Clinical and Applied Thrombosis/Hemostasis
Volume 28: 1-8
© The Author(s) 2022
Article reuse guidelines:
sagepub.com/journals-permissions
DOI: 10.1177/10760296221116353
journals.sagepub.com/home/cat
SAGE

Mehran 2 Contrast-Associated Acute Kidney Injury Risk Score: Is it Applicable to the Asian Percutaneous Coronary Intervention Population?

Ying Guo, MD[1*], Xue Xu, MD[1*], Yunjing Xue, MD[1], Chunling Zhao, MD[1], Xiaohong Zhang, MD[1], and Hongfu Cai, MD[2]

Abstract

Contrast-associated acute kidney injury (CA-AKI) can occur after percutaneous coronary intervention (PCI). The Mehran score is the gold standard for predicting CA-AKI risk, and it has recently been updated. The Mehran 2 CA-AKI risk score, based on existing variables in patients undergoing PCI, can accurately differentiate the risk of CA-AKI. This study aimed to verify whether the new Mehran score is applicable to the Asian PCI population. The study included the clinical data of 2487 patients undergoing PCI from August 2020 to February 2022. The goodness-of-fit test (Hosmer-Lemeshow) was used to evaluate the correction ability of the Mehran 2 score, and the area under the receiver operating characteristic curve (ROC) was used to evaluate the accuracy of the Mehran 2 score in predicting CA-AKI. CA-AKI occurred in 28 of 2487 patients, with an incidence rate of 1.12%. The proportion of high risk factors for AKI in the cohort was lower than that in the Mehran 2 cohort (a large contemporary PCI cohort to develop the Mehran 2 score). The Mehran 2 risk score had excellent goodness-of-fit ($\chi 2 = 5.320$, $df = 6$, $P = 0.503$) and high predictive accuracy (area under the ROC curve 0.836, $P < 0.0001$). The Mehran 2 score shows good predictive and correction performance in the Asian population and has good clinical application value. The inclusion of the Mehran 2 risk score in patients hospitalised for coronary angiography appears to be good practice.

Keywords

mehran 2, contrast, acute kidney injury risk, CA-AKI, percutaneous coronary intervention

Date received: 27 April 2022; revised: 8 July 2022; accepted: 11 July 2022.

Introduction

With the increasing use of contrast agents in clinical diagnosis and treatment, acute kidney injury caused by contrast agents, namely, contrast-associated acute kidney injury (CA-AKI), has gradually gained attention. CA-AKI is the third leading cause of hospital-acquired acute kidney injury after renal hypoperfusion and nephrotoxic drugs.[1,2] Patients undergoing percutaneous coronary intervention (PCI) typically have a high prevalence of comorbidities and might also sustain acute haemodynamic disturbances; hence, contrast-associated acute kidney injury has been studied extensively in the context of PCI. CA-AKI can increase mortality and hospitalisation costs and prolong hospital stay; however, there is no effective treatment plan.[3,4] Therefore, it is particularly critical to assess the risk of CA-AKI, identify high risk groups, and provide

[1] Department of Radiology, Fujian Medical University Union Hospital, Fujian Medical University, Fuzhou, China
[2] Department of Pharmacy, Fujian Medical University Union Hospital, Fujian Medical University, Fuzhou, China

*Ying Guo, Xue Xu, These authors have contributed equally to this work

Corresponding Authors:
Hongfu Cai, Department of Pharmacy, Fujian Medical University Union Hospital, Xinquan 29 Road, Gulou District, Fuzhou, China.
Email: caihongfu31@126.com

Xiaohong Zhang, Department of Radiology, Fujian Medical University Union Hospital, Xinquan 29 Road, Gulou District, Fuzhou, China.
Email: zhangxhfj@163.com

