.80 ± 1.49	2.67 ± 1.32	4.09 ± 2.25	< 0.001
Plasma osmolality, mOsm/kg	282.0 ± 9.2	281.7 ± 9.1	284 ± 9.1	0.006
Angiographic data				
Multi-vessel stenosis (> 50%), *n* (%)	253 (19.8)	216 (18.6)	37 (31.4)	0.001
Time to treatment, hours	2.9 ± 1.87	2.87 ± 1.83	2.97 ± 1.92	0.424
LAD as the infarct-related artery, *n* (%)	567 (44.3)	500 (43)	67 (56.8)	0.004
Contrast volume, mL	242 ± 83	241 ± 82	253 ± 87	0.176
TIMI < 3 flow after the intervention, *n* (%)	128 (10)	118 (10.2)	10 (8.5)	0.562
PRECISE-DAPT score	15 (9–26)	14 (9–23)	31 (24–41)	< 0.001
Mehran score	6.52 ± 4.8	5.84 ± 3.8	12.6 ± 7.4	< 0.001
Major bleeding, *n* (%)	44 (3.7)	28 (2.6)	16 (13.6)	< 0.001
Minor bleeding, *n* (%)	23 (1.9)	20 (1.9)	3 (2.5)	< 0.001
In-hospital mortality, *n* (%)	40 (3.1)	23 (2)	17 (14.4)	< 0.001
Length of hospital stay, days	4 (3–6)	4 (3–6)	5 (3–6)	0.400

Continuous variables are presented as mean ± SD or mean. Nominal variables are presented as frequency (%)

CIN contrast-induced nephropathy, *CHF* congestive heart failure, *COPD*; chronic obstructive pulmonary disease, *CVA* cerebrovascular accident, *CRP* C-reactive protein, *eGFR*; estimated glomerular filtration rate, *TG* triglycerides, *HDL* High-density lipoprotein, *LDL* low-density lipoprotein, *LAD* Left anterior descending

Table 2 Univariate and multivariate analyses for the predictor of CIN

	Univariate analysis		Multivariate analysis	
	p value	OR (95% CI)	*p* value	OR (95% CI)
Peak CK–MB	< 0.001	1.002 (1.001–1.003)	< 0.001	1.006 (1.004–1.008)
LAD as the infarct-related artery	0.005	1.739 (1.187–2.549)	0.002	2.391 (1.388–4.119)
PRECISE-DAPT score	< 0.001	1.096 (1.079–1.113)	< 0.001	1.090 (1.066–1.114)
Mehran score	< 0.001	1.573 (1.414–1.749)	< 0.001	1.292 (1.231–1.356)
Contrast volume/eGFR ratio	< 0.001	1.235 (1.190–1.281)	< 0.001	1.290 (1.111–1.496)

All clinically relevant parameters were included in the model

OR odds ratio, *CI* confidence interval, *CIN* contrast-induced nephropathy, *LAD* left anterior descending, *CK–MB* creatine kinase–myocardial band

anemia, hemoglobin level, white blood cell count, admission blood glucose, peak CK–MB, and LAD as the infarcted related artery, multi-vessel involvement, contrast volume/eGFR ratio, the Mehran score, and the PRECISE-DAPT score were found to be predictors of CIN in the univariate analyses. To identify the independent predictors of CIN, multivariate regression analyses with a stepwise backward model were performed using the variables that showed a marginal association with CIN in the univariate analyses. In multivariate regression analysis, peak CK–MB [odds ratio (OR) 1.006, 95% confidence interval (CI) 1.004–1.008, $p < 0.001$], LAD as the infarcted related artery (OR 2.391, 95% CI 1.388–4.119, $p = 0.002$), contrast volume/eGFR ratio (OR 1.290, 95% CI 1.111–1.496, $p < 0.001$), the Mehran score (OR 1.292, 95% CI 1.231–1.356, $p < 0.001$), and PRECISE-DAPT score (OR 1.090, 95% CI 1.066–1.114, $p < 0.001$) were found as independent predictors of CIN.