preventive measures. The Mehran score is a CA-AKI risk assessment score based on patients who underwent PCI in 2004.[5] This risk score has been widely used in clinics and has been validated in relevant studies.[6,7] However, the Mehran score excluded patients admitted for shock or acute myocardial infarction. With significant advances in PCI, there have also been developments in risk assessment and preventive measures for CA-AKI, such as the use of hypotonic or isotonic contrast agents instead of hyperosmolar agents as well as the cautious use of contrast media dose volume, thereby reducing the occurrence of contrast-related renal injury.[8] Therefore, Mehran et al investigated a contemporary simple risk score for predicting CA-AKI after PCI (Mehran 2 CA-AKI risk score) in November 2021.[9] Patients who underwent PCI at a large tertiary care centre from 1 January 2012 to 31 December, 2020, in the United States and had preoperative and 48-h postoperative creatinine data were included. Independent predictors of CA-AKI were derived from multivariate logistic regression analysis. Model 1 included only preoperative variables and Model 2 included surgical variables. Inclusion of intraoperative variables in the models improved the distinction in risk scores only marginally (C-statistic in the derived cohort: 0.72 for Model 1 and 0.74 for Model 2; in the validation cohort: 0.84 for Model 1 and 0.86 for Model 2). Patients with CA-AKI had a significantly increased 1-year risk of death (10.2% vs 2.5%; adjusted hazard ratio, 1.76; 95% confidence interval [CI] 1.31−2.36; p = 0.0002). The Mehran 2 CA-AKI risk score, based on existing variables in patients undergoing PCI, can accurately differentiate the risk of CA-AKI.[9]

The identification of at-risk patients and appropriate preventive management remain key factors for improving patient outcomes. However, this score lacks verification data for the Chinese population. This study retrospectively collected clinical data of patients undergoing coronary angiography and interventional therapy in a large tertiary first-class hospital to verify the validity of the Mehran 2 score in the risk assessment of CA-AKI in the Asian population and to provide clinical reference.

Materials and Methods

Patients

Data from 8950 patients who underwent PCI for the first time between August 2020 and February 2022 in Fujian Medical University Union Hospital, a Chinese regional medical centre, were retrospectively collected. The following patients were excluded: patients with no basal creatinine value before PCI (n = 661), patients with no 48-h postoperative creatinine values (n = 5712), patients with pre-PCI end-stage renal disease requiring dialysis (n = 86), and patients with unobtainable preoperative haemoglobin (n = 4). Finally, 2487 patients were included in this study. PCI was performed according to standard techniques, and the options for stents and surgical techniques were not excluded. Routine hydration took place during the perioperative period. This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Fujian Medical University Union Hospital.

Definitions

The primary endpoint was acute kidney injury (AKIN). AKIN was defined as an increase in serum creatinine of at least 50% or at least 0.3 mg/dL within 48 h after PCI, compared with pre-PCI serum creatinine.[10–12] The Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) evaluation formula was used to calculate the estimated glomerular filtration rate (eGFR). Chronic kidney disease was defined as eGFR <60 mL/min/1.73 m2.[13]

Calculation of the Mehran 2 CA-AKI Risk Score

Model 2, which included postoperative parameters, only slightly improved the risk score distinction; therefore, this study only validated Model 1, which included only preoperative variables. The scoring method of Mehran 2 (Model 1) was as follows (table 1): asymptomatic or stable angina, 0 points; unstable angina, 2 points; non-ST-elevation myocardial infarction (NSTEMI), 4 points; ST-elevation myocardial infarction (STEMI), 8 points; eGFR (30-59 mL/min/1.73 m^2), 1 point; eGFR (<30 mL/min/1.73 m^2), 4 points; left ventricular ejection fraction (LVEF) <40%, 2 points; diabetes (non-insulin-treated), 1 point; diabetes (insulin-treated), 2 points; haemoglobin <11 g/dL, 1 point; basal glucose ≥150 mg/dL, 1 point; congestive heart failure on presentation, 1 point; and age ≥75 years, 1 point. The total score of the above variables was calculated and divided into 4 risk levels: low risk (0-2 points), moderate risk (3-7 points), high risk (8-11 points) and very high risk (≥12 points).[9]

Statistical Analysis

Data Collection and Descriptive Statistics

Statistical Package for the Social Sciences 20.0 (IBM, Chicago, IL, USA) was used for statistical analysis, and categorical or dichotomous variables were expressed as absolute values and percentages and were compared using the Pearson $\chi 2$ test. Continuous variables are described as mean ± standard deviation (SD). Student's t-test was used for comparisons of continuous variables between the two groups of patients. In a two-sided test, a P-value <0.05 indicated statistical significance. All patients were scored according to the variables included in the model and their weights.

A receiver operating characteristic (ROC) curve was used to evaluate the predictive ability of the model for CA-AKI. A model with a C-statistic >0.70 is generally considered to have acceptable discriminatory capacity.[14] The ability to correct was tested using a goodness-of-fit test (Hosmer-Lemeshow [H-L]) and a graph reflecting the agreement between model-predicted probabilities and actual values.[6,14] This test determines how closely the predicted event rate approximates the observed event rate over a range of scores.