The survival and ROC curves analysis

Kaplan–Meier analysis showed that patients with CIN had a significantly higher incidence of death [*p* (log-rank) < 0.001] (Fig. 1). In ROC analysis, area under the ROC curve (AUC) values of PRECISE-DAPT, the Mehran score, age, white blood cell count, and hemoglobin, for the development of CIN were 0.834 (0.812–0.854 95% CI, $p = 0.017$), 0.807 (0.764–0.851 95% CI, $p = 0.022$), 0.726 (0.701–0.751 95% CI, $p = 0.024$), 0.519 (0.491–0.547 95% CI, $p = 0.030$), and

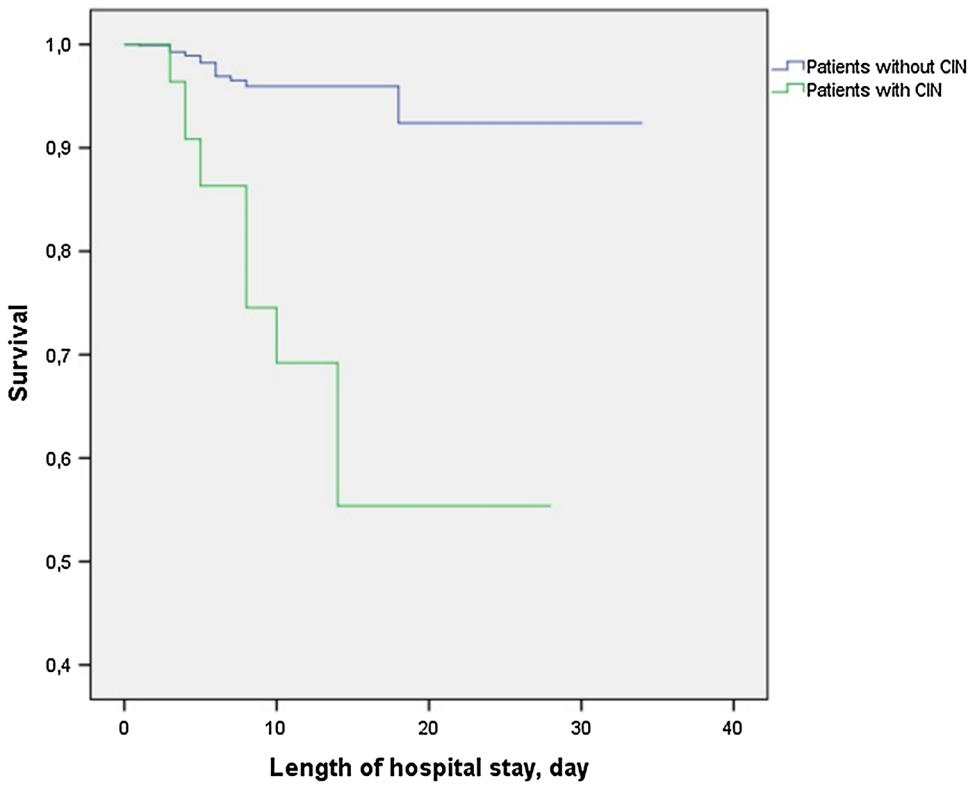

Fig. 1 Kaplan–Meier curve for the overall survival in patients with and without CIN

0.60 (0.573–0.627 95% CI, $p=0.029$),respectively (Fig. 2). The best cut-off value for PRECISE-DAPT score obtained by the ROC curve analysis was > 21 for the prediction of CIN (sensitivity: 81.3%, specificity: 72.7%). A pairwise comparison of ROC curves showed that the predictive value of the PRECISE-DAPT score with regarding to occurrence of CIN was superior compared to age, white blood cell count, and hemoglobin. In addition, the PRECISE-DAPT score was found to be a non-inferior compared to the Mehran score with regarding to predict CIN (Fig. 3).

Discussion

The present study is the first study evaluating the association between admission PRECISE-DAPT score and CIN occurrence in patients with STEMI who undergone primary PCI. We found that the PRECISE-DAPT score had a predictive value for the development of CIN in patients with STEMI treated with primary PCI.