Table 1. Mehran Versus Mehran 2 Risk Score (Model 1 for Mehran 2 Included Only Pre-Procedural Variables, Model 2 for Mehran 2 Included Pre-Procedural and Procedural Variables).

Risk Factors	Integer Score	Risk Factors	Integer Score
Mehran risk score			
Hypotension	5	eGFR, mL/min per 1.73m^2 40–60	2
Intra-aortic balloon pump	5	20–40	4
Congestive heart failure class III/IV	5	<20	6
Age>75 years	4	or	
Anemia	3	Serum creatinine>1.5 mg/dL	4
Diabetes	3		
Contrast media volume	1 for each 100cc^3		
Mehran 2 risk score			
pre-procedural (Model 1)		**Procedural (other variables for Model 2)**	
Presentation		Contrast volume, mL	
Asymptomatic or stable angina	0	<100	0
Unstable angina	2	100–199	1
NSTEMI	4	200–299	2
STEMI	8	≥300	4
eGFR, mL/min per 1.73 m^2		Procedural bleed	4
≥60	0	Slow flow or no flow post procedure	2
30-59	1	Complex anatomy	1
<30	4		
Left ventricular ejection fraction <40%	2		
Diabetes			
No diabetes	0		
Non-insulin-treated	1		
Insulin-treated	2		
Haemoglobin <11 g/dL	1		
Basal glucose ≥150 mg/dL	1		
Congestive heart failure on presentation	1		
Age >75 years	1		

eGFR = estimated glomerular filtration rate. NSTEMI = non-ST-elevation myocardial infarction. STEMI = ST-elevation myocardial infarction.
Mehran total score: Low risk (0 to 5); Moderate risk (6 to 10); High risk (11 to 15); Very high risk (≥16).
Mehran 2 total score: Low risk (0 to 2); Moderate risk (3 to 7); High risk (8 to 11); Very high risk (≥12).

Results

Baseline Characteristics

Among the 2487 patients, there were 1898 males and 589 females, with an average age of 64.30 ± 11.13 years. Of the 28 patients (1.13%) who developed AKI, all had serum creatinine increased by ≥0.3 mg/dL compared with the baseline value. The serum creatinine level in 9 cases increased by ≥50% compared with the baseline value. The basic clinical characteristics of the study cohort and the Mehran 2 cohort are shown in Table 2. The mean age of the cohort in the study was similar to that of the Mehran 2 cohort, but the Mehran 2 cohort had a higher proportion of older age, lower eGFR, higher prevalence of diabetes, higher proportion of LVEF <40, higher prevalence of heart failure, and higher proportion of stable or asymptomatic patients. The haemoglobin values were similar. Overall, the proportion of high risk factors for AKI was higher in the Mehran 2 cohort than that in this cohort.

Table 3 shows the differences between patients with different AKI outcomes. Patients with CA-AKI were older and had a higher incidence of unstable angina, NSTEMI, STEMI, left ventricular blood uptake fraction, basal blood glucose, insulin use, haemoglobin level, and renal insufficiency. Patients in our cohort were administered iohexol or iodixanol. There was no statistical difference in the AKI outcomes between the two drugs (P = 0.565).

Mehran 2 Score Population Distribution

According to the Mehran 2 score,[9] the proportions of people with low, moderate, high, and very high risks of AKI were 40.8% (n = 1015), 47.6% (n = 1184), 10.2% (n = 254), and 1.4% (n = 34), respectively. According to the diagnostic criteria for AKI that the serum creatinine increased by ≥0.3 mg/dL or ≥50% from the baseline value within 48 h after the use of the contrast agent, the incidence of AKI was 0.3%, 0.4%, 5.5%, and 17.7% in the low, moderate, high, and very high risk groups, respectively, and the incidence of AKI increased with increasing risk.

Calibration and Discrimination

The area under the ROC curve for CA-AKI was 0.836 (95% CI 0.744–0.928, P < 0.0001) (Figure 1). The H-L test analysis found that the Mehran 2 risk score had a very strong

Table 2. Comparison of Clinical Data Between Validation Cohort and Mehran 2 Score Cohort.