CIN generally develops within 48–72 h after exposure to intravascular iodinated contrast media [15]. Owing to increasing frequency of coronary angiography or PCI, the incidence of CIN is growing. Besides that, when compared to elective procedures, primary PCI is related to a higher incidence of CIN. The incidence of CIN has varied from roughly 6.4% to as high as 27.7% depending on the definition criteria used [16, 17]. In the present study, CIN occurred in 9.2% of the patients. The development of CIN after STEMI is associated with increased in-hospital mortality. Bouzas-Mosquera et al. [18] reported that, following primary PCI, the in-hospital mortality was 13.9% in patients with CIN vs. 0.7% in patients without CIN. These data were also confirmed in our study. The patients with CIN had higher in-hospital mortality (14.4% vs. 2.0%) compared to patients without CIN. Multiple risk factors are proposed to explain the occurrence of CIN including renal insufficiency, hypotension, CHF, older age, anemia, and diabetes mellitus [19]. Consistent findings were also found in our study as patients with CIN tend to be older, had a higher frequency of diabetes mellitus, anemia, and CHF compared to patients without CIN. In addition, as shown in our study and previous studies, the risk for development of CIN is higher in patients with more extensive atherosclerotic disease and history of stroke [20]. Both the amount of contrast volume administered during PCI procedure and the preexistence of renal dysfunction are also important issues for the development of CIN. Therefore, a formula that includes these variables, namely contrast volume/eGFR ratio, is recommended to be a better predictor of CIN than the amount of contrast medium or renal dysfunction alone [21]. A study reported by Nozue et al. [21] demonstrated that the contrast volume/eGFR ratio might be a useful predictor of CIN developing after elective PCI. Similar to this reported study, we also showed that the contrast volume/eGFR ratio was significantly higher in patients who developed CIN after primary PCI.

Even though the exact mechanism of CIN is still not completely known in STEMI patients, there is increasing evidence to support the critical role of enhanced inflammatory response, oxidative stress, and metabolic factors including acidosis and acute hyperglycemia in the pathogenesis of CIN [22]. Stolker et al. [23] demonstrated that the elevated preprocedural glucose is associated with greater risk for CIN in patient STEMI treated with primary PCI. In our study population, we also found that admission of blood glucose level was associated with CIN development on univariate analysis, even though it did not reach statistical significance on multivariate logistic regression analysis. The large-scale Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI) trial [20], which included a total of 3602 patients with STEMI presenting within 12 h of symptoms, revealed that LAD as the infarcted related artery was independently associated with CIN development in patients with and without chronic renal disease. In addition, peak CK–MB has been demonstrated as an independent predictor for CIN in the literature

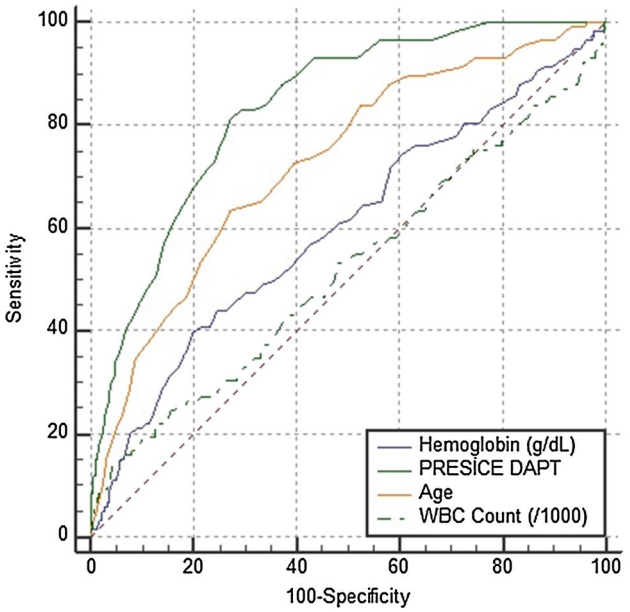

Fig. 2 Receiver-operating characteristic (ROC) curves of the PRECISE-DAPT score, age, white blood cell count, and hemoglobin. The PRECISE-DAPT score, age, white blood cell count, and hemoglobin had an area under the curve (AUC) value of 0.834 (0.812–0.854 95% CI, $p=0.017$), 0.726 (0.701–0.751 95% CI, $p=0.024$), 0.519 (0.491–0.547 95% CI, $p=0.030$), and 0.60 (0.573–0.627 95% CI, $p=0.029$), respectively. A pairwise comparison of ROC curves showed that the predictive value of the PRECISE-DAPT score with regards to development of CIN was superior compared to age, white blood cell count, and hemoglobin. *ROC* receiver-operating characteristic curve, *AUC* area under the curve, *CI* confidence interval

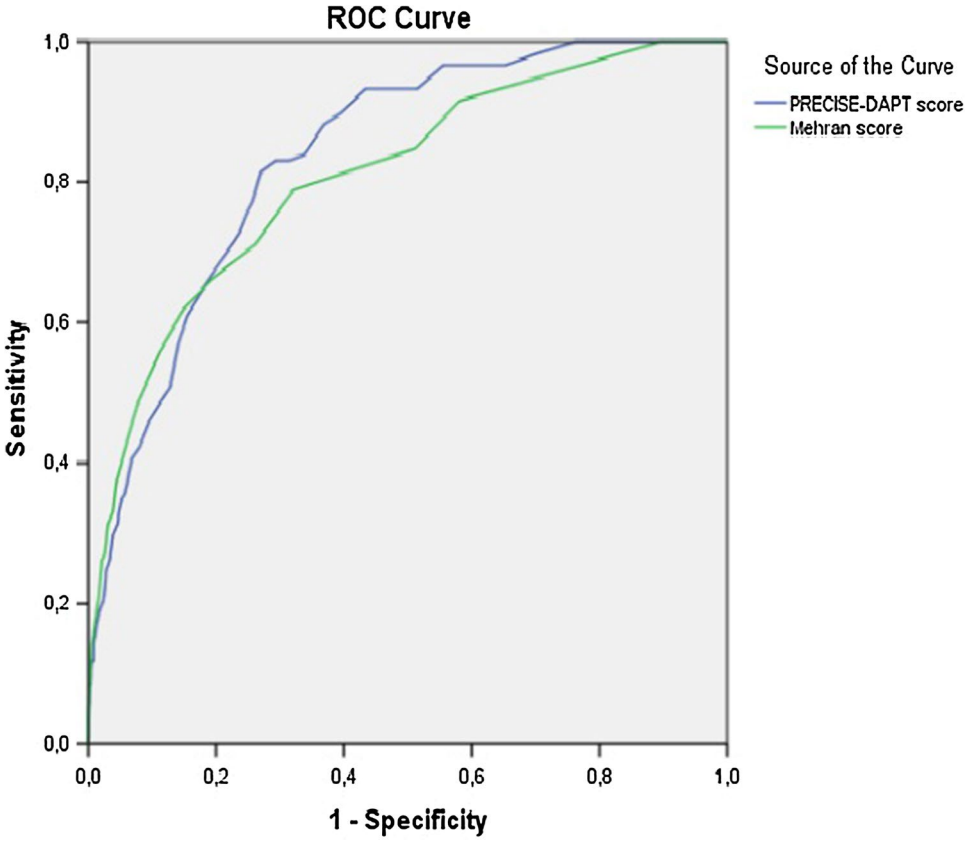

Fig. 3 Receiver-operating characteristic (ROC) curves of the PRECISE-DAPT score and the Mehran score. The PRECISE-DAPT score and the Mehran score had an area under the curve (AUC) value of 0.834 (0.812–0.854 95% CI, $p=0.017$) and 0.807 (0.764–0.851 95% CI, $p=0.022$), respectively. *ROC* receiver-operating characteristic curve, *AUC* area under the curve, *CI* confidence interval

[24]. These parameters also remained independent predictor of CIN on multivariate logistic regression analysis in our cohort.