	Mehran2's Derivation Cohort (n = 14616)	Mehran2's Validation Cohort (n = 5606)	Present Population (n = 2487)
Age, years	66.2 (11.6)	67.0 (11.3)	64.30 (11.1)
Age>75 years	3701 (25.3%)	1535 (27.3%)	396 (15.9%)
Sex			
Female	4268 (29.2%)	1481 (26.4%)	589 (23.7%)
Male	10348 (70.8%)	4125 (73.6%)	1898 (76.3%)
Diabetes			
Diabetes	6967 (47.6%)	2573 (45.9%)	944 (38.0%)
Non-insulin-treated	4668 (31.9%))	1631 (29.1%)	673 (27.1%)
Insulin-treated	2299 (15.7%)	942 (16.8%)	271 (10.9%)
Left ventricular ejection fraction<40%	1470 (10.1%)	-	92 (3.7%)
eGFR, mL/min per 1·73 m^2	73.4 (22.4)	75.1 (22.7)	82.0 (20.6)
Chronic kidney disease	4123 (28.2%)	1806 (32.2%)	540 (21.7%)
Congestive heart failure at presentation	1221 (8.4%)	754 (13.4%)	28 (1.1%)
Clinical presentation			
Asymptomatic/Stable angina	8040 (55.0%)	3654 (65.2%)	631 (25.4%)
Unstable angina	4687 (32.1%)	797 (14.2%)	1100 (44.2%)
NSTEMI	1411 (9.7%)	946 (16.9%)	546 (22.0%)
STEMI	466 (3.2%)	209 (3.7%)	210 (8.4%)
Haemoglobin, g/dL	12.9 (1.7)	13.2 (1.8)	13.5 (1.8)

eGFR = estimated glomerular filtration rate NSTEMI = non-ST-elevation myocardial infarction. STEMI = ST-elevation myocardial infarction

Table 3. The Association of Variables with CA-AKI in the Cohort.

	CA-AKI			
Variable	YES N = 28	NO N = 2459	Statistics	p-Value
Age	66.36	64.27	8.927	0.003
Age>75	10	386	8.286	0.004
Sex（female)	11	578	3.814	0.051
Stable angina or Asymptomatic	3	628	3.213	0.073
Unstable angina	4	1096	10.294	0.01
NSTEMI	9	537	1.716	0.19
STEMI	12	198	43.381	<0.001
Left ventricular ejection fraction<40%	6	86	24.987	<0.001
Diabetes	15	929	2.932	0.087
Insulin treated	12	259	29.793	<0.001
Basal glucose>150 mg/dL	13	349	23.131	<0.001
Congestive heart failure on presentation	1	27	15.22	0.217
Drug (Iohexol Injection)	26	2341	0.331	0.565
Dosage	67.76	66.71	0.033	0.856
Haemoglobin, g/L	130.07	134.84	6.056	0.014
eGFR, mL/min per 1·73 m^2	67.34	82.18	13.659	<0.001

CA-AKI = contrastassociated acute kidney injury. NSTEMI = non-ST-elevation myocardial infarction. STEMI = ST-elevation myocardial infarction. eGFR = estimated glomerular filtration rate.

goodness-of-fit ($\chi 2 = 5.320$, df = 6, P = 0.503). Figure 2 shows that the predicted and observed values of CA-AKI were almost identical in the different risk groups.

Discussion

Here, we present the applicability of the Mehran 2 risk model for predicting acute kidney disease induced by coronary angiography in the Chinese population. The main results are as follows: (1) the Mehran 2 risk score can predict the risk of CA-AKI in patients undergoing coronary angiography; (2) the area under the ROC curve was the same as the validation cohort of Mehran 2 (0.836 vs 0.840, respectively).

Contrast-induced nephropathy is a common complication of PCI, which can prolong hospital stay, increase treatment costs, and lead to irreversible kidney damage and adverse outcomes

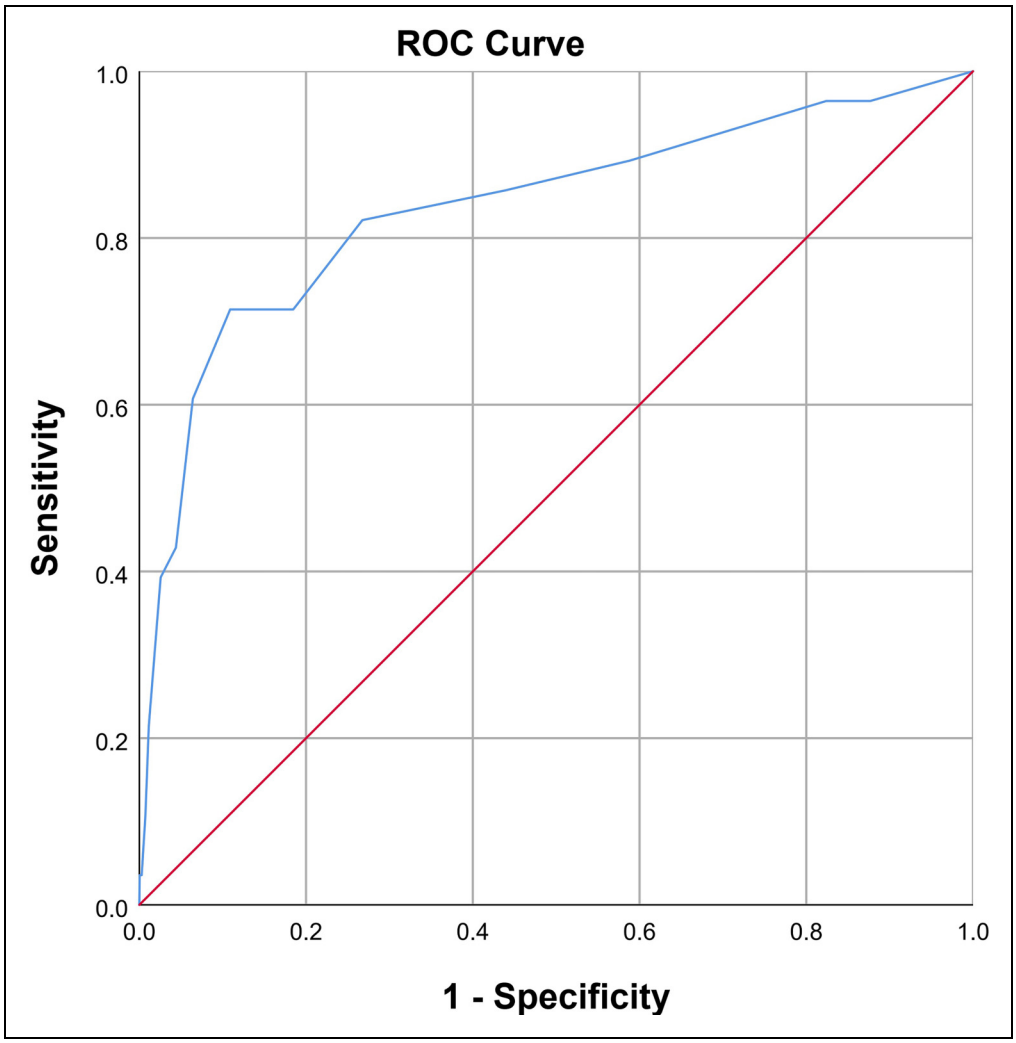

Figure 1. ROC curve for model.

such as dialysis and even death.[15,16] The pathogenesis of CA-AKI is complex and may be related to contrast media nephrotoxicity, allergic reactions, and changes in renal haemodynamics. The symptoms of most patients with CA-AKI include mild and transient renal impairment; however, if the patient has multiple risk factors at the same time, persistent renal impairment may occur, which is life-threatening in severe cases.[1,17,18] Currently, there is no effective treatment option for CA-AKI. Several preoperative preventive interventions, including intravascular volume expansion, acetylcysteine use, and statin use, can reduce the incidence of CA-AKI.[19–21] Intensive preventive interventions may benefit high risk patients with CA-AKI, and preoperative risk assessments as well as intraoperative and postoperative risk management, play key roles in improving patient outcomes. Physicians can balance the benefits and risks of PCI angiography, choose the best timing, and employ the most appropriate strategy to avoid AKI.[22]

Common clinical risk factors for CA-AKI include aging, hypertension, diabetes, chronic kidney disease (eGFR decline), cardiac insufficiency, peripheral vascular disease, anaemia, nutritional deficiencies, and the type and dosage of contrast agents.[6,23] More importantly, these risk factors have an additive effect, and the accumulation of multiple risk factors will directly lead to a significant increase in the risk of AKI.[24] There are many risk warning systems for CA-AKI, and different models have a variety of predictors and risk thresholds for the same predictor.[25,26] The Mehran score is a classic and the most commonly used scoring system; it has long been used as the "gold standard" to measure the discriminative ability of other novel pre-glance models.[27] The system was developed by Mehran et al in 2004,[5] but it still has some flaws. For example, it contains up to eight variables, including perioperative variables, which greatly reduce the system's clinical application ability, especially for patients who need PCI surgery due to acute myocardial infarction, with a C-statistic of only 0.67; thus, standard risk scores developed in patient cohorts nearly 20 years ago may no longer be the most accurate option.[9] The model was updated in 2021 by Mehran et al[9] The updated model is an AKI risk prediction score based on data