The PRECISE-DAPT is a novel risk score that mainly developed to guide the optimal duration of dual anti-platelet treatment in patients after PCI. According to the current guidelines, in patients with PRECISE-DAPT score ≥ 25, the risk of bleeding is high, favoring shorter duration of dual anti-platelet treatment among these patients [25]. In patients with acute coronary syndrome, dual anti-platelet treatment is recommended for 12 months. However, in case of high bleeding risk, discontinuation of dual anti-platelet treatment at 6 months should be considered. The parameters used in the calculation of the PRECISE-DAPT score include age, hemoglobin level, white blood cell count, creatinine clearance rate, and prior history of bleeding. The components of PRECISE-DAPT score have been shown as important contributors to CIN development [19]. However, the ability of PRECISE-DAPT risk score for predicting CIN in patients with STEMI remains unclear. The present study might be the first to demonstrate that PRECISE-DAPT risk score had a good predictive value for CIN in patients with STEMI treated primary PCI. In addition, in comparison to the Mehran score, a well-known and validated CIN risk tool, the PRECISE-DAPT score was found to be non-inferior with regarding to predict CIN. However, because the patients with cardiogenic shock were excluded to create a more homogeneous group, this undoubtedly affected the predictive value of the Mehran score in the present study.

When patients admitted to hospital with the diagnosis of STEMI, primary PCI, where available, is the recommended treatment strategy according to the current guidelines. After the successful restoration of blood flow with angioplasty and/or stent, clinicians should calculate the PRECISE-DAPT score to estimate the optimal duration of dual anti-platelet treatment in these patients. The PRECISE-DAPT is a simple, user-friendly score, and can be calculated easily after the first medical contact. Moreover, it is easy to memorize, and can be rapidly applied by healthcare professionals without advanced medical training. As previously mentioned, the incidence of CIN is higher in patients with STEMI treated with primary PCI. Therefore, as shown in our study, the PRECISE-DAPT risk score might be useful not only for estimating optimal duration of dual anti-platelet treatment but also for predicting CIN risk in patients with STEMI undergoing primary PCI. Because the exact mechanisms responsible for the development of CIN have been unknown, and it mostly develops within the first 72 h, identifying the patients who are at high risk for CIN is a key step. Hence, clinicians should calculate the PRECISE-DAPT score after the first medical contact and be more alert in patients with higher PRECISE-DAPT score due to high risk for the development

of CIN. Particularly, the patients with higher PRECISE-DAPT score should be treated with adequate prophylactic strategies. The prophylactic regimens, which are capable of preventing CIN among these patients, may include stopping any nephrotoxic agents such as non-steroidal anti-inflammatory drugs, *N*-acetylcysteine administration, and normal saline administration [26]. Moreover, a close follow-up with the amount of urine volume in an hour or total urine volume in 24 h should be implemented in these patients. Nonetheless, further studies with long-term follow-up and large-scale prospective data are needed to elucidate the association of the PRECISE-DAPT score with the development of CIN in patients with STEMI treated with primary PCI.

Limitations of the study

The present study had some limitations. First, it was a retrospective and observational study; however, our cohort was relatively large and consecutive patients were included in the study. Second, some confounders of CIN, such as proteinuria, might not be fully evaluated. Third, the end-point of the study was CIN incidence after primary PCI; however, adverse events other than CIN were not evaluated. Fourth, in the present study, we were only able to evaluate the all-cause mortality. The data about causes of death could not evaluate due to missing data. Finally, multicenter, prospective, and large studies are needed to confirm our results.

Conclusion

This is the first study that demonstrated the predictive value of the PRECISE-DAPT score for the occurrence of CIN in patients with STEMI treated with primary PCI. The PRECISE-DAPT is a simple score and can be easily calculated at the bedside. This scoring system may be useful not only for the early estimation of CIN but also in deciding on therapeutic strategies. Follow-up of patients with higher PRECISE-DAPT score should be performed more cautiously, and it should be noted that the development of CIN risk of these patients group is high.

Compliance with ethical standards

Conflict of interest All authors declare that they do not have any conflict of interest.

Ethical standards All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed consent The need for written informed consent was waived due to retrospective study design.

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