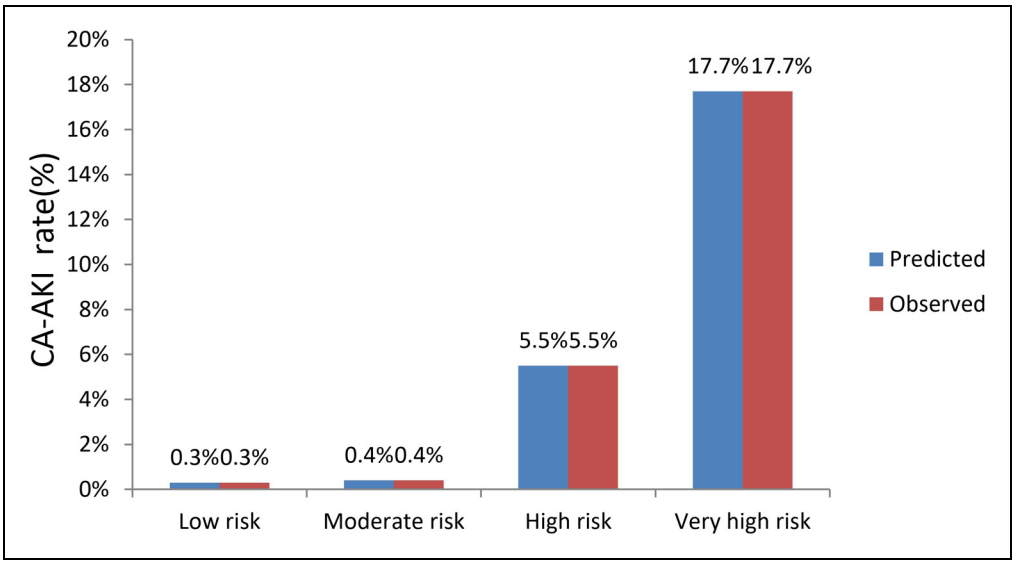

Figure 2. Observed versus predicted contrast-associated acute kidney injury from the Mehran 2 score.

from 14616 PCI patients in the United States, including Model 1 with preoperative variables and Model 2 with additional postoperative variables. Compared with the original Mehran score, there was higher discrimination, and patients with STEMI were included. The Mehran 2 score may be more aligned with predictions in modern PCI populations, resulting in a reduced prevalence. Whether Mehran's updated version can be widely used has attracted much attention. The Mehran 2 score has not been well validated externally in a large Chinese cohort, limiting its use. Among the populations studied by Mehran et al,[9] only 18.2% of the derivation cohort and only 3% of the validation cohort were Asian. Therefore, it is necessary to validate the Mehran score for the Asian population.

Our study exclusively validated Model 1 for the following reasons. Only eight common preoperative clinical variables (age, clinical presentation, eGFR, congestive heart failure, diabetes, haemoglobin, basal glucose, and left ventricular ejection fraction) were included in Model 1, which were got available. Model 2 provided only a slightly higher discrimination than Model 1, and preoperative variables were relatively easy to calculate and predict. Compared with Model 2, Model 1 was easier to promote and use in clinical practice. A variable for contrast volume was also included in Model 2; however, the required contrast dose is often unknown until surgery is complete. Since decisions on diagnostic testing and preventive treatment are often made before surgery, the inclusion of contrast volume as a model variable limits the usefulness of Model 2.[28]

In our cohort, only 28 patients (1.13%) developed AKI, which was lower than the 4.3% reported in the Mehran 2 cohort. This may be due to the fact that, compared with the Mehran 2 study, we included fewer high risk factors, such as a lower proportion of elderly patients, better preoperative renal function, lower prevalence of diabetes, lower proportion of LVEF <40, and lower prevalence of heart failure. These are important variables that affect the outcomes of AKI. In addition, many patients in the study were excluded because they did not have 48-h postoperative creatinine values, which could explain the differences in AKI rates.

Related studies have reported that the incidence of CA-AKI is 2–25%,[29] which is different from our results. This may be due to several factors. Chronic kidney disease is the strongest independent risk factor for CA-AKI, and its incidence is closely related to the degree of renal impairment before angiography.[30–32] The 2487 patients with CKD included in this study had an average eGFR of 82 mL/min/1.73 m2, which may further explain the low incidence of AKI. However, it may be that CA-AKI has received a higher degree of attention at present, and the preventive measures received by patients in the hospital are more comprehensive and complete, therefore, the incidence of CA-AKI has decreased. Another possible reason is that there are two different sets of diagnostic criteria for contrast nephropathy. The old standard was a >44 μmol/L increase in serum creatinine 2–5 days after receiving the contrast medium or a 25% increase in serum creatinine from baseline. The new criterion is defined as an increase in serum creatinine of ≥50% or ≥0.3 mg/dL within 48 h after PCI. The benefit of the new criterion is increased contrast nephropathy sensitivity but decreased specificity.[33]

This study had some limitations. First, this was a retrospective study, and many cases were excluded due to failure creatinine measurement at 48 h after PCI, therefore, the incidence of AKI may be underestimated. Further, the final number of patients and events included was limited, which may have affected the bias of the results. Additionally, single-centre data was used to validate the model, therefore, the general

applicability of the results is limited. Therefore, larger cohort studies are needed to verify whether the Mehran 2 score is applicable to the current Chinese population.

Conclusions

Mehran 2, a new, updated, and contemporary user-friendly risk score, shows good predictive and correction performance in the Asian population and has high clinical application value. The inclusion of the Mehran 2 risk score in patients hospitalised for coronary angiography appears to be a beneficial practice.

Authors' Note

All authors contributed to: (1) substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; (2) drafting the article or revising it critically for important intellectual content; and (3) final approval of the version to be published.

Acknowledgements

We would like to thank the big data center of Fujian Medical University Union Hospital and the patients and clinicians who took part in this study.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics Statement

The studies involving human participants were reviewed and approved by Fujian Medical University Union Hospital Ethics Committee.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the Fujian Provincial Department of Science and Technology, (grant number 2020Y9070).

ORCID iD

Ying Guo https://orcid.org/0000-0001-8983-8448

References

1. McCullough PA, Choi JP, Feghali GA, et al. Contrast-Induced acute kidney injury. *J Am Coll Cardiol.* 2016;68(13):1465–1473.
2. Maeder M, Klein M, Fehr T, Rickli H. Contrast nephropathy: review focusing on prevention. *J Am Coll Cardiol.* 2004;44(9): 1763–1771.
3. Nijssen EC, Rennenberg RJ, Nelemans PJ, et al. Prophylactic hydration to protect renal function from intravascular iodinated contrast material in patients at high risk of contrast-induced nephropathy (AMACING): a prospective, randomised, phase 3, controlled, open-label, non-inferiority trial. *Lancet.* 2017;389(10076): 1312–1322.
4. NICE guidelines. Acute kidney injury: prevention, detection and management. 18 December 2019. https://www.nice.org.uk/guidance/ng148 (accessed Mar 30, 2022).
5. Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol.* 2004;44(7):1393–1399.
6. Abellás-Sequeiros RA, Raposeiras-Roubín S, Abu-Assi E, et al. Mehran contrast nephropathy risk score: is it still useful 10 years later. *J Cardiol.* 2016;67(3):262–267.
7. Zhou X, Sun Z, Zhuang Y, et al. Development and validation of nomogram to predict acute kidney injury in patients with acute myocardial infarction treated invasively. *Sci Rep.* 2018;8(1):9769.
8. Lun Z, Liu L, Chen G, et al. The global incidence and mortality of contrast-associated acute kidney injury following coronary angiography: a meta-analysis of 1.2 million patients. *J Nephrol.* 2021;34(5):1479–1489.
9. Mehran R, Owen R, Chiarito M, et al. A contemporary simple risk score for prediction of contrast-associated acute kidney injury after percutaneous coronary intervention: derivation and validation from an observational registry. *Lancet.* 2021;398(10315): 1974–1983.
10. Mehta RL, Kellum JA, Shah SV, et al. Acute kidney injury network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care* 2007;11(2):R31.
11. Khwaja A. KDIGO Clinical practice guidelines for acute kidney injury. *Nephron Clin Pract* 2012;120(4):c179–c184.
12. KDIGO. Clinical practice guideline for the management of blood pressure in chronic kidney disease. *Kidney Int.* 2021;99(3S): S1–S87.
13. Levey AS, Stevens LA. Estimating GFR using the CKD epidemiology collaboration (CKD-EPI) creatinine equation: more accurate GFR estimates, lower CKD prevalence estimates, and better risk predictions. *Am J Kidney Dis.* 2010;55(4): 622–627.
14. Hanley JA, McNeil BJ. The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology.* 1982; 143(1):29–36.
15. Fähling M, Seeliger E, Patzak A, Persson PB. Understanding and preventing contrast-induced acute kidney injury. *Nat Rev Nephrol.* 2017;13(3):169–180.
16. van der Molen AJ, Reimer P, Dekkers IA, et al. Post-contrast acute kidney injury - part 1: definition, clinical features, incidence, role of contrast medium and risk factors : recommendations for updated ESUR contrast Medium safety committee guidelines. *Eur Radiol.* 2018;28(7):2845–2855.
17. Pistolesi V, Regolisti G, Morabito S, et al. Contrast medium induced acute kidney injury: a narrative review. *J Nephrol.* 2018;31(6):797–812.
18. Azzalini L, Kalra S. Contrast-Induced acute kidney injury-definitions, epidemiology, and implications. *Interv Cardiol Clin.* 2020;9(3):299–309.
19. Subramaniam RM, Suarez-Cuervo C, Wilson RF, et al. Effectiveness of prevention strategies for contrast-induced nephropathy: a systematic review and meta-analysis. *Ann Intern Med.* 2016;164(6):406–416.

20. Hossain MA, Costanzo E, Cosentino J, et al. Contrast-induced nephropathy: pathophysiology, risk factors, and prevention. *Saudi J Kidney Dis Transpl.* 2018;29(1):1–9.
21. Hong WY, Kabach M, Feldman G, Jovin IS. Intravenous fluids for the prevention of contrast-induced nephropathy in patients undergoing coronary angiography and cardiac catheterization. *Expert Rev Cardiovasc Ther.* 2020;18(1):33–39.
22. Leoncini M, Toso A, Maioli M, Tropeano F, Villani S, Bellandi F. Early high-dose rosuvastatin for contrast-induced nephropathy prevention in acute coronary syndrome: results from the PRATO-ACS study (protective effect of rosuvastatin and antiplatelet therapy on contrast-induced acute kidney injury and myocardial damage in patients with acute coronary syndrome). *J Am Coll Cardiol.* 2014;63(1):71–79.
23. James MT, Samuel SM, Manning MA, et al. Contrast-induced acute kidney injury and risk of adverse clinical outcomes after coronary angiography: a systematic review and meta-analysis. *Circ Cardiovasc Interv.* 2013;6(1):37–43.
24. Cochran ST, Wong WS, Roe DJ. Predicting angiography-induced acute renal function impairment: clinical risk model. *AJR Am J Roentgenol.* 1983;141(5):1027–1033.
25. Ma B, Allen DW, Graham MM, et al. Comparative performance of prediction models for contrast-associated acute kidney injury after percutaneous coronary intervention. *Circ Cardiovasc Qual Outcomes.* 2019;12(11):e005854.
26. Li D, Jiang H, Yang X, et al. An online Pre-procedural nomogram for the prediction of contrast-associated acute kidney injury in patients undergoing coronary angiography. *Front Med (Lausanne*). 2022;9:839856.
27. Parco C, Brockmeyer M, Kosejian L, et al. National cardiovascular data registry-acute kidney injury (NCDR) vs. Mehran risk models for prediction of contrast-induced nephropathy and need for dialysis after coronary angiography in a German patient cohort. *J Nephrol.* 2021;34(5):1491–1500.
28. Silver SA, Shah PM, Chertow GM, Harel S, Wald R, Harel Z. Risk prediction models for contrast induced nephropathy: systematic review. *BMJ.* 2015;351:h4395.
29. Crimi G, Leonardi S, Costa F, et al. Incidence, prognostic impact, and optimal definition of contrast-induced acute kidney injury in consecutive patients with stable or unstable coronary artery disease undergoing percutaneous coronary intervention. Insights from the all-comer PRODIGY trial. *Catheter Cardiovasc Interv.* 2015;86(1):E19–E27.
30. Liu YH, Liu Y, Tan N, et al. Contrast-induced nephropathy following chronic total occlusion percutaneous coronary intervention in patients with chronic kidney disease. *Eur Radiol.* 2015;25(8):2274–2281.
31. Kroneberger C, Enzweiler CN, Schmidt-Lucke A, Rückert RI, Teichgräber U, Franiel T. Contrast-induced nephropathy in patients with chronic kidney disease and peripheral arterial disease. *Acta Radiol Open.* 2015;4(6):1–7.
32. Marenzi G, Marana I, Lauri G, et al. The prevention of radiocontrast-agent-induced nephropathy by hemofiltration. *N Engl J Med.* 2003;349(14):1333–1340.
33. Mehran R, Dangas GD, Weisbord SD. Contrast-Associated acute kidney injury. *N Engl J Med.* 2019;380(22):2146–2155